



# High Lights

Anesthesia Management of Patients with Mental Retardation Effect of Acetaminofen Versus Lornoxicam Admistration on Oxidative Stress Relationship Between Premature ventricular complexes and Neutrophil Lymphocyte Ratio Treatment Of Postoperative Atelectasis With Dornase Alpha (Pulmozyme)

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# Vagus nerve stimulation in patients with Alzheimer's disease

Metin Tulgar<sup>1</sup>, Erol Ozan<sup>2\*</sup>, Zafer Akan<sup>3</sup>

Dear Editor,

Alzheimer's disease (AD) is a devastating, progressive condition associated with memory and cognitive disturbances, mood swings and behavioral changes, and the quality of life reduction (1). AD is the most common cause of dementia in person older than 65 years, with 1 in 10 older than 65 years afflicted and half among those older than 85 years afflicted. More than 4.5 million persons are currently affected in the United States by AD, and is expected to nearly triple by the year 2050 in the absence of preventive treatments (2).

AD is pathologically characterized by the accumulation of amyloid plaques and tau-associated neurofibrillary tangles (3). Patients with AD have increased cerebrospinal fluid (CSF) levels of tau protein and decreased CSF levels of β-amyloid.

Although, patients with AD experience significant elevations in CSF tau levels, CSF tau levels have been shown to remain stable over extended periods of time (4). Several neurotransmitter systems are pathologically altered in AD. Cholinergic neurons in the nucleus basalis of Meynert degenerate early in the course of the disease.

These neurons provide wide spread projections to the association cortices, and loss of acetlycholine the mechanistic basis for is cholinesterase inhibition in AD treatment. Glutamatergic function also is disregulated in AD, with inhibition of the pathological stimulation of the NMDA receptor providing the scientific rationale for the mechanism of the noncompetitive glutamate antagonist, memantine.

In addition to the atrophy of the basal forebrain cholinergic system, marked neuronal loss occurs within the locus ceruleus and the raphe nucleus in AD (5). Significant reduction of norepinephrine in the temporal cortex occur in AD and correlates with the degree of cognitive impairment (6).

Disturbances in serotonin metabolism also have been reported in AD (7). The U.S. Food and Drug Administration (FDA) had approved 5 drugs (tacrine, donepezil, rivastigmine, and galantamine) for the treatment of AD (8).

None of these treatments have been shown to modify the disease process in patients with AD, but they provide benefit to the patient, family, and caregivers by slowing the patient's progressive decline.

Vagus nerve stimulation (VNS) has been shown to activate the locus ceruleus (9) and to increase norepinephrine (10) output into the basolateral amygdala and hippocampus in animal models.

Activation of the raphe nucleus with VNS also has been recently demonstrated (11). Merill et al reported cognitive-enhancing effects of VNS during the first 6 months of treatment in a small pilot study of 10 patients with AD (12). Later, they published a follow-up report including additional 7 patients, with for at least 1 year for all 17 patients.

Vagus nerve stimulation method promising new powerful alternative approaches in some neurological disorders especially in the treatment of refractory epilepsy (13).

In this clinical research, 14 patients with Alzheimer's disease who were treated with VNS were reviewed and the results were assed in the light of the literature. Only first and second phase of AD patients were included in the study. In total 14 AD patients (7 man and 7 women aged between 68 and 82 year old) participated in this study. Patients were followed up for one year.

As soon as each implant was activated (two weeks after the implantation) attention of patients dramatically were improved, progressively going better On the other hand, 3 patients, who had difficulty in speaking along AD, improved by the VNS their talk within 3 months.

According to our observed preliminary results, a new therapy potential is arising struggling against Alzheimer diseases.

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#### References

- Evans DA, Funkenstein HH, Albert MS, et al. Prevalence of Alzheimer's disease in a community population of older persons: higher than previously reported. JAMA 1989;262:2551–2556
- Ernst RL, Hay JW. The US economic and social costs of Alzheimer's disease revisited. Am J Public Health 1994;84:1261–1264
- Sunderland T, Linker G, Mirza N, et al. Decreased betaamyloid1–42 and increased tau levels in cerebrospinal fluid of patients with Alzheimer disease. JAMA 2003;289:2094–2103
- Sunderland T, Wolozin B, Galasko D, et al. Longitudinal stability of CSF tau levels in Alzheimer patients. Biol Psychiatry 1999;46:750–755
- Reisberg B, Doody R, Stoffler A, et al. Memantine in moderate-to-severe Alzheimer's disease. N Engl J Med 2003;348:1333–1341
- Matthews KL, Chen CP, Esiri MM, et al. Noradrenergic changes, aggressive behavior, and cognition in patients with dementia. Biol Psychiatry 2002;51:407–416
- Gottfries CG. Disturbance of the 5-hydroxytryptamine metabolism in brains from patients with Alzheimer's dementia. J Neural Transm Suppl 1990;30:33–43
- Knapp MJ, Knopman DS, Solomon PR, et al. A 30-week randomized controlled trial of high-dose tacrine in patients with Alzheimer's disease. The Tacrine Study Group. JAMA 1994;271:985–991

- Naritoku DK, Terry WJ, Helfert RH. Regional induction of fos immuno-reactivity in the brain by anticonvulsant stimulation of the vagus nerve. Epilepsy Res 1995;22:53– 62
- Hassert DL, Miyashita T, Williams CL. The effects of peripheral vagal nerve stimulation at a memorymodulating intensity on norepinephrine output in the basolateral amygdala. Behav Neurosci 2004;118:79–88
- Kling MA, Loyd D, Sansbury N, et al. Effects of shortterm VNS therapy on fos expression in rat brain nuclei. Presented at the 59th An-nual Scientific Convention of The Society of Biological Psychiatry; May 15–17, 2003; San Francisco, CA.
- Sjögren MJ, Hellstrom PT, Jonsson MA, et al. Cognition-enhancing effect of vagus nerve stimulation in patients with Alzheimer's disease: a pilot study. J Clin Psychiatry 2002;63:972–980
- Merrill CA, Jonsson MAG, Minthon L, Ejnell H, Silander HC, Blennow K, Karlsson M, Nordlund A, Rolstad S, Warkentin S, Menachem E, Sjögren MJC. Vagus Nerve Stimulation in Patients With Alzheimer's Disease: Additional Follow-Up Results of a Pilot Study Through 1 Year. J Clin Psychiatry 2006; 67:1171–1178.
- Tulgar M, Bilgin S, Yildirim A. The Human Body's Own Language to be Considered for Safe and Effective Neurostimulation. Neurol Ther. 2012; 9;1:2-7

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# Study of Matrix Gla Protein G-7A and T-138C Gene Polymorphisms in Patients with Type 2 Diabetes Mellitus

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#### Abstract

**Objectives:** Type 2 diabetes mellitus is a major risk factor for aortic calcifications and cardiovascular diseases. Gamma-carboxyglutamic acid (G-7A) protein has a significant role in control of the process of calcification. The purpose of this study was to investigate the role of G-7A and T-138C gene polymorphisms in development of aortic calcification and cardiovascular disease in patients with type 2 diabetes mellitus.

**Material and Methods:** The study included 120 patients with type 2 diabetes mellitus and 134 control group. G-7A and T-138C gene polymorphisms were identified using polymerase chain reaction and followed by restriction fragment length polymorphism methods.

**Results:** The G-7A genotype distribution in patients with type 2 diabetes mellitus AA=10.8%, GA=41.7% and GG=47.5% did not significantly differ from those in control group AA=15.7%, GA=48.5% and GG=35.8% (P=0.146). The T-138C genotype distribution in patients with type 2 diabetes mellitus CC=8.4%, CT=40.8% and TT=50.8% were also not significantly different from those in control group CC=3.7%, CT=39.6% and TT=56.7% (P=0.259). On the other hand; age, fasting blood glucose, cholesterol, high density lipoprotein cholesterol as expected were significantly differed between the patient-control groups (p<0.05).

**Conclusion:** This patient-control study shown that G-7A and T-138C gene polymorphisms of gamma-carboxyglutamic acid protein are not genetic risk factors for type 2 diabetes mellitus

**Key words:** Gamma-carboxyglutamic acid protein, G-7A gene polymorphism, restriction fragment length polymorphism, T-138C gene polymorphism, Type 2 diabetes mellitus

#### Introduction

Diabetes mellitus is an important health problem due to the illness itself and its life threatening complications. Type 2 diabetes mellitus is a disease characterized by insulin resistance and cell failure leading to elevated blood glucose levels (1). Aortic calcifications and cardiovascular diseases (CVD) are an important factor in the pathophysiology of type 2 diabetes mellitus (2).

In various tissues, tissue calcification is expected to be high due to high extracellular fluid concentrations of calcium and phosphate ions (3). Gamma-carboxyglutamic acid protein (MGP), which is vitamin K dependent calcium/phosphate binder protein and as the first protein known to act as a calcification inhibitor in vivo, takes control of the process of calcification (4, 5). The human MGP gene is located on the short arm of chromosome 12 and comprises 5 exons (12p12.3). This gene has 4 transcripts. We were taken as a reference MGP-004 in our study. (Transcript ID: ENST00000228938).

(http://www.ensembl.org/Homo\_sapiens/Tra nscript/Summary?db=core;g=ENSG00000111341;r=1 2:14881181-14885926;t=ENST00000228938). In vitro studies suggest that the gene encoding MGP has several single nucleotide polymorphisms (SNPs) in its promoter and coding regions (5). We selected two common SNPs on the MGP gene promoter

G-7A (http://www.ncbi.nlm.nih.gov/projects/ SNP/snp\_ref.cgi?rs=1800801)

and

T-138C (http://www.ncbi.nlm.nih.gov/projects/ SNP/snp\_ref.cgi?rs=1800802).

G-7A gene polymorphism, located in the promoter region of the MGP gene and characterized by replacing of guanine with adenine (G/A). T-138C gene polymorphism, has located in the promoter region of the MGP gene which is characterized by replacing of the thymine/cytosine (T/C). Various environmental and genetic risk factors have been considered to contribute in increasing the effects of polymorphisms such these as geographic, socioeconomic and racial features. In some studies, MGP gene polymorphisms were shown to be associated with arterial calcification or with CVD,

while in others, no association between MGP gene polymorphisms and arterial calcification or with CVD were found (6,7).

The purpose of this study was to investigate G-7A and T-138C gene polymorphisms of gammacarboxyglutamic acid protein in patients with type 2 diabetes mellitus and the possible role of these genes in the development of aortic calcification and CVD.

#### Material and method

The study included 120 patients with type 2 diabetes mellitus (55,8% men and 44,2% women) and 134 control group (45,5% men and 54,5% women). The mean age of the patients were ( $62.76\pm11.15$ ) and the mean age of the control group were (51.  $10\pm10.78$ ), respectively. Patients who had been diagnosed with type 2 diabetes mellitus were included to the y. In the control group type 2 diabetes mellitus, ischemic heart disease (IHD), peripheral arterial disease (PAD), stroke and any chronic illness patients were not included in this study. All participants gave informed consent that was approved by the local ethics committee.

#### **DNA** isolation

Deoxyribonucleic acid (DNA) was isolated from peripheral blood, containing ethylene-diamine tetra-acetic acid (EDTA) as an anticoagulant, by e.Z.N.A. (EaZy Nucleic Acid Isolation) blood DNA kits (BOGA, Istanbul, Turkey). DNA purity and quality were assessed by absorbance values in spectrophotometer (Shimadzu UV-1208). The ratio of optical density (O.D.) at 260 nm to O.D. at 280 nm was found about 1.8 for all samples. DNA bands were observed by ethidium bromide-stained 0.8 % agarose gel electrophoresis (Figure 1).



**Figure 1.** The ethidium-bromide stained 0.8% agarose gel of representative DNA samples.

#### Genotyping of MGP G-7A gene polymorphism

To determine the MGP G-7A gene polymorphism of the Type 2 diabetic and the control groups, a genomic DNA fragment on promoter region of the MGP gene was amplified by polymerase chain reaction (PCR) in a  $25\mu$ l PCR reaction mixture containing 100 ng of DNA, dNTP (0.2 mM of each), upstream 5'-CTAGTTCAGTGCCAACCCTTCCCCACC-3' and downstream 5'- TAGCAGCAGTAGGGAGAGAGAGGCTCCCA-3' primers (3,5 pmol), 1XTaq Buffer (10 mM Tris-HCl, pH 8.3, 50 mM KCL), 2 mM MgCl2 and 1.25 U of Taq DNA polymerase (Fermentas Life Sciences).

Amplification were performed with 3 minute of denaturation at 94°C, followed by 30 cycles with denaturation for 30 second at 94°C, annealing for 1 minute at 64°C, and extension for 1 minute at 72°C, followed by 5 minute of extension at 72°C (8). The PCR products were electrophoresed in 2 % agarose gels, and visualized under UV light Trans-illumination (ETX-F26.M) by ethidium bromide staining. 5 µl of PCR product were digested with 5U of the restriction enzyme NcoI in 1XM Buffer (10 mM Tris-HCl pH 7.5, 10 mM MgCl2, 1 mM Dithiothreitol and 50 mM NaCl) for 3 hours at 37°C. The restriction digest products were visualized by UV light transillumination (ETX-F26.M) after electrophoresis on a 3 % agarose gel and ethidium bromide staining (Figure 2).



**Figure 2.** Ethidium bromide stained 3% agarose gel of representative PCR digested products of G-7A gene polymorphism shows the AA genotype (lane 1), the GA (lane 6, 7) and the GG genotype (lane 2, 3, 4 and 5); lane 100 bp is a size marker.

#### Genotyping of MGP T-138C gene polymorphism

MGP T-138C gene polymorphism was identified with PCR and followed by restriction fragment length polymorphism (RFLP) with the restriction enzyme BsrSI. 25µl PCR reaction mixture containing 200 ng, dNTP (0.2 mM of each), upstream 5'-AAGCATACGATGGCCAAAACTTCTGCA-3' and downstream 5'-GAACTAGCATTGGAACTTTTCCCAACC-3' primers (3 pmol), 1XTaq Buffer (75 mM Tris-HCl pH 8.8, 20 mM (NH4)2SO4, 0.01% Tween 20), 2.5 mM MgCl2 and 1.25 U of Taq DNA polymerase.

Amplification were performed with denaturation for 3 minute at 94°C, followed by 30 cycles with denaturation for 30 second at 94°C, annealing for 1 minute at 57°C, and extension for 1 minute at 72°C, followed by 5 minute of extension at 72°C (8). The PCR products were electrophoresed in 2 % agarose gel, and visualized under UV light transillumination (ETX-F26.M) by ethidium bromide staining. 5  $\mu$ l of PCR product were digested with 5U

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# Original Article

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# **Does Attending Clinical Wards Increase Nasal Carriage of Staphylococcus Aureus Among Medical Students?**

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#### Abstract

**Objectives:** The carriage rates of S. aureus among medical students were reported to notably increase after they attended medical wards. We aimed to investigate the nasal colonization rates of S. aureus in medical students and assess whether attending clinical wards has an impact on nasal carriage rates of S. aureus.

**Methods:** Medical students from Namik Kemal University were divided into two groups, 'preclinical' and 'clinical'. Nasal swabs from both anterior nares were obtained and transferred to the laboratory for culturing.

Results: Community-acquired methicilline-susceptible S. aureus (MSSA) was detected in 2 out of 75 students (2.66%) in the preclinical group. In the clinical group, 3 out of 75 students (4%) were carriers for community-acquired MSSA. No statistically significant difference in terms of MSSA carriage was observed between the groups (p>0.05, p=0.649). Neither community-acquired methicilline-resistant S. aureus nor hospital-acquired S. aureus was detected.

**Conclusions:** The results of the study indicated that attending clinical wards did not increase nasal carriage rates in medical school students. Low carriage rates may be explained by strict adherence to the rules of disinfection and antisepsis by the medical students attending clinical wards and the comprehensive hygienic precautions taken by the infection control committee of the hospital.

Keywords: Nasal carriage, Staphylococcus aureus, medical students, clinical wards

#### Introduction

Staphylococcus aureus is a gram positive bacterium that can either display a commensal type of living in human hosts or cause significant morbidity and mortality by leading to a variety of infections. Severe S. aureus infections involve the skin, soft tissues, cardiovascular system, and central nervous system and can reportedly cause mortality rates ranging from 8–49% [1]. In its commensally form, S. aureus may survive by binding to skin and mucosal surfaces. The most commonly reported site of colonization is the anterior nares. The bacterium was also found to be localized in the pharynx, vagina, axilla, perineal region, and gastrointestinal tract [2].

Prevention and treatment of nosocomial infections associated with S. aureus are considered to be major public health concerns [3]. A significant source of nosocomial infection was proposed to be the nasal carriage of S. aureus among the health care personnel. The carriage rates were found to vary from 20-40% [4,5].

In the literature, the carriage rates of the pathogen among medical students were reported to notably increase after attending medical wards.

It was suggested that after attending medical wards, medical students become both victims of *S. aureus* colonization and significant sources for the

further spread of the pathogen [6]. Conversely, other authors pointed out that there was no association between attending medical wards and increased nasal carriage rates of S. aureus among medical students [7,8].

In the present study, we aimed to define nasal carriage rates of S. aureus in medical students and investigate whether attending clinical wards impacts nasal carriage rates of the pathogen in this population. The implications of these findings were also discussed using the existing literature

#### **Material and Methods**

The study group was comprised of medical school students from Namik Kemal University who volunteered to be enrolled in the study. Approval of the Local Ethics Committee from the same institute and students' informed consent were obtained. The students were divided into two groups. The first group was called the 'preclinical' and was composed of first, second, and third class students who had not previously attended clinical wards.

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|                                       |                                  | Students       |             |        |
|---------------------------------------|----------------------------------|----------------|-------------|--------|
|                                       |                                  | Preclinical G. | Clinical G. | Total  |
| <i>S.</i> a <i>ureus</i> colonization | Number of S. aureus (+) students | 2              | 3           | 5      |
|                                       | %                                | 2.7 %          | 4.0%        | 3.3 %  |
|                                       | Number of S. aureus (-) students | 73             | 72          | 145    |
|                                       | %                                | 97.3 %         | 96 %        | 96.7 % |
|                                       | Total number                     | 75             | 75          | 150    |

| Tabla 1  | Overall C    | aurous colonization | rates in the prealinies | and alinical groups    |
|----------|--------------|---------------------|-------------------------|------------------------|
| I adie i | L Overall S. | aureus colonization | rates in the preclinica | li and clinical groups |

The second group was composed of fourth, fifth, and sixth class students who regularly attended clinical wards and were therefore called the 'clinical' group. Students with a history of medical conditions requiring hospitalization or antibiotic use within the last sixth months were excluded. All students were examined by the same otorhinolaryngologist. Following nasal endoscopic examinations, students with acute respiratory tract infections (e.g., swelling, hyperemia, or purulent secretions) were also excluded. Seventy-five out of 82 students in the preclinical group and 75 out of 85 students in the clinical group fulfilled the criteria and were included in the study. Nasal swabs from both anterior nares were obtained and transferred to a microbiology laboratory in a transport medium for culturing. Specimens were inoculated on 5% sheep blood agar using the striking method and incubated at 35-37°C for 18-20 hours. Suspected colonies were yellow pigmented and betahemolytic; they were identified using conventional and semi-automatic methods. Methicillin resistance was evaluated according to CLSI documents [9]

#### **Statistical Analyses**

Data analyses were conducted using SPSS (Version 18.0., Chicago). Results were analyzed using the  $\chi 2$  test

#### Results

Carriage of community-acquired methicillinesusceptible S. aureus (MSSA) was detected in 2 out of 75 students (2.66%) in the preclinical group. In the clinical group, 3 out of 75 students (4%) were found to be carriers for community-acquired MSSA (Table 1). A comparison of the two groups revealed no statistically significant difference in terms of S. aureus carriage (p>0.05, p=0.649). Community-acquired MSSA was not detected in both groups. Hospitalacquired S. aureus (either susceptible or resistant to methicilline) was not detected.

#### Discussion

The colonization of S. aureus in health care personnel is a potentially life threatening challenge, especially for patients who are diabetic or receiving hemodialysis.

Patients staying in intensive care units and patients with cardiac or other co-morbid disorders, including immune deficiency and cystic fibrosis, were also reported to be at risk for S. aureus associated nosocomial infections [10]. Incidences of nosocomially acquired *S. aureus* infections were found to be increased in certain conditions. A history of surgical operations, hospitalization in an intensive care unit, prolonged antibiotic therapy or hospitalization, or intimate contact with carriers or previously infected patients or health care personnel are all important risk factors for increasing the likelihood of having a nosocomial S. aureus infection [11].

Among these factors, nasal carriage of SA in health care personnel was particularly found to have a significant impact on the dissemination of the disease [12]. Medical students, especially interns attending clinical wards, were proposed to contribute to the spread of S. aureus from health care settings to the community [13]. Aside from being suspected vectors for the transmission of the pathogen, medical students were also considered victims. During clinical training, the risk of conversion from a non-carrier state to carrier state was found to be increased for medical students. In a follow up study, it was concluded that attending clinical rotations in a hospital setting could increase the prevalence of asymptomatic nasal carriage of S. aureus among medical students [6].

A previous study investigated the prevalence rate and risk factors of nasal carriage of S. aureus in medical students in Turkey. It was reported that the pathogen was isolated in 50 out of 179 students (27.9%). They concluded that increasing clinical exposure led to an increase in S. aureus carriage rates. They also pointed out that although statistically insignificant, the number of resistant cases increased in proportion to clinical exposure [14].

In another study, 38.6% of the medical students investigated were not aware of a hand hygiene protocol, and more than 30% of the students ignored that protocol. Their results revealed that of the medical students they investigated, 39.3% were colonized by SA and 2.1% were colonized by methicilline-resistant S. aureus. They pointed out the importance of including medical students in hospital infection control programs and suggested that hand hygiene training should be given to students prior to attending clinical wards [15].

Despite convincing evidence that being in close contact with patients, other health care personnel, and the hospital environment may increase the likelihood of the colonization of SA in medical students, some authors still insist that attending clinical wards does not increase S. aureus carriage rates. Nasal carriage of S. aureus in medical students was reported to be very low in a study by Trépanier et al [16]. Furthermore, Slifka et al. analyzed whether exposure to patients created a risk of S. aureus carriage for American medical students in their study. They concluded that patient exposure was not an occupational hazard for students attending clinical wards [17]. Another study proposed that the carriage of S. aureus was not affected by one to two years of clinical exposure in the hospital [18].

In a recent study, low nasal colonization rates of S. aureus were reported among clinically exposed medical students at the University of Vienna. They attributed this finding to their well-established hygienic precautions and comparably low circulation of resistant bacteria [8].

The results of our study also demonstrated that nasal carriage rates of S. aureus in both preclinical and clinical medical students at our university were low. In addition, no statistically significant difference was observed between preclinical and clinical groups. Attending clinical wards seemed to have no significant impact on nasal carriage rates of medical students. Furthermore, the isolated pathogens in both groups were community acquired and not hospital acquired. This finding also indicated that nosocomial transmission of hospital-acquired S. aureus was not a matter of concern for medical students attending clinical wards. Our findings were consistent with the findings of previous studies that proposed that there was no association between attending clinical wards and increased nasal carriage rates of SA in medical students [8,16-18].

#### Conclusion

The findings of our study indicated that attending clinical wards did not increase the prevalence of nasal carriage of SA in medical school students. The authors of this study are of the opinion that low carriage rates can be explained by both the strict hygiene protocols applied in the hospital and the comprehensive counseling of medical staff, including medical students. These measures may protect novice clinical students and prevent the creation of new sources for the nosocomial transmission of SA.

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consent was obtained from the individuals who were included into the study.

#### References

- 1. Bischoff WE, Wallis ML, Tucker KB, Reboussin BA, Sherertz RJ. Staphylococcus aureus nasal carriage in a student community: prevalence, clonal relationships, and risk factors. Infect Control Hosp Epidemiol. 2004; 25: 485-491.
- Wertheim HF, Melles DC, Vos MC, van Leeuwen W, van Belkum A, Verbrugh HA, Nouwen JL. The role of nasal carriage in Staphylococcus aureus infections. Lancet Infect Dis. 2005; 5: 751-762.
- Fredheim EG, Flægstad T, Askarian F, Klingenberg C. Colonisation and interaction between S. epidermidis and S. aureus in the nose and throat of healthy adolescents. Eur J Clin Microbiol Infect Dis. 2015 Jan;34:123-9. doi: 10.1007/s10096-014-2197-5. Epub 2014 Aug 1.
- Krespi YP, Kizhner V. Laser-assisted nasal decolonization of Staphylococcus aureus, including methicillin-resistant Staphylococcus aureus. Am J Otolaryngol. 2012 Sep-Oct;33:572-5. doi: 10.1016/j.amjoto.2012.02.002. Epub 2012 Apr 12.
- Steed LL, Costello J, Lohia S, Jones T, Spannhake EW, Nguyen S. Reduction of nasal Staphylococcus aureus carriage in health care professionals by treatment with a nonantibiotic, alcohol-based nasal antiseptic. Am J Infect Control. 2014; 42: 841-846. doi: 10.1016/j.ajic.2014.04.008.
- Treesirichod A, Hantagool S, Prommalikit O. Nasal carriage and antimicrobial susceptibility of Staphylococcus aureus among medical students at the HRH Princess Maha Chakri Sirindhorn Medical Center, Thailand: a follow-up study. J Infect Public Health. 2014; 7: 205-229. doi: 10.1016/j.jiph.2013.12.003.
- Syafinaz AM, Nur Ain NZ, Nadzirahi SN, Fatimah JS, Shahram A, Nasir MD. Staphylococcus aureus Nasal Carriers Among Medical Students in A Medical School. Med J Malaysia. 2012; 67: 636-638.
- Gualdoni GA, Lingscheid T, Tobudic S, Burgmann H. Low nasal carriage of drug-resistant bacteria among medical students in Vienna. GMS Krankenhhyg Interdiszip. 2012;7: Doc04. doi: 10.3205/dgkh000188. Epub 2012 Apr 4.
- CLSI. Surveillance for Methicillin-Resistant Staphylococcus aureus: Principles, Practices, and Challenges; A Report. CLSI document X07-R. Wayne, PA: Clinical and Laboratory Standards Institute; 2010.
- Long SW, Beres SB, Olsen RJ, Musser JM. Absence of Patient-to-Patient Intrahospital Transmission of Staphylococcus aureus as Determined by Whole-Genome Sequencing. MBio. 2014; 5: e01692-01714. doi: 10.1128/mBio.01692-14.

- Salgado CD, Farr BM, Calfee DP. Community-acquired methicillin-resistant Staphylococcus aureus: a metaanalysis of prevalence and risk factors. Clin Infect Dis. 2003; 36: 131-139.
- Shibabaw A, Abebe T, Mihret A. Nasal carriage rate of methicillin resistant Staphylococcus aureus among Dessie Referral Hospital Health Care Workers; Dessie, Northeast Ethiopia. Antimicrob Resist Infect Control. 2013; 2: 25. doi: 10.1186/2047-2994-2-25.
- Ma XX, Sun DD, Wang S, Wang ML, Li M, Shang H, Wang EH, Luo EJ. Nasal carriage of methicillin-resistant Staphylococcus aureus among preclinical medical students: epidemiologic and molecular characteristics of methicillinresistant S. aureus clones. Diagn Microbiol Infect Dis. 2011; 70: 22-30. doi: 10.1016/j.diagmicrobio.2010.12.004.
- Güçlü E, Yavuz T, Tokmak A, Behçet M, Karali E, Oztürk O, Egeli E. Nasal carriage of pathogenic bacteria in medical students: effects of clinic exposure on prevalence and antibiotic susceptibility. Eur Arch Otorhinolaryngol. 2007; 264: 85-88.

- López-Aguilera S, Goñi-Yeste Mdel M, Barrado L, González-Rodríguez-Salinas MC, Otero JR, Chaves F. [Staphylococcus aureus nasal colonization in medical students: imprtance in nosocomial transmission]. [Article in Spanish] Enferm Infecc Microbiol Clin. 2013; 31: 500-505. doi: 10.1016/j.eimc.2012.12.005.
- Trépanier P, Tremblay C, Ruest A. Methicillin-resistant Staphylococcus aureus colonization among medical residents. Can J Infect Dis Med Microbiol. 2013; 24: e39-41.
- Slifka KJ, Nettleman MD, Dybas L, Stein GE. Is acquisition of methicillin-resistant Staphylococcus aureus an occupational hazard for medical students? Clin Infect Dis. 2009; 49: 482-483. doi: 10.1086/600824.
- Chen CS, Chen CY, Huang YC. Nasal carriage rate and molecular epidemiology of methicillin-resistant Staphylococcus aureus among medical students at a Taiwanese university. Int J Infect Dis. 2012; 16: e799-803. doi: 10.1016/j.ijid.2012.07.004.

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of the restriction enzyme BsrSI in 1XM Buffer (10 mM Tris-HCl pH 7.5, 10 mM MgCl2, 1 mM Dithiothreitol and 50 mM NaCl) for 3 hours at 65°C. When mutant allele (Thymine), digested with BsrSI that yield two fragments 118 bp and 24 bp, whereas a wild allele (Cytosine) at nucleotide position 138, had no cutting site for BsrSI, so that the 142 bp PCR product was not cleaved into 118 bp and 24 bp fragments (8). The restriction digest products were electrophoresed in 3 % agarose gel, and visualized under UV light trans-illumination (ETX-F26.M) by ethidium bromide staining (Figure 3).



Figure 3. Ethidium bromide-stained 3% agarose gel of representative PCR digested products of T-138C gene polymorphism shows the TT genotype (118 bp and 24 bp lane 4,5), the CC genotype (142 bp,lane 2) and the CT genotype (142 bp, 118 bp and 24 bp, lane 1,3 and 6); lane 100 bp is a size marker.

#### **Statistical Analyses**

Genotype distribution of G-7A and T-138C of Type 2 diabetic patients and control groups were evaluated by Chi-square test ( $\chi$ 2 test). Age, fasting blood glucose (FBG), triglyceride (TG), cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), systolic blood pressure (SBP) and diastolic blood pressure (DBP) were examined by independent student's t-test. The all values are represented as mean ± standard deviation (SD). Significance was defined as p<0.05

#### Results

Age, FBG, TG, cholesterol, HDL-C, LDL-C, SBP and DBP of 120 patients with type 2 diabetes mellitus and 134 controls are presented in Table 1. No significant differences were detected between type 2 diabetes mellitus and control groups for TG, SBP, DBP (p>0.05). On the other hand, age, FBG, cholesterol, HDL-C and LDL-C as expected were significantly differed between the patient-control groups (p<0.05).

| Table 1. The clinical  | characteristics of | of control | and type 2 |
|------------------------|--------------------|------------|------------|
| diabetes mellitus grou | ups                |            |            |

|                      | Control         | Type 2 DM       |         |
|----------------------|-----------------|-----------------|---------|
|                      | Group           | Group           | Р       |
|                      | (n=134)         | (n=120)         |         |
| Mean Age (Year)      | 51.1±11         | 62.8±11         | 0.001   |
| FBG (mg/dl)          | 98.5±12         | 169,4±64        | 0.001   |
| TG (mg/dl)           | 147.4±91        | 148.2±103       | ns      |
| CH (mg/dl)           | 210.9±43        | 179.8±52        | 0.001   |
| HDL- C (mg/dl)       | 41.2±11         | 33.8±18         | 0.001   |
| LDL- C (mg/dl)       | 130.4±33        | 115.7±42        | 0.001   |
| SBP (mmHg)           | 123.4±14        | 125.7±23        | ns      |
| DBP (mmHg)           | 76.5±11         | 76.1±13         | ns      |
| DM; Diabetes Mellit  | us NS; not si   | gnificant, FBG  | Fasting |
| blood glucose, TG;   | Triglyceride;   | CH; Cholestero  | l, HDL- |
| C; High density lip  | poprotein cho   | olesterol, LDL- | C; Low  |
| density lipoprotein  | cholesterol,    | SBP; Systolic   | e blood |
| pressure, DBP; Diast | olic blood pres | ssure           |         |

Genotype frequencies for the G-7A gene polymorphism are presented in Table 2. The overall frequencies of the genotypes AA, GA and GG in type 2 diabetes mellitus group were not significantly differ from those in control group (p=0.146).

 Table 2. Genotype frequencies for the G-7A gene of type 2 diabetes mellitus and control groups.

|       | Group     |           |           |       |
|-------|-----------|-----------|-----------|-------|
|       | Patient   | Control   | Total     | Р     |
|       | (n=120)   | (n=134)   |           |       |
| A A   | 13        | 21        | 34        |       |
| AA    | (% 10.8)  | (% 15.7)  | (% 13.4)  |       |
| GA    | 50        | 65        | 115       |       |
| UA    | (% 41.7)  | (% 48.5)  | (% 45.3)  | 0.146 |
| GG    | 57        | 48        | 105       |       |
| 00    | (% 47.5)  | (% 35.8)  | (% 41.3)  |       |
| Total | 120       | 134       | 254       |       |
| Total | (% 100.0) | (% 100.0) | (% 100.0) |       |

Genotype frequencies for the T-138C gene polymorphism are presented in Table 3. No differences were detected in alleles (C or T) frequency between type 2 diabetes mellitus and control groups Our findings, in this patient-control study populating, indicate that the G-7A and the T-138C gene polymorphisms were not genetic risk factors for type 2 diabetes mellitus

| Table 3. | Genotype | frequencies   | for the | T-138C   | gene of type |
|----------|----------|---------------|---------|----------|--------------|
|          | 2 diabet | es mellitus a | nd cont | rol grou | 20           |

|       | 2 diabetes i | nenntus and et | million groups |       |
|-------|--------------|----------------|----------------|-------|
|       | (            | Group          |                |       |
|       | Patient      | Control        | Total          | Р     |
|       | (n=120)      | (n=134)        |                |       |
| CC    | 10           | 5              | 15             |       |
| ιι    | (% 8.4)      | (% 3.7)        | (% 5.9)        |       |
| СТ    | 49           | 53             | 102            |       |
| CI    | (% 40.8)     | (% 39.6)       | (% 40.2)       | 0.259 |
| тт    | 61           | 76             | 137            |       |
| 11    | (% 50.8)     | (% 56.7)       | (% 53.9)       |       |
| Total | 120          | 134            | 254            |       |
| Total | (% 100.0)    | (% 100.0)      | (% 100.0)      |       |

#### Discussion

Type 2 diabetes mellitus is characterized by two discrete pathophysiological defects: impaired pancreatic  $\beta$ -cell function, and insulin resistance in muscle, fat and liver. The prevalence of type 2 diabetes mellitus is increasing dramatically in the world (9). CVD is by far the most common and serious complication of type 2 diabetes mellitus. Increased inflammatory response as lead to insulin resistance and endothelial dysfunction contributes to cardiovascular complications (10).

Individuals with type 2 diabetes mellitus are at least twice as likely to have coronary artery disease compared to non-diabetic individuals, and CVD accounts for 80 % of mortality in type 2 diabetes mellitus patients (11). Increased vascular calcification is associated with an increased risk of cardiovascular events. Type 2 diabetes mellitus is a significant independent risk factor for increased vascular calcification (12). MGP, which is Vitamin K dependent protein that is expressed by smooth muscle cells, fibroblasts, chondrocytes and endothelial cells in a variety of tissues, plays a role in bone metabolism and vascular calcification (13, 14). Several studies identified various candidate genes involved in vascular calcification. MGP plays a key role in the preventing of vascular deposition of the calcium in the matrix (4, 5, 15).

Many researchers investigated the relationship of vascular calcification and the common gene polymorphisms of the MGP. Two of the most intensively investigated genetic polymorphisms are the G-7A and the T-138C gene polymorphisms. The frequency of the G-7A and the T-138C genotypes and their possible associations with the risk of vascular calcification has been investigated in different populations. Studies of the association of G-7A and the T-138C gene polymorphisms with vascular calcification reported both positive (16) and negative (7) results.

In Ukrainian population, the allelic AA promoter variant of MGP G-7A polymorphism was associated with acute coronary syndrome in males but not in females. However, there was not found any relationship between the T-138C gene polymorphisms with acute coronary syndrome (17). Ataman et al. showed an association for G-7A polymorphism in Ukraine's population. However, they reported that other polymorphic sites such as T-138C do not relate to ischemic stroke (18).

Afshin et al. discovered that the MGP gene starts with the T-138C polymorphism which influences gene expression level; CC genotype MGP showing the highest levels in blood serum followed by CT and TT. The C genotype (CT+CC) tended to show a higher calcification factor than the TT genotype (8).

In another study, associations between MGP single nucleotide polymorphisms and coronary artery calcification in older men and women of European descent from Massachusetts (USA) were examined.

Various methods of analysis revealed that in men, homozygous carriers of the minor allele of G-7A, T-138C and Thr83 $\rightarrow$ Ala polymorphisms were associated with a decreased level of coronary artery calcification relative to major allele carriers. This association was not found in women. In addition, genetic variation in MGP was shown to associate with serum MGP concentrations, but there were no association between serum MGP levels and coronary artery calcification (19).

In conclusion, our data do not support any association between G-7A and T-138C gene polymorphisms and type 2 diabetes mellitus disease in this case-control study and is not a determinant for cardiovascular risk factors or complications associated with type 2 diabetes mellitus.

#### Conclusion

The determination of the decline in the severity of ED in overall and the complete improvement of ED in some patients after CABS revealed that CABS had positive impacts on EF in patients with CAD. Furthermore, the demonstration of a significant improvement in the majority of subgroups of SF in IIEF questionnaire was concluded that CABS had also an encompassing positive impact on almost all domains of sexual function in patients with CAD. On the other hand, the analysis of possible influences of the comorbidities on the impact of CABS on sexual function showed that while the lonely presence of diabetes mellitus did not significantly affected the impact of CABS on sexual function, the sexual function most improved in both diabetic and hypertensive cases after CABS. We supposed that these results need to be confirmed by prospective and randomized trials in greater series.

#### **Conflict of Interest**

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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#### References

- Nowotny K, Jung T, Höhn A, Weber D, Grune T. Advanced Glycation End Products and Oxidative Stress in Type 2 Diabetes Mellitus. Biomolecules 2015;5:194-222.
- Esen B, Daşdemir S, Çinçin Z, Özdemircan A, Çakmakoğlu B. The Analysis Of Osteoprotegerin (Opg) Protein T950c Gene Polymorphism in Diabetes. İ. Ü. Deneysel Tıp Araştırma Enstitüsü Dergisi. 2011;l(1):40-46.
- Metabolik Kemik Hastalıkları. Türkiye Endokrinoloji ve Metabolizma Derneği Dergisi. 2013;19

- 4. Tunon-Le Poultel D, Cannata-Andia J. B, Roman-Garcia P, et al. Association of matrix Gla protein gene functional polymorphisms with loss of bone mineral density and progression of aortic calcification. Osteoporos Int 2014;25: 1237–1246.
- Yoshikawa K, Abe H, Tominaga T, et al. Polymorphism in the human matrix Gla protein gene is associated with the progression of vascular calcification in maintenance hemodialysis patients. Clin Exp Nephrol 2013;17:882–889.
- Andrea E. Cassidy B, Lawrence F, et al. Matrix Gla Protein Gene Polymorphism Is Associated With Increased Coronary Artery Calcification Progression. Arterioscler Thromb Vasc Biol. 2013;33:645-651.
- Najafi M, Roustazadeh A, Amirfarhangi A, Kazemi B. Matrix Gla protein (MGP) promoter polymorphic variants and its serum level in stenosis of coronary artery. Mol Biol Rep 2014;41:1779–1786.
- Farzaneh-Far A, Davies J.D, Braam L, et al. A Polymorphism of th Human Matrix \_-Carboxyglutamic Acid Protein Promoter Alters Binding of an Activating Protein-1 Complex and Is Associated with Altered Transcription and Serum Levels. The Journal Of Biological Chemistry © 2001 by The American Society for Biochemistry and Molecular Biology, Inc. 2001;276(35),32466–32473.
- Keshavarz P, Inoue H, Sakamoto Y, et al. No evidence for association of the ENPP1 (PC-1) K121Q variant with risk of type 2 diabetes in a Japanese population. J Hum Genet 2006;51:559–566.
- 10. Keskin Ö. Diabetes Mellitus ve Kardiyovasküler Komplikasyonlar. Kafkas J Med Sci 2011;1(2):81–85.
- Emral R. Diyabetik Hastalardaki Kardiyovasküler Etkilenimler. Türk Farmakoloji Derneği 21. Ulusal Farmakoloji Kongresi Bildiri Özet Kitabı. 2011;40-42.

- Demer L.L, Tintut Y. Vascular Calcification Pathobiology of a Multifaceted Disease. Circulation. 2008;117:2938-2948.
- Liabeuf S, Olivier B, Vemeer C, et al. Vascular calcification in patients with type 2 diabetes: the involvement of matrix Gla protein. Liabeuf et al. Cardiovascular Diabetology 2014;13-85.
- Tavridou A, Petridis I, Vasileiadis M. Association of VKORC1 S1639 G > A polymorphism with carotid intima-media thickness in type 2 diabetes mellitus. Diabetes Research and Clinical Practice 2011;94:236-241.
- 15. Wang Y, Chen J,Zhang Y, et al. Common Genetic Variants of MGP Are Associated With Calcification on the Arterial Wall but Not With Calcification Present in the Atherosclerotic Plaques. Circ Cardiovasc Genet. 2013;6:271-278.
- 16. Herrmann S.M, Whatling C, Brand E, et al. Polymorphisms of the Human Matrix Gla Protein (MGP) Gene, Vascular Calcification, and Myocardial Infarction. Arterioscler Thromb Vasc Biol. 2000;20:2386-2393.
- 17. Garbusova V.Y, Gurianova V.L, Stroy D. A, et al. Allelic polymorphism of matrix Gla promoter  $G-7\rightarrow A$ , but not  $T-138\rightarrow C$  nor exon 4 Thr83 $\rightarrow A$ la, is positively associated with acute coronary syndrome in the Ukrainian population. Exp Clin Cardiol 2012;17(1):30-33.
- Ataman A. V, Garbusova V. Y, Ataman Y. A, et al. Investigation of the MGP promoter and exon 4 polymorphisms in patients with ischemic stroke in the Ukrainian population. Journal of Cell and Molecular Biology 2012;10(1):19-26.
- Crosier M. D, Booth S. L, Peter I, et al. Matrix Gla Protein Are Associated with Coronary Artery Calcification in Men. J Nutr Sci Vitaminol, 2009;55:59-65.

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# Is myomectomy safe during cesarean section in large myomas?

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#### Abstract

**Objectives:** The rate of cesarean section, gravida and pregnancy age have been gradually increasing. Thus the rates of myoma have increased during cesarean. But it may difficult to make decision about myomectomy during cesarean especially large myomas because of complications. Therefore, several obstetricians do not recommend cesarean myomectomy, except in cases of small and pedunculated uterine fibroids that do not result in complications. The aim of this study to evaluate safety of myomectomy during emergency cesarean section in large myomas

Material and Methods: We examined retrospectively the results of 190 patients who underwent cesarean myomectomy in two non-tertiary hospitals in southeastern Anatolia. We compared complications, hemoglobin change, length of stay in hospital and blood transfusion according to diameter of myoma. Complication and blood transfusion rates in relation to the diameter of uterine fibroids were evaluated using Fisher's exact test. Changes in hemoglobin levels from baseline value and length of stay in hospital were compared using the Mann Whitney U test. Significance level was set at P <0.01 and 0.05

Results: No significant association was found between complication rates and diameter of the fibroids (P=0.633). Similarly, no significant association was observed between the type of uterine fibroids and complication rates (P=1.000). We also found no significant difference among the changes in hemoglobin levels, length of stay in hospital and transfusion (P=0.835, p=0.184, p=0.633).

**Conclusions:** Myomectomy is safe procedure at the emergency cesarean section in large myoma with attention. Key Words: Cesarean section; Complications; Myoma

Keywords: Cesarean section; Complications; Myoma

#### Introduction

The incidence of uterine fibroids during pregnancy ranges from 1.6% to 10.7% (1-4). The rate of cesarean sections, pregnancy age and gravida has been gradually increasing, and thus, the chances of an obstetrician encountering uterine fibroids during cesarean section have increased (5). However, a negative stance towards performing cesarean myomectomy has traditionally existed due to the risk of persistent bleeding that may require hysterectomy and cause increased postoperative morbidity (6-8). Therefore, several surgeons (9-11) do not recommend cesarean myomectomy, except in cases of small and pedunculated uterine fibroids that do not result in excessive bleeding in the myoma bed.

Cesarean myomectomy in large myoma is also still considered to be high risk for massive uterine hemorrhage, atony and peripartum hysterectomy. On the other hand, many retrospective and prospective studies (8,9,12-15) suggest that cesarean myomectomy can be performed safely and in a cost-effective manner in selected cases even in large myomas. In the southern region of Turkey, which has the highest population growth in the country, maternity hospitals predominantly treat obstetric patients.

In the present study, we reviewed the outcomes of cesarean myomectomy performed in patients complaining of pain who presented to the emergency gynecology department of two different centers. The safety of large cesarean myomectomy procedures was investigated

#### **Material and Methods**

This was retrospective study. We included pregnant women with myomas who delivered via emergency cesarean section at Batman and Diyarbakir Gynecology and Obstetrics Hospital between 2011 and 2014. All patients were initially evaluated in the emergency department. Diagnoses of uterine fibroid were established by ultrasonography performed prior to cesarean section or during surgery. Informed consent was obtained from all patients. Ages, gravid, parity, length of stay in hospital, gestational week, and reason for surgery indication were recorded for each patient. Laporotomy was made with Pfannenstiel incision. In all patients, the cesarean section was followed by myomectomy after closure of the lower uterine segment; hemostasis in the myoma bed was achieved by suturing.

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Myomectomies were performed using sharp dissection from the capsule with scissors. If major hemorrhage was observed during surgery, hemoglobin changes was assessed and blood transfusion was performed when required. Postoperative hemoglobin value was assessed 8 hours after surgery. Location, number, and diameter of uterine fibroids, as well as preoperative and postoperative hemoglobin values and development of complications, length of stay in hospital, blood transfusion rate were recorded. Complications were considered uterine atony and hemorrhage. Cases were assigned to one of two groups, according to the diameter of uterine fibroids (<5 or  $\geq$ 5 cm) (16). Development of complications, length of stay in hospital, transfusion and hemorrhage rates were evaluated in relation to the size and type of uterine fibroids. There was no need ethics committee approval because of retrospective design.

Statistical analyses were performed using the Number Cruncher Statistical System (NCSS) 2007 (Utah, USA). Complication and blood transfusion rates in relation to the diameter of uterine fibroids were evaluated using Fisher's exact test. Changes in hemoglobin levels from baseline value and differences between two groups, length of stay in hospital were compared between the two groups using the Mann Whitney U test, student t test and Paired Samples test. Significance level was set at P <0.01 and 0.05.

#### Results

Total of number births were 18412 in Batman State Hospital, 58308 in Diyarbakir State Hospital while the number of cesarean section were 5255 and 15945 respectively. The cesaraen section ratios were 28.54% and 27.34% respectively. Both hospitals are non-tertiary center. The mean age of patients was 30.2. The numbers of previous pregnancy were differenced from 0 to 9 while the mean value was 2.27. The gestational age were differenced from 32 to 42. Patients' characters are presented in Table 1.

Most of reasons for cesarean section were previous cesarean section. The reasons for cesarean section were maternal, fetal and labor related factors. Maternal reasons were considered as placental ablation, previous cesarean section, patients' request, myomectomized patients, placenta previa and preeclapsia. Fetal reasons were considered fetal distress, mal-presentation, twin pregnancy, umbilical cord prolapsus and oligohydroamnios. Labor related factors were non-progressive labor and cephalopelvic disproportion (Table 2).

Among all patients, 154 (81.1%) presented a solitary uterine fibroid, 31 (16.3%) had two fibroids, 2 (1.0%) had three fibroids, and 3 (1.6%) had four fibroids. In addition, 12.6% (24) presented uterine fibroids with the largest one measuring  $\geq$ 5 cm, and 166 (87.4%) showed uterine fibroids <5 cm. Of these patients, 86 (45.3%) presented intramural fibroids, and 104 (54.7%) had subserosal fibroids. Characteristics of uterine fibroids are presented in Table 3.

Among the 3 patients who developed hemorrhage, one presented an intramural fibroid measuring 7 cm in diameter, and 2 showed cornual fibroids measuring 4 and 5 cm in diameter, respectively. All of those patients required blood transfusion.

Eight patients who developed uterine atony also received blood transfusion. Ligation of the hypogastric artery was performed in only 3 patients who developed uterine atony; the rest of these patients were managed with uterine massage and oxytocin infusion. None of the patients required hysterectomy. Preoperative and postoperative hemoglobin changes are presented in table 4.

No significant association was found between complication rates and diameter of the fibroids (P=0.633) (Table 5). Similarly, no significant association was observed between the type of uterine fibroids and complication rates (P=1.000) (Table 5) or between the diameter of uterine fibroids and the mean changes in hemoglobin levels from baseline value (P=0.835) (Table 6). There were also no significant differences between two groups in terms of blood transfusion rate and length of stay in hospital (p=0.633 and p=0.183 respectively) (Table 6).

 Table 1. Patients' characteristics

|                                   | Min-Max | Mean±SD        |
|-----------------------------------|---------|----------------|
| Age (years)                       | 13–52   | 30.92±6.1<br>5 |
| Gravidity                         | 1-10    | 3.30±1.83      |
| Parity                            | 0–9     | 2.27±1.83      |
| Gestational week                  | 32–42   | 37.87±2.0<br>7 |
| Length of Hospital<br>Stay (days) | 1–6     | 2.14±0.66      |

| Table 2. Reason for cesarean section indication |     |      |  |
|---|-----|------|--|
|   | n   | %    |  |
| Fetal Reason                                    | 46  | 24,4 |  |
| Maternal Reasons                                | 126 | 66,2 |  |
| The reasons about labor                         | 18  | 09,4 |  |

**Table 3.** Characteristics of uterine fibroids

|                     |               | n (%)      |
|---------------------|---------------|------------|
| Type of Uterine     | Intramural    | 86 (45,3)  |
| Fibroids            | Subserosal    | 104 (54,7) |
| Diameter of Uterine | <5 cm         | 166 (87,4) |
| Fibroids            | ≥5 cm         | 24 (12,6)  |
|                     | Anterofundal  | 6 (3,2)    |
|                     | Anteroisthmic | 2 (1,1)    |
|                     | Anterior      | 57 (30)    |
| Location of Uterine | Fundal        | 64 (33,7)  |
| Fibroids            | Isthmus       | 14 (7,4)   |
|                     | Cornual       | 5 (2,6)    |
|                     | Posterior     | 40 (21,1)  |
|                     | Cervical      | 2 (1)      |
|                     |               |            |

 $\langle 0/ \rangle$ 

| *                           | <b>•</b> •                  | Diameter of Uterine Fibroid           |                   |                |
|-----------------------------|-----------------------------|---------------------------------------|-------------------|----------------|
|                             |                             | <5 cm (n=166)                         | ≥5cm (n=24)       | <sup>a</sup> p |
|                             |                             | Mean±SD                               | Mean±SD           |                |
| Preoperative H              | emoglobin                   | 11,76±1,39                            | 11,19±1,46        | 0,063          |
| <b>Postoperative H</b>      | lemoglobin                  | $10,35\pm1,48$                        | 9,75±1,42         | 0,063          |
|                             | °р                          | 0,001**                               | 0,001**           | bp             |
| Preop-Postop H              | emoglobin; (Median          | a) $1,40\pm0,87(1,20)$                | 1,44±0,88 (1,59)  | 0,835          |
| <sup>a</sup> Student t test | <sup>b</sup> Mann Whitney U | Test <sup>c</sup> Paired Samples Test | **p<0,01          |                |
| Table 5. Compli             | cations by type and d       | iameters of uterine fibroids          |                   |                |
|                             |                             | Number of Cases                       | Number of Cases   |                |
|                             |                             | without Complication                  | With Complication | Р              |
|                             |                             | (Total n = 179)                       | (Total n = 11)    |                |
|                             |                             | n (%)                                 | n (%)             | 0.633          |
| Diameter of                 | < 5 cm                      | 157 (94.6%)                           | 9 (5.4%)          |                |

#### **Table 4.** Preoperative and postoperative Hemoglobin changes

| Uterine Fibroid        | ≥ 5 cm     | 22 (91.7%) | 2 (8.3%) | 1.000 |
|------------------------|------------|------------|----------|-------|
| Type of                | Intramural | 81 (94.2%) | 5 (5.8%) |       |
| <b>Uterine</b> Fibroid | Subserosal | 98 (94.2%) | 6 (5.8%) |       |
| Fisher's Exact Te      | st         |            |          |       |
|                        |            |            |          |       |

**Table 6.** Comparison of mean change in hemoglobin values from baseline value, length of stay in hospital and transfusion rate between patients with uterine fibroids <5 cm and  $\ge 5$  cm in Diameter

| 1                                |         |                             |                  |                    |
|----------------------------------|---------|-----------------------------|------------------|--------------------|
|                                  |         | Diameter of Uterine Fibroid |                  | Р                  |
|                                  |         | < 5 cm (n=166)              | ≥ 5 cm (n=24)    |                    |
|                                  |         | Mean±SD (Median)            | Mean±SD (Median) |                    |
| Mean Change in Hemoglob          | in from | 1.40±0.87 (1.20)            | 1.44±0.88 (1.59) | <sup>a</sup> 0.835 |
| baseline                         |         |                             |                  |                    |
| Length of stay in hospital (day) |         | 2.37±2.34 (2)               | 2.25±0.44 (2)    | <sup>a</sup> 0.184 |
| Transfusion No                   |         | 157 (94.6)                  | 2.25±0.44 (2)    | <sup>b</sup> 0.633 |
| Transfusion Yes                  |         | 9 (5.4)                     | 2 (8.3)          |                    |
|                                  |         |                             |                  |                    |

#### Discussion

This study demonstrated that there were no differences between large and small size myomectomy during cesarean section in terms of complications, changes in hemoglobin levels, length of stay in hospital and blood transfusion rate.

Most obstetricians are opposed to performing myomectomy during cesarean section, except for cases with pedunculated and small fibroids, owing to the risk of excessive hemorrhage that may require hysterectomy or result in mortality. On the other hand, if myomectomy is avoided, it may increase the patient's risk of another operation that may unfavorably affect future pregnancies (17). Especially, large myomas >5 cm are associated with higher risk of adverse pregnancy complications such us preterm delivery, short cervix, premature rupture of membrane, postpartum bleeding when compared with small or no myomas (18). If safely performed, the procedure is cost-effective and can prevent morbidities associated with potential future surgeries. Myomectomy also reduces puerperal uterine involution and could decrease complications related to uterine fibroids, such as menorrhagia, anemia, and pain (19).

The safety of cesarean myomectomy has been previously studied. These studies have mostly been retrospective, with only a limited number of them being prospective in design. Cesarean myomectomy appears to be a safe procedure, as shown by retrospective studies involving small numbers of patients (8,17,20,21). Similarly, in a study that evaluated 368 patients with uterine fibroids, 111 patients underwent myomectomy and 257 patients underwent cesarean section only patients were selected for myomectomy using similar criteria as in a previous study.

But this study (14) noted that myomectomy should be avoided in the presence of large fundal or intramural fibroids, and that cesarean myomectomy could be a safe procedure in selected cases in the hands of experienced surgeons. In both studies, myomectomy was performed at surgeons' discretion. In a meta-analysis of nine studies (11), cesarean myomectomy was an appropriate procedure for cases in which there were no risks of complication, as determined by experienced surgeons. Although that review (11) did not report cases of hysterectomy, it emphasized that the procedure would not have been an appropriate approach in patients with large intramural fibroids owing to the risk of massive hemorrhage and the consequent need of hysterectomy.

In another review (22), in which the majority of removed fibroids were pedunculated and had <6 cm diameter, the authors note that great caution must be exercised while performing cesarean myomectomy, even if it appears to be a safe procedure, and they suggest that this procedure should be limited to cases of pedunculated or lower uterine segment fibroids that interfere with the evacuation of the fetus. There are some case reports on cesarean myomectomy in large myomas. Leanza et al. (23) demonstrated a successful cesarean myomectomy in a large myoma with 22 cm in diameter.

Ma et al. (24) also reported that cesarean myomectomy in a 40 cm sized moms was uneventful after the ligation of bilateral uterine arteries for the prevention bleeding. In a retrospective cohort study conducted by Kwon et al. (9) have shown that there were no statistical differences between cesarean myomectomy in large (>5 cm) and small (<5 cm) myomas in terms of hemoglobin changes, operative time, postoperative fever and hospitalized days.

Our study is meaningful, because we compared two groups to evaluate the safety of cesarean myomectomy in large myomas. Our study demonstrated that there were no differences between two groups in terms of complications, hemoglobin changes, blood transfusion and length of stay in hospital. Two patients with cornual fibroids and one patient with intramural fibroid (7 cm in diameter) developed hemorrhage; this observation supports the concerns discussed in the literature. However, these patients did not require additional surgical procedures other than simple suturing but erythrocyte suspension was given to these patients.

Various approaches have been suggested to reduce the bleeding during cesarean myomectomy. In a study of 9 cases, (25) selective uterine artery de vascularization and ligation of ovari-proprium ligament appeared to reduce hemorrhage. Ligation of the ascending or descending uterine artery was performed depending on the location of uterine fibroids. None of the patients required hysterectomy, and only one patient received blood transfusion. In a controlled prospective study, (26) patients who had undergone cesarean myomectomy were compared to patients who had undergone uterine artery occlusion and, with the exception of operation times, no significant differences were reported between the two groups in terms of complications, such as hemorrhage rates and mean change in hemoglobin values from baseline. In that study, patients in the control group had undergone cesarean section only, and this fact was considered a study limitation. In a randomized study of 52 patients (27), bilateral ligations of the ascending uterine arteries were compared to tourniquet application. Although one patient in the tourniquet group required bilateral ligation of the hypogastric artery postoperatively, no significant difference was

observed between the two groups in terms of hemorrhage or other complications. In our study, 8 patients developed uterine atony and 3 patients underwent bilateral ligation of the hypogastric artery. Bilateral ligation of the hypogastric arteries has been used to control uterine hemorrhage by reducing the pulse pressure of blood flowing to the uterus (28). The technique is challenging even for an experienced pelvic surgeon, especially when there is a large uterus, a transverse lower abdominal incision, ongoing pelvic hemorrhage, or the patient has a high body mass index. Successful and safe bilateral hypogastric ligation becomes even more difficult when attempted by a surgeon who rarely operates deep in the pelvic retroperitoneal space (29). For these reasons, uterine compression sutures and, less commonly, uterine artery ligation, have largely replaced this procedure as first-line surgical options.

Among the patients who developed hemorrhage, 3 presented cornual uterine fibroids and one had an intramural fibroid (7 cm in diameter). This patients did not require additional surgical procedures other than the suturing of the myoma bed.

The most important limitation of the present study is its retrospective design. The most important advantage of the present study is that studied patients had no antenatal data and were diagnosed with uterine fibroids shortly after presentation to the emergency room or during surgery. This has raised the question of whether random cesarean myomectomy could be a safe procedure even in large myomas. Patients with placenta previa totalis and placental detachment also underwent myomectomy, suggesting that selection criteria were less stringent than those used in previous studies.

#### Conclusion

Our results suggest that myomectomy in large sized is a safe procedure under emergency conditions during cesarean section. However, large intramural or cornual fibroids must be approached with great caution. In such cases, bilateral ligation of the uterine artery or ligation of the hypogastric artery together with ligation of the ovarian ligament should be considered before proceeding with myomectomy in order to reduce hemorrhage. Finally, recommend performing myomectomy under conditions in which blood products are readily available

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#### References

- Exacoustòs C, Rosati P. Ultrasound diagnosis of uterine myomas and complications in pregnancy. Obstet Gynecol. 1993;82(1):97-101.
- Strobelt N, Ghidini A, Cavallone M, Pensabene I, ceruti P, Vergani P. Natural history of uterine leiomyomas in pregnancy. J Ultrasound Med. 1994;13(5):399-401.
- Qidwai GI, Caughey AB, Jacoby AF. Obstetric outcomes in women with sonographically identified uterine leiomyomata. Obstet Gynecol. 2006;107(2 Pt 1):376-82.
- Laughlin SK, Baird DD, Savitz DA, Herring AH, Hartmann KE. Prevalence of uterine leiomyomas in the first trimester of pregnancy: an ultrasound-screening study. Obstet Gynecol. 2009;113(3):630-5
- Coleman-Cowger VH, Erickson K, Spong CY, Portnoy B, Croswell J, Schulkin J. Current practice of cesarean delivery on maternal request following the 2006 state-ofthe science conference. J Reprod Med. 2010;55(1-2):25-30.
- Sheiner E, Bashiri A, Levy A, Hershkovitz R, Katz M, Mazor M. Obstetric characteristics and perinatal outcome of pregnancies with uterine leiomyomas. J Reprod Med 2004;49:182-6.
- Davis JL, Ray-Mazumder S, Hobel CJ, Baley K, Sassoon D. Uterine leiomyomas in pregnancy: a prospective study. Obstet Gynecol 1990;75:41-4.
- Ortac F, Gungor M, Sonmezer M. Myomectomy during cesarean section. Int J Gynaecol Obstet 1999;67:189-90.
- Kwon DH, Song JE, Yoon KR, Lee KY. The safety of cesarean myomectomy in women with large myomas. Obstet Gynecol Sci. 2014;57(5):367-72.
- Kaymak O, Ustunyurt E, Okyay RE, kalyoncu S, Mollamahmutoğku L. Myomectomy during cesarean section. Int J Gynaecol Obstet. 2005;89(2):90-3.
- Song D, Zhang W, Chames MC, Gou J. Myomectomy during cesarean delivery. Int J Gynaecol Obstet. 2013;121(3):208-13.
- Burton CA, Grimes DA, March CM. Surgical management of leiomyomata during pregnancy. Obstet Gynecol. 1989;74(5):707-9
- Febo G, Tessarolo M, Leo L, Arduino S, Wierdis T, Lanza L. Surgical management of leiomyomata in pregnancy. Clin Exp Obstet Gynecol 1997;24(2):76-8.
- Roman AS, Tabsh KM. Myomectomy at time of cesarean delivery: a retrospective cohort study.BMC Pregnancy Childbirth. 2004;16;4(1):14.
- Hassiakos D, Christopoulos P, Vitoratos N, Xarchoulakou E, Vaggos G, Papadias K. Myomectomy during cesarean section: a safe procedure? Ann N Y Acad Sci.2006;1092:408-13.

- Vergani P, Locatelli A, Ghidini A, Andreani M, Sala F, Pezzullo JC. Large uterine leiomyomata and risk of cesarean delivery. Obstet Gynecol 2007;109(2 Pt 1):410-4.
- Park BJ, Kim YW. Safety of cesarean myomectomy. J Obstet Gynaecol Res. 2009;35(5):906-11.
- Shavell VI, Thakur M, Sawant A, Kruger ML, Jones TB, Singh M et al.. Adverse obstetric outcomes associated with sonographically identified large uterine fibroids. Fertil Steril 2012;97:107-10.
- Kwawukume EY. Caesarean myomectomy. Afr J Reprod Health. 2002;6(3):38-43.
- Celal K1, Hülya C. The evaluation of myomectomies performed during cesarean section in our clinic. Niger Med J. 2011;52(3):186-8
- Incebiyik A, Hilali NG, Camuzcuoglu A, Vural M, Camuzcuoglu H. Myomectomy during caesarean: a retrospective evaluation of 16 cases. Arch Gynecol Obstet. 2014;289(3):569-73
- Awoleke JO. Myomectomy during Caesarean Birth in Fibroid-Endemic, Low-Resource Settings. Obstet Gynecol Int. 2013:520834. doi: 10.1155/2013/520834
- 23. Leanza V, Fichera S, Leanza G, Cannizzaro MA. Huge fibroid (g. 3.000) removed during cesarean section with uterus preservation: a case report. Ann Ital Chir 2011;82:75-7.
- Ma PC, Juan YC, Wang ID, Chen CH, Liu WM, Jeng CJ. A huge leiomyoma subjected to a myomectomy during a cesarean section. Taiwan J Obstet Gynecol 2010;49:220-2.
- Desai BR, Patted SS, Pujar YV, Sherigar BY, Das SR, Ruge JC. A novel technique of selective uterine devascularization before myomectomy at the time of cesarean section: a pilot study. Fertil Steril. 2010;94(1):362-4
- Lin JY, Lee WL, Wang PH, Lai MJ, Chang WH, Liu WM. Uterine artery occlusion and myomectomy for treatment of pregnant women with uterine leiomyomas who are undergoing cesarean section. J Obstet Gynaecol Res. 2010;36(2):284-90.
- Sapmaz E, Celik H, Altungül A. Bilateral ascending uterine artery ligation vs. tourniquet use for hemostasis in cesarean myomectomy. A comparison.J Reprod Med. 2003;48(12):950-4.
- Evans S, McShane P. The efficacy of internal iliac artery ligation in obstetric hemorrhage . Surg Gynecol Obstet 1985;160:250
- 29. Joshi VM, Otiv SR, Majumder R, Nikam YA, Shrivastana M. İnternal iliac artery ligation for arresting postpartum hemorrhage. BJOG 2007; 114:356

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# Educational needs of patients undergoing coronary artery bypass graft

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## Abstract

**Objectives:** Cardiac surgery associates with potential physiological, mental, and emotional, growth and spiritual consequences due to the existence of many stressful factors. Training and learning patients can decrease riskcreating and stressful factors. Such a learning approach should be designed based on patients' needs and such needs should be identified by nurses before to any learning. However, there are rare studies focusing on the identification of patients' learning needs. Thus, this study aims to determine patients' awareness of learning needs for Coronary Artery Bypass Graft (CABG).

Material and Methods: This is a descriptive study carried out within 6 months on 101 cases undergone CABG (36 females and 65 males) in Tabriz Shahid Madani education and treatment center. They were investigated at the time of discharging from ICU and surgery departments. Patients' needs were determined using Cardiac Patients' Needs Learning Inventory (CPNLI) tool.

**Result:** According to findings, patients give the most important priority to the drug index. The next learning needs perceived by patients are :"How will my heart be treated?" related to "anatomical and physiological learning needs category", "what kind of activities can be done to reduce heart attack chance?" related to "risk factors learning needs category" and "how patients' activity level can be increased?" related to "physical activity learning needs category".

**Conclusion:** The prerequisite for implementing any learning program is the investigation and measurement of learning needs. Therefore, before initiating any learning program nurses need to continuously assay learning needs of patients while considering patients' abilities.

Key words: Coronary Artery Bypass Graft, Educational Needs

#### Introduction

The prevalence of cardiovascular problems and, as a result, the number of people with cardiovascular disease is increasing in Middle East, especially in Iran every year (1). Coronary Artery Bypass Graft surgery, (CABG), is the only essential and vital solution for cardiovascular patients (2) and is one of the most important surgeries conducted frequently every day (3). CABG surgery is an important event with deep mental and emotional effects on patients and their families (4).

Today, patients experience a shorter postsurgery hospitalization than before and according to expectations, this period will be shortened more in future. This new situation demands new measures for caring patients at home (5). Since most patients have no sufficient time to acquire necessary information about their disease during hospitalization, they view discharging as a very stressful event (6).

Therefore, learning can serve as an effective approach for promoting patients' awareness and decreasing their anxiety (7). According to studies, patients' learning improves the sense of post-surgery improvement in patients (8). Learning needs during hospitalization differ from learning needs during discharging and learning needs during self-caring period at home. The assessment of learning needs can be helpful in responding such needs and can affect patients' life style (9).

It is necessary to identify and explain these beliefs in order to effectively learn patients. It seems that such interventions would be more effective if they are designed based on coping style of each patient (10). Nurses need to personally assess their patients and establish an open relation with them (11). Learning needs during hospitalization differ from learning needs during discharging and learning needs during next steps.

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Such needs should be assessed accurately in order to make proper actions to respond them (12). For this purpose, by considering the ability of patients, all influential factors should be investigated and learning content should be designed based on learning needs (1).

#### **Material and Methods**

This is a descriptive study carried out within six months from October 2014 to March 2015 in ICU and surgery departments of education and treatment center of Shahid Madani hospital in Tabriz, Iran. First of all, patients' who satisfied inclusion measures were determined. Patients who undergo CABG surgery for the first time and were conscious and aware of time and location were studied at the time of discharging from ICU and surgery departments.

In 1984, Gerard developed Cardiac Patients Needs Learning Inventory (CPNLI) tool with questionnaire as data collection tool. This questionnaire consists of two parts. The first part covers demographic information while the second part deals with CPLNI tool including 37 questions of 6 categories: learning, anatomical and physiological risk factors medicinal questions, questions, information questions, diet information questions, physical activity questions and miscellaneous questions. The questions are scored as per Likert 5point scale from 1 to 5 where 1 stands for not important, 2 stands for less important, 3 stand for relative important, 4 stands for important and 5 stands for very important.

All statistical analyses were carried out by SPSS 13. Frequency, percentage, mean and standard deviation were calculated using descriptive statistics. The total score of CPLNI factors was calculated using the scores allocated by each patient to all 37 questions and the maximum and the minimum scores were determined.  $P \leq 0.05$  was set as the limit of statistical significance

#### Results

Thirty four (85%) did not have any university education. Twenty-four (23.7%) patients were seen training before surgery (Table 1). Sixty-five (64.3%) of patients were male. Total standard deviation of CPLNI was obtained as 4.29±0.56 implying the importance of patients' awareness of learning needs for CABG surgery. Regarding the obtained mean value, the learning needs of patients in the subsystems of anatomical and physiological questions, risk factors questions, medicinal information questions, diet information questions, physical activities questions and miscellaneous information questions lie inside "important" range where learning needs for medicinal information is more important than other subsystems(Table 2).

The most perceived learning needs are "how will my heart be treated" belonging to "anatomical and physiological learning needs category", "what kind of activities can be done to reduce heart attack chance?" belonging to "risk factors learning needs" and "How patients' activity can be increased" belonging to "physical activity learning needs". However, the lowest perceived learning needs are "being in sick mood" belonging to "anatomical and physiological learning needs", "restarting sexual activities" belonging to "physical activities learning needs" and "the meaning of cholesterol and triglyceride words" belonging to "diet information learning needs" (Table3).

21.7

25.7

|                               | Characteristics            | Frequency | Percent |
|-------------------------------|----------------------------|-----------|---------|
| Education                     | Illiterate                 | 34        | 85      |
|                               | Pre – university education | 47        | 46.5    |
|                               | Diploma                    | 11        | 22.2    |
|                               | University education       | 9         | 38.1    |
| Marital status                | Single                     | 9         | 8.91    |
|                               | Married                    | 92        | 91      |
| <b>Preoperative Education</b> | Yes                        | 24        | 23.7    |
|                               | No                         | 77        | 76.2    |
| Ward                          | Men Surgical Department    | 53        | 52.4    |

22

26

Female Surgical Department

Intensive Care Unit

**Table 1:** Baseline personal characteristics of patients who were undergoing coronary artery bypass surgery

|--|

| Training Needs           |             |  |
|--------------------------|-------------|--|
| Titles of training needs | Calibration |  |
| Drug Information         | 1           |  |
| Physical Activity        | 2           |  |
| Risk Factors             | 3           |  |
| Other Information        | 4           |  |
| Anatomy and Physiology   | 5           |  |
| Diet Information         | 6           |  |

**Table 3:** The mean level of awareness the needs of patients with coronary artery bypass surgery

| Titles of training needs | Mean and standard deviation | Confidence interval |
|--------------------------|-----------------------------|---------------------|
| Anatomy and Physiology   | 4.26 ±0.55                  | 4.07 -4.44          |
| Risk Factors             | 4.36±0.58                   | 4.16-4.56           |
| Drug Information         | 4.42±0.65                   | 4.20-4.64           |
| Information Diet         | 4.25±0.73                   | 4.01-4.50           |
| Physical Activity        | 4.37±0.70                   | 4.13-4.61           |
| Other Information        | 4.34±0.72                   | 4.09-4.58           |

## Discussion

Learning is a dynamic issue of nursing. The determination of the priority of learning needs is the first step for learning programming. Therefore, nurses need to determine learning needs during hospitalization period or a short time after the illness (13). The extent of patients' awareness of CABG surgery lies inside "very important" category. This implies that patients need more learning. Through individual assessments and establishing an open relation with their patients, therefore, nurses can aid patients to satisfy their learning needs.

With an average score of 4.42, medicinal information subsystem is a high required learning need in our study. The study of Vahabi and Berner with the title of "investigating the learning needs of people with heart failure" showed that the most required learning need for patients is medicinal information need (14) that agrees with our study. It is better for nurses to pay more attention to learning medicinal information to their patients.

With a mean score of 4.54 the learning need of "actions to be done to reduce heart attacks" has the highest rank. In Rostami and etal study with the title of "learning needs of patients with myocardial infraction", this learning need had a mean score of 4.78 (15) implying that this study's patients have more learning needs than those of our study. However, it is better for nurses to inform patients of necessary actions to be done to reduce heart attack through establishing an open and continuous relation with patients.

With a mean score of 4.14, the learning need of "restarting sexual activities" is the least scored measure with the lowest priority. In Rostami and etal study with the title of "learning needs of patients with myocardial infraction" this learning need had a mean score of 2.30 (14). Also, Torenton has express that sexual issues should be ranked as lower priorities and learning programs should be practiced in accordance with learning needs of patients (16).

## Conclusion

The main emphasis of this study is patients' awareness of learning needs for CABG surgery. According to results and considering the importance of patients' learning needs for drug consumption, this necessity, especially the need for heart drugs information, should be explained to patients. It is recommended to prioritize the learning needs of patients at first and then put necessary training into practiced on this basis

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#### References

- Nasiri M, Rahimian B, Larihani M, Norozi SH. Survey the educational needs and quality of the patients with chronic myocardial infarction in Emam Sajjad hospital in Ramsar. Journal of Urmia Nursing AND Midwifery Faculty. 2008; 6(3): 145-150.
- 2. Finkelmeier BA. Cardiothoracic Surgical Nursing. Philadelphia: W. B. Lippincott Co; 2000.

- Barnett SD, Halpin LS. Functional status improvement in the elderely followin coronary artery bypass graft. J Nurs CQ. 2003; 18(4): 281-7.
- Stroobant N, Vingerhoets G. Depression, anxiety, and neuropsychological performance in coronary artery bypass graft patients: a follow-up study. Psychosomatics. 2008; 49(4): 326-331.
- Lukkarinen H, & Kyngäs H, Experiences of the onset of coronary artery disease in a spouse. European Journal of Cardiovascular Nursing. 2003; 2: 189–194.
- Taherian A, Mohammadi F, Hosseini MA, Rahgozar M, Fallahi M. The effectiveness of patient education and home- based follow up on knowledge and health behavior in the patients with myocardial infarction. Iranian Journal of Nursing Research. 2007;2(4-5): 7-13.
- 7. Astley CM, Chew DP, Aylward PE, Molloy DA, De Pasquale CG. A randomised study of three different informational AIDS prior to coronary angiography, measuring patient recall, satisfaction and anxiety. Heart Lung Circ. 2008; 17(1): 25-32.
- Ozar N, Hacialioglu N. The Problems and Needs of Caregivers after Coronary Artery Bypass Graft Surgery. Atatürk Üniversitesi Hamşirelik Yüksekokulu Dergisi. 2008; 11:2.
- Smelter S, Bare B. Brunnerand Suddarth text book of medical –surgical nursing. Philadelphia, lippincott: Williams & wilkins, 2004; 46.

- Martelli MF, Auerbach SM, Alexander J, Mercuri LG. Stress management in the health care setting: matching interventions with patient coping styles. J Consult Clin Psychol. 1987; 55: 201–7.
- Mousavi Shabestari M, Parizad R .Stressors in Patients Undergoing Cardiac Surgery and Attitudes of Nurses and Patients., Crescent Journal of Medical and Biological Sciences. 2014;1(1): 1-3.
- Brunner LS, Smeltzer SCOTT. Brunner & Suddarth's textbook of medical- surgical nursing. 12th ed. Philadelphia: Lippincott Williams & Wilkins.2010; 46.
- Coatos, E. Education for patients and clients. London: Routledge Co. 1999; 118-223.
- 14. Wehby D, Brenner PS. Perceived learning needs of patients with heart failure. Heart Lung. 1999; 28(1): 31-40.
- Rostami H, Ghahramanian A, Golchin M. Educational needs of myocardial infarction patients. Journal of Urmia Nursing and Midwifery Faculty. 2011; 9(3):157-64.
- Turton J. Importance of information following myocardial infarction study of the perceived information needs of patients and their spouse – partner compared with perceptions of nursing staff. J Adv Nurs . 1998; 27:770-8.

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# Case Report

# Development of cerebral infarction after stripping of vena saphena magna

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#### Abstract

Cerebrovascular disease is the most frequent cause of neurological diseases. Embolic infarction is one of the most common causes of stroke. In the majority of patients with cerebral embolism, it is shown that emboli mostly originate from the heart via arterial circulation. Embolism rarely occurs as a paradoxical embolism through atrial / ventricular septal defect (ASD/VSD) or patent foramen ovale (PFO). In our clinical case, cerebral infarction is caused by ASD after vena saphena magna (VSM) stripping. Thirty- four- year- old female patient was admitted with motor and sensory loss at left lower extremity below the knee after stripping of VSM. Detailed neurological examination was done. She had left foot motor deficit, anesthesia of left lower extremity below the knee, impairment of motor strength (0/5 dorsiflexion and plantar flexion) of the left foot. After radiological investigation, diffusion weighted magnetic resonance imaging (MRI) revealed several, millimetric, nonspecific, nodular signal changes in some parts of bilateral periventricular, subcortical white matter of the brain that showing no diffusion limitation. In systematic examination, atrial septal defect was detected by echocardiography. Patient was treated with anticoagulant therapy. This case highlights detailed cardiological examination should be done in patients with cerebral infarction

Key words: Atrial septal defect, Cerebral Ischemia, Lower extremity motor, Sensory loss

#### Introduction

Cerebrovascular disease is a leading cause of neurological events, in terms of importance and frequency in adulthood. Embolic infarction is one of the most common causes of stroke. The most common sites of origin of cerebral emboli are cardiac thrombus. Embolism may break from occluded or severely stenotic carotid artery, distal end of the thrombus of vertebral artery or an ulcerated atheromatous plaque extending into the lumen of the carotid sinus. 5-10% of cerebral ischemia is caused by paradoxical embolism. One of the reasons that may be able to cause paradoxical embolism is atrial septal defect (2). ASD is characterized by a hole between atriums and causes abnormal blood flow. Thus, a portion of blood abnormally flows to the right heart. This situation may cause damage to blood vessels in the lungs and the heart muscle over the years due to increased blood flow to the lung. The patients which has ASD usually lives with asymptomatic for many years. Definitive diagnosis is based on echo cardiograph. ASD is a rare cause of embolism (3, 4). The aim of this study was to report a case with acute cerebral ischemia caused by ASD.

### Case

34 year old female patient was admitted to department of Cardiovascular Surgery Clinic at Gazi University Medical School with left lower extremity motor and sensory loss below the knee. In the clinical history, it is learned that she underwent vena saphena magna stripping surgery in another clinical centre and she applied on the third postoperative day with motor and sensory loss. There was no positive sign in her family history. In the neurological examination, the patient was awake, cooperated, oriented and there were no signs of meningeal irritation or neck stiffness. Motor deficit is found in the left food; the grade of dorsiflexion and plantar flexion was 0/5. Left lower extremity below the knee was anesthetic, routine complete blood count, biochemical tests were normal. The patient was consulted to neurology, neurosurgery, orthopedics, physical therapy and rehabilitation Electromyography departments. (EMG) was performed and reported as normal. The symptoms are thought to be related with the central nervous system pathology. Electroencephalography (EEG), MRI and cranial computerised tomography (CT) was performed.

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Figure 1. Axial T2-weighted (A) and FLAIR (B) images show nonspecific hyper intense signal changes (arrows).

EEG and cranial CT were reported as normal. Bilateral carotid and vertebral artery Doppler ultrasonography was performed and reported as normal. On the diffusion-weighted cranial MRI a few millimetric nonspecific noduler signal changes exhibiting no diffusion restriction was observed in bilateral periventricular, subcortical white matter areas of the brain (figure 1). Echocardiography was planned. Left ventricular cavity size was normal, measurement of left ventricle walls thickness was normal, and analysis of systolic and diastolic left ventricular wall motion was normal. Shunt flow was observed through inter atrial septum. QP / QS ratio was 1. The patient's anticoagulant therapy was planned physical therapy program was started and the patient was discharged from hospital.

#### Discussion

Embolic stroke constitutes 15-20% of all strokes. In recent years because of widespread availability of trans-esophageal echocardiography (TEE) the frequency of the cardiac sourced embolism has increased. Transthoracic echo-cardiography (TTE) has been shown to have sensitivity for indicating the left ventricle and mitral valve disease. If the patient is old and the cardio-embolic reason is obvious, TTE is enough (3). TEE wasn't performed, as other tests that may be related with the ethology were normal and ASD was enough to explain this case. Especially in younger patients ASD should be considered as the cause of cardio-embolic stroke. In a study which included 152 young stroke patients, %1 of the patients with ASD had been reported by the Segmen et al (3). In recent years, ASD is represented as a major cause of retinal and cerebral embolism (5). Ash et al reported three cases with ASD and ASD was thought to be the possible cause of cerebral embolism. They explained the importance of TEE and TTE in unexplained cerebral ischemia cases especially in the young (5). Devuyst et al reported the importance of the aortic arch atheroma, atrial septal aneurysm (ASA) in cardiac stroke (6).

Sayin et al reported a case with atrial septal defect, retinal artery occlusion and cerebral infarct and explained the importance of detailed cardiological examination in cases detected transient ischemic attack or cerebral infarction (7). The findings in our patient's case are likely explained by ASD.

#### Conclusion

ASD is the most common congenital heart defect encountered in adults. Small ASDs are usually asymptomatic and compatible with a normal life. We believe that the potential cause of cerebral ischemia in our patient is ASD. We would like to emphasize once more the importance of detailed cardiological examination in the cases detected cerebral infarction

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#### References

- Ropper A. H, Brown R. H. Adams and Victor's Principles of Neurology. In: Togay Işıkay C., editor. 8th ed. Vision Disorders.2006: 209.
- Abu Rahma AF, Richmond BK., Robinson PA. Etiology of peripheral arterial tromboembolism in young patients. American Journal of Surg. 1998; 176: 158-161.
- Seğmen H, Kayım Ö., Bolayır E. Echocardiographic findings in young ischemic patients. Journal of Cumhuriyet University Faculty of Medicine 2007; 29 : 109-112.
- 4. Crump R, Shanding AH., Van nata B. Ellestad M. Prevalance of patient foramen ovale in patients with acute myocardial infarction and angiographically normal coronary arteries. American Journal of Cardiol. 2000; 85 : 1368-70.
- Ash N, Lubetsky A, Ezra D, Olchovsky D. Atrial septal defects and embolic stroke inyoung adults. Harefuah. 1992 Aug;123(3-4):91-4, 155.
- G. Devuyst, J. Bogousslavsky. Status of Patent Foramen ovale, Atrial Septal Aneurysm, Atrial Septal Defect and Aortic Arch Atheroma as Risk Factors for Stroke.Neuroepidemiology 1997;16: 217-223.
- Refah Sayın, Tezay Çakın Güleç, Temel Tombul A Case with Atrial Septal Defect, retinal artery occlusion and Cerebral Infarction.Van Medical Journal: 16 (3):109-112, 2009.

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# **Open Laryngeal Fracture:** A case report and review of the literature

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#### Abstract

**Objectives:** Laryngeal fracture is a rare and life threatening entity which is caused by blunt or penetrating neck trauma. Majority of laryngeal fractures are closed type and caused by blunt neck trauma. They are frequently left under diagnosed due to their closed nature and accompanying multisystem trauma. Also, open laryngeal fracture is exceptional and seen particularly in penetrating neck traumas like gunshot injuries. We describe an unusual case of open laryngeal fracture in which the patient was totally breathing from the wound as if a coniotomy had done

Case: A 23-year-old male presented to the emergency department after getting involved in a motorcycle accident resulting with a blunt neck injury. The patient was breathing from a horizontal open neck wound below the level of the thyroid cartilage. Computed tomography revealed a laryngeal fracture involving thyroid, cricoid cartilages and the first ring of trachea. Given the extent of injury, a laryngeal reconstruction was performed. Six weeks after surgery he had no stridor, with evidence of full vocal cord mobility on both sides.

**Conclusion:** Early diagnosis is indispensable because it may cause acute airway compromise or significant long term squeal like dysphonia, aspiration, airway stenosis and tracheoesophageal fistula. In laryngeal fracture transoral intubation may lead irremediable results especially in the comminuted fractures or if there's arytenoid dislocation. Thus, in suspicion of laryngeal fracture associated with airway compromise one should always discuss tracheotomy rather than trans-oral intubation

Key words: Larynx, fracture, blunt, neck injury

#### Introduction

Laryngeal trauma (LT) is very rare (1-3) owing to elastic nature of larynx and protective effect of mandible and sternum. It has a reported incidence of 1 in 30000 patients among severe trauma admissions to the emergency department (1,3). LT may range from simple mucosal tears to fractured and comminuted cartilage. If it is not thoroughly identified and properly treated LT may cause death due to airway compromise or may cause significant long term squeal like dysphonia, aspiration, and airway stenosis (4). Additionally, trans-oral intubation may lead catastrophic results like worsening the preexisting injury, causing further tears or cricotracheal separation.

Cervical and intracranial injuries are also common in these patients (5-9). For this reason, trauma guidelines concerning the potentially affected sites should also be considered. LT can be in 2 forms as penetrating or blunt. Penetrating injury commonly result from gunshots while blunt injury occurs mostly due to motorcycle accidents.

We report an extraordinary blunt neck trauma occurred during a motorcycle accident in which the patient was presented with breathing from the wound site as if a coniotomy had been done. The condition was due to a serious open laryngeal fracture including both the thyroid and cricoid cartilages associated with

partial thyrocricoid separation. We provide a review of pertinent literature, with an emphasis on presentation to the emergency department, airway management. surgical reconstruction and classification of laryngeal traumas.

#### Case

A 23-year-old male presented to the emergency department after getting involved in a motorcycle accident resulting with a blunt injury of the neck. He was conscious but not able to talk. Upon initial ENT evaluation, it was noted that the patient was breathing from a horizontal open neck wound as if a coniotomy had been done (Figure 1).

Borders of the horizontal wound were clean with a length of 3 centimeters and there was no sign of any massive hemorrhage. Flexible laryngoscopic examination revealed paralyses of both vocal cords. There were also a considerable amount of edema and ecchymosis at both false vocal cords and aryepiglottic folds. After acute stabilization, the patient underwent computed tomography (CT) scan, revealing a laryngeal fracture involving thyroid, cricoid and the first ring of trachea.

Non comminuted fractures were also determined in the level of 5th, 6th and 7th cervical vertebra.



**Figure 1** (a) Horizontal wound at the level of Thyroid Cartilage. (b) Closed view showing fracture line on the thyroid cartilage in relationship with the wound



**Figure 2** (a) Thyroid cartilage fracture line is seen while the intubation tube is oriented superiorly. (b) Fracture line involving the cricoid and first ring of trachea is seen while the intubation tube oriented inferiorly

Neurological examination was normal except a minimal numbress at the right hand and forearm. Neurosurgical department did not suggest any surgical intervention. They applied only a cervical collar.

In the light of these findings we decided to perform a surgical exploration. The patient was intubated through the open wound and exploration was carried out via this open neck wound with the endotracheal tube in view. It was seen that there was a clean fracture line starting from the thyroid notch including the whole anterior wall of the thyroid and cricoid cartilages, then extending inferiorly down to the inferior edge of first tracheal ring (Figure 2).

The cricothyroid membrane and anterior mucosa of subglottic region was totally defective leading a partial separation of thyroid and cricoid cartilages. After first evaluation, a 2nd vertical skin incision, starting from the wound, was performed and a tracheotomy between the 3rd and 4th tracheal ring was carried out. Then the endotracheal tube was replaced inferiorly to this tracheotomy site for better visualization of the posterior wall of subglottic area and cricoid cartilage. After replacement of the endotracheal tube it was noted that the subglottic mucosa was ripped and peeled off the posterior wall of the cricoid cartilage. A non-displaced fracture line was also noted on the stripped posterior wall of cricoid cartilage. Besides, the cricoarytenoid joint of both sides were intact.

Given the extent of injury, a thyrocricoidal reconstruction was attempted. Firstly, we started to the reconstruction with the repair of subglottic mucosa from posterior to anterior. The teared superior and inferior edges of posterior subglottic mucosa were approximated and sutured by using 5-0 Vicryl. Then the laryngeal fractures were reduced and fixated using 2-0 Prolene sutures. Attention was then devoted to the management of partial thyrocricoidal separation. Using 3-0 Vicryl, the thyroid cartilage was sutured anterolaterally to the cricoid cartilage. These sutures were loosely tied to help re-approximate the airway. Two 2-0 Prolene stay sutures were placed through the thyroid cartilage and the 1st tracheal ring to secure this repair.

The patient's postoperative course was uncomplicated. The patient was observed in the intensive care unit for 2 days and then total parenteral nutrition was started. At the second day of surgery a barium esophagogram was done which was suspicious about leakage. So an esophagoscopy was also done demonstrating no evidence of an esophageal disruption.

Oral intake was started approximately 4 weeks after injury. The patient showed signs of vocal cord recovery 6 weeks after the injury on both sides, and the tracheostomy was closed. On last follow-up (3 months after the injury), he had no stridor, with evidence of full vocal cord mobility on both sides.

#### Discussion

Laryngeal framework is protected superiorly by maxilla and mandibula, inferiorly by sternum. Due to this protective effect and elastic nature of larynx, laryngeal fracture is very rare (1-3). It is rare also because severe laryngeal trauma patients die be¬fore reaching a medical facility owing to serious airway compromise or associated multisystem trauma. Thus, a considerable amount of laryngeal fracture cases become under-diagnosed (2,3).

Laryngeal fracture can be caused by 2 mechanisms as blunt neck trauma or penetrating neck trauma. Blunt neck trauma almost always occurs in connection with motor vehicle accidents, and it has been reported to be the most frequent cause (10). But in recent years it has a decreasing tendency due to improvement of airbags and abun¬dant safety devices for riders (11). Besides, blunt trauma related laryngeal fracture due to sportive events in which motorcycles, bicycles, skis, etc. are used is on increase (12).

Penetrating neck trauma is relatively rare. In the past, gunshots were the leading causes. But, nowadays owing to industrialization and urbanization penetrating trauma is also on increase because of machines related accidents and stabbing (10). Laryngeal trauma patients may present with hoarse¬ness, stridor. respiratory distress. subcutaneous emphysema, hemop¬tysis, hematomas, subcutaneous hemorrhage, laryngeal tenderness, vocal cord dysfunction, and the loss of an anatomical landmark in the neck (1, 13). Our case was a blunt neck trauma due to a motorcycle accident in which the patient hit a lamppost.

The type of injury was pertinent with the literature but the presentation was very exceptional. At the admission, the patient was totally breathing from a midline horizontal open wound at the level of the cricothyroid membrane as if a cricothyrotomy (coniotomy) was done (Figure 1). There was no respiratory distress.

Esophageal injury may also occur in LT Hence, barium esophagogram and/or cases. esophagoscopy should be done to rule out esophageal disruption (3,14,15). In our case the barium esophagogram was suspicious but the esophagoscopy was normal. So the patient able to fed trans-orally. Vocal cord paralyses are also common in laryngeal fracture cases and may occur due to direct injury or secondary injury caused by edema or hematoma. In the present case the paralyses recover at the 6th week so the probable mechanism was vocal cord injury secondary hematoma and edema.

Open fractures of the laryngeal framework are quite uncommon, and likely to be missed only by failure to examine the neck, which may perhaps be obscured by a cervical collar as in our case. On the contrary, fractures of the larynx are more likely to be "open" internally, where the defect may be through the mucosa rather than the skin, and therefore missed by external examination alone. These injuries may ultimately require endoscopic or open repair, or tracheotomy (16).

In our case there was an open wound through which the fractured thyroid and cricoid cartilage were seen by close observation (Figure 1). This type of presentation is very uncommon for a blunt neck injury. We think that this is caused by high torsional forces leading the neck to over-extension during the accident. This also may be the liable mechanism explaining the cervical fractures in the case reported.

Schaefer had classified the laryngeal injuries into V groups according to the severity (1). Our case was compatible with group IV which is defined as having disruption of anterior larynx, unstable fractures, two or more fractures lines, or severe mucosal injuries. Not all the laryngeal trauma patients need surgical intervention. Group-I injuries can be managed conservatively. Group II to IV patients commonly necessitates surgical intervention as endoscopic, open or both. In the present case open surgical repair was inevitable.

Additionally, in this classification system the condition of the trachea had not been taken into account except the group V which is defined as complete laryngotracheal separation. However, concerning the concurrent tracheal injury in laryngeal trauma patients there should be a more detailed definition. For example in our case there was a midline cut of first tracheal ring leading a partial laryngotracheal separation which is not defined in the aforementioned classification. Thus, we think that a more precise classification should be defined.

In the literature, 3 different external techniques for the reduction of LF were defined. These are make use of miniplates, wire or primary suturation for reconstruction of the laryngeal skeleton (14,16,17). We preferred to use primary suturation because there are some reports about fibrous healing especially with wire usage. Concerning the miniplate usage; it is more suitable in comminuted or severely displaced fractures.

Consequently, open laryngeal fracture following blunt neck trauma is very rare and the reported cases, unlike our case, were internally open fractures due to torn laryngeal mucosa. Additionally, totally breathing from the wound is a very exceptional presentation. Emergency physician should avoid intubation in suspicion of laryngeal trauma otherwise it may worsen the preexisting injury and may cause further tears or even cricotracheal separation leading long term complications like dysphonia, aspiration, and airway stenosis.

**Conflict of Interest:** The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

#### References

- Schaefer SD. The acute management of external laryngeal trauma. A 27-year experience. Arch Otolaryngol Head Neck Surg. 1992 Jun;118(6):598-604.
- 2. Gussack GS, Jurkovich GJ, Luterman A. Laryngotracheal trauma: a protocol approach to a rare injury. Laryngoscope 1986 Jun;96(6):660-5.
- 3. Jalisi S, Zoccoli M. Management of laryngeal fractures--a 10year experience. J Voice. 2011 Jul;25(4):473-9.
- 4. Mendelsohn AH, Sidell DR, Berke GS, John MS. Optimal timing of surgical intervention following adult laryngeal trauma. Laryngoscope. 2011 Oct;121(10):2122-7
- 5. Butler AP, Wood BP, O'Rourke AK, Porubsky ES. Acute external laryngeal trauma: experience with 112 patients. Ann Otol Rhinol Laryngol. 2005 May;114(5):361-8.
- Yen PT, Lee HY, Tsai MH, Chan ST, Huang TS. Clinical analysis of external laryngeal trauma. J Laryngol Otol. 1994 Mar;108(3):221-5.
- Harris HH, Tobin HA. Acute injuries of the larynx and trachea in 49 patients. (Observations over a 15-year period). Laryngoscope. 1970 Sep;80(9):1376-84.
- Leopold DA. Laryngeal trauma. A historical comparison of treatment methods. Arch Otolaryngol. 1983 Feb;109(2):106-12.
- 9. Schild JA, Denneny EC. Evaluation and treatment of acute laryngeal fractures. Head Neck. 1989 Nov-Dec;11(6):491-6.
- Kim JP, Cho SJ, Son HY, Park JJ, Woo SH. Analysis of clinical feature and management of laryngeal fracture: recent 22 case review. Yonsei Med J. 2012 Sep;53(5):992-8.
- Irish JC, Hekkenberg R, Gullane PJ, Brown DH, Rotstein LE, Neligan P, Ali J. Penetrating and blunt neck trauma: 10-year review of a Canadian experience. Can J Surg. 1997 Feb;40(1):33-8.
- 12. Lee WT, Eliashar R, Eliachar I. Acute external laryngotracheal trauma: diagnosis and management. Ear Nose Throat J. 2006 Mar;85(3):179-84.
- 13. Hwang SY, Yeak SC. Management dilemmas in laryngeal trauma. J Laryngol Otol. 2004 May;118(5):325-8.
- Bent JP 3rd, Silver JR, Porubsky ES. Acute laryngeal trauma: a review of 77 patients. Otolaryngol Head Neck Surg. 1993 Sep;109(3 Pt 1):441-9.
- Vassiliu P, Baker J, Henderson S, Alo K, Velmahos G, Demetriades D. Aerodigestive injuries of the neck. Am Surg. 2001 Jan;67(1):75-9.
- Chitose S, Sato K, Nakazono H, Fukahori M, Umeno H, Nakashima T. Surgical management for isolated cricoid fracture causing arytenoid immobility. Auris Nasus Larynx. 2014 Apr;41(2):225-8.
- Thor A, Linder A. Repair of a laryngeal fracture using miniplates. Int J Oral Maxillofac Surg. 2007 Aug;36(8):748-50.

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# Susac's syndrome: 2 cases without hearing loss

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#### Abstract

**Objectives:** Susac's syndrome is a disease of the microvasculature in the brain, inner ear (cochlea and vestibular apparatus) and retina that consists of a clinical trial of encephalopathy, hearing loss and visual disturbance. It is usually seen in young women. It is first defined by Susac et al. in 1979. Diagnosis is often difficult because the classic triad may take up to 2 years to develop and radiological findings are easily confused with multiple sclerosis. Also, encephalopathy may mask some other findings.

**Case:** We present two cases that we think are particularly instructive because they were male patients, had somewhat unusual presenting symptoms, had no accompanying hearing loss, required relatively less aggressive and less sustained immunosuppression than usual, and followed a relatively short and benign course, without relapse and with excellent outcome..

**Conclusion:** This two case serve as good examples of the apparent efficacy and sufficiency (in their cases) of Intravenous immunoglobulin for Susac's syndrome

Key words: Susac's syndrome, Hearing loss

#### Introduction

Susac's Syndrome is usually seen in young women and characterized by microvascular occlusion and either microischemia injury or microinfarction in the brain, retina, and inner ear. It can cause a wide spectrum of neuro-psychiatric symptoms, sensorineural hearing loss, vestibular dysfunction, and a spectrum of visual symptoms, including branch retinal arteriolar occlusion (1-5). Susac syndrome appears to represent an autoimmune microvascular endotheliopathy. It is first defined by Susac et al. in 1979 (5). It typically occurs between ages 18 and 50, with a mean age of 25. Female to male ratio is 5(1-4).

Although presenting symptoms vary, the most common one is headache (6-9). 75% of the patients have cognitive and behavioral symptoms Because the disease (1,3,4,5,6,8).is а microvasculopathy with multiple lesions, the variety of neurological symptoms is wide, according to the affected region. In addition to cognitive dysfunction, ataxia, hemiparesis, paresthesias, brisk deep tendon reflexes, Babinski sign, bladder dysfunction, and seizures may be seen (4,10).

The typical ophtalmological finding is branch retinal artery occlusion (BRAO) with associated visual field deficit (1,2). Fluorescein angiography typically reveals segmental hyperfluorescence and leakage in one or more vessels. The specific audiologic finding is bilateral low frequency sensorineural hearing loss (11-14).

The disease may follow a relapsing and remitting course. However, full and sustained remission after an initial attack has been reported, as have very aggressive forms with frequent attacks and devastating outcome. Relapses may occur despite aggressive immunosuppressive treatment, particularly if treatment is tapered too much, too soon. Moreover, if the treatment is inefficient or delayed; varying degrees of visual, auditory or neurological sequelaecan occur. (13-16). Prompt aggressive, and sustained immunosuppressive therapy has been advocated for Susac syndrome, with a realization that there is a spectrum regarding the amount of treatment needed. (14-17).

#### Case 1

A 25 year-old male patient was brought to the psychiatry outpatient clinic by family members due to decreased social interaction and change in personality. In addition to acute psychotic behaviors, neurologic findings were evident, and MRI revealed a few plaque-like lesions both in the periventricular region and corpus callosum. These findings prompted admission. He was disoriented, agitated, had increased need for sleep, incoherent speech and mild left central facial paralysis. He had a right ataxia and an extensor Babinski reflex on the right. Remaining neurological examination was inconclusive. CSF analysis revealed no cells, but a protein level of 280 mg/dl (N:15-45 mg/dl).

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Oligoclonal band test was negative. Blood tests revealed no evidence of systemic inflammation. ANA, ANCA, ACE were negative or normal.

On the second day of hospitalization, the patient developed right hemiparesis, and repeat cranial MRI revealed an increased number of lesions (with enhancement) in white matter, especially in the corpus callosum, and leptomeningeal enhancement (Fig. 1ABC). EEG showed diffuse slow (theta, delta) waves. A diagnosis of Susac Syndrome was made after seeing many occlusions in FFA (Fig.1D). He did not cooperate in audiometric examination, but he was not complaining of any auditory symptoms. Ear examination and whispering test were reported as normal. He was initially given methylprednisolone 1 g/day for 5 consecutive days but no improvement was noted. Intravenous immunoglobulin (IVIG), 0.4 g/kg/day was given for the next 5 days.

During these 5 days, his right hemiparesis and central facial paralysis improved, and they vanished after the subsequent week. His speech became meaningful, he became cooperative, more oriented and his ataxia almost disappeared. A repeat MRI (on second week of hospitalization) showed that the supratentorial and infratentorial lesions had significantly decreased in number and size, and there was no longer any leptomeningeal enhancement. After the second week, when the patient was observed as more cooperative and not agitated, audiometric tests were applied and were normal. Also, he was no longer noting visual symptoms, and a repeat FFA showed subsidence of his previous abnormalities. Then he was discharged to continue the monthly IVIG therapy in outpatient setting. One month after discharge examined his neurologic exam was completely normal. His EEG showed a normal wave pattern, with normal amplitudes and rhythm.

This patient has now been followed for one year, during which time he has been asymptomatic and his only treatment has been monthly IVIG (0.4 g/kg/day).

#### Case 2

A 22 year-old male patient consulted an ophthalmologist after noticing some dark areas in his visual field when he woke up in the morning. He was complaining of being unable to see the lower halves of objects with his right eye. FFA revealed BRAO(fig. 2AB). The patient was given hyperbaric oxygen therapy for 5 sessions but no alleviation was observed in his complaints.

Neurology consultation was requested and revealed that he had been having a unilateral throbbing headache accompanied with nausea, photofhobia and phonophobia for a two months. He had no motor deficits, but had bilateral achilles clonus with 3-4 beats and brisk deep tendon reflexes in both lower extremities. Cranial MRI, revealed gliotic lesions in periventricular area and corpus callosum (fig. 2CD). His blood tests revealed no evidence of systemic inflammation. CSF analysis revealed no pleocytosis, a negative oligoclonal band test, but a protein level was 170 mg/dl. The distribution of the lesions in cranial MRI, the branch retinal arteriolar occlusions, and the CSF results led to a diagnosis of Susac Syndrome. His audiometric tests were normal. He was treated with methylprednisolone 1 g/day for 7 consecutive days, but his symptoms did not subside and he did not otherwise seem to improve. Then, he received IVIG 0.4 g/kg/day for 5 days, after which his symptoms improved, his headaches fully resolved, and a follow-up MRI (2 weeks later) showed a marked decrease in lesion burden.

Repeat FFA showed some residual zones of infarction. After discharge, he was treated only with monthly IVIG.

He has now been followed for one year and has shown remarkable recovery, with no additional MRI lesions and no evidence of disease relapse. He is still receiving monthly IVIG.

#### Discussion

Susac Syndrome is a disease of the microvasculature in the brain, inner ear, and retina, that consists the clinical triad of encephalopathy, hearing loss and visual loss. It is seen most commonly in young women, has a wide variety of clinical manifestations, and is important because it can cause devastating sequelae, is treatable, and is often confused with multiple sclerosis, both clinically and radiologically (1,3,4,5,8,17).

The most common symptom is headache. Visual complaints may mislead the physician to a diagnosis of migraine (1,3,5-9). Moreover, the patients may have psychiatric symptoms, changes in personality and present with encephalopathy. Many patients initially consult a medical doctor because of visual disturbance or hearing loss, or both (2,7,12,13).

If encephalopathy occurs, it may be hard to evaluate the patient in terms of visual and audiologic examination, which in turn complicates the diagnosis. There have been 2 cases in literature from Turkey (18,19). This may indicate underdiagnosis, but could, conceivably, also be due (at least in part) to a decreased incidence in the Turkish population.

In our cases, the reasons to consult a hospital were the family members' recognition of personality change and increased need for sleep in our first case, and visual loss in the second case. It should be appreciated that patients with encephalopathy or a psychotic condition may have problems with expressing their visual and hearing loss.

The retinal arteriolar occlusions were detected after we suspected Susac Syndrome and ordered a retinal angiography. The typical visual loss in this disease is a segmental visual field loss which is secondary branch retinal artery occlusion.



**Figure 1.** ABC: Leptomeningeal contrast enhancement in inferior fossa in T1 weighted image, Elliptical hyper intense lesions in corpus callosum and white matter in axial FLAIR MRI, Elliptical hyper intense lesions in corpus callosum in sagittal FLAIR MRI. D: Peripheral retinal artery occlusions in FFA



**Figure 2.** AB: Peripheral retinal artery occlusions in the FFA. CD: Lesions in corpus callosum in sagittal FLAIR MRI and elliptical hyperintense lesions in corpus callosum

FFA findings (BRAO and segmental hyperfluorescence and leakage) contribute greatly to our ability to make a diagnosis of Susac syndrome(2,15). Both of our patients had retinal artery occlusions in FFAs and benefited from IVIG therapy, the first patient without retinal sequelae and the second one with mild retinal sequelae. These occlusions are thought to be caused by endothelial injury and swelling, rather than emboli (2).

In Susac syndrome, a low frequency sensorineural hearing loss is most common. It is thought to be due to the microvascular ischemic injury of the apical cochlea, and may not be seen in some cases at the time of diagnosis or during the disease process (10,14). Thus, in our first case, the patient was unable to cooperate with audiometric tests because of encephalopathy, but the whispering test was reported as normal. He had no auditory complaints during the treatment. In the second case, our patient stated that he had had a hearing loss for a short period of time but

had not consulted a physician. He also had no auditory complaints during the follow-ups.

Key MRI characteristics of Susac Syndrome are the typical subcortical white matter lesions, particularly in the corpus callosum. The central part of the corpus callosum is expected to be injured, and as the active lesions resolve, central callosal "holes" may develop. Typically, the callosal lesions consist of round lesions ("snowballs") of various size, but linear defects ("spokes") may also be seen in the callosum. These white matter lesions tend to enhance while the disease is active. Lesions may be seen in other sbcortical white matter (particularly in periventricular regions) and also in deep grey matter. Leptomeningeal enhancement is common. Our two cases had MRI findings typical of Susac Syndrome. In the first case, some of those lesions were contrast-enhancing. There was leptomeningeal contrast enhancement in the first case, but not in the second. Microinfarction is the fundamental histopathologic pathology in the brain tissue, and this appears to be due to an ischemic microvascular endotheliopathy, with swollen endothelial cells (6,8,11,14).

In Susac Syndrome, CSF analysis may show high protein levels, pleiocytosis, or both. The oligoclonal band test is expected to be negative (5,6,8,16,14). In fact, high protein levels in CSF and oligoclonal band negativity led us to exclude multiple sclerosis in our cases. Protein levels being this high and oligoclonal band test being negative are important in differential diagnosis with multiple sclerosis (14,16).

A wide spectrum of EEG findings have been reported in susac syndrome, from nonspecific slow wave abnormalities to triphasic wave patterns that are consistent with encephalopathy, (6,10). Our first case had an EEG indicating encephalopathy.

Although Susac syndrome may be monocyclic and self-limited and may fully remit within several months, some patients experience early and/or late relapses, and some patients experience a much more prolonged and severe course of active disease than did our patients.

Because the immunopathogenesis and clinical course of Susac syndrome appear to have common with that much in of juvenile dermatomyositis (both representing autoimmune microvascular endotheliopathies, but affecting different triads of tissues), recommendations for treatment of Susac syndrome have been based on what has been effective and necessary to successfully treat juvenile dermatomyositis (JDM). As with JDM, there is a spectrum of treatment needs for Susac syndrome, due to a spectrum of disease intensity/severity and disease course/duration. As with JDM, a patient whose Susac syndrome is mild in severity and follows a relatively short monocyclic course will not need as aggressive or as prolonged immunosuppression as patients with much more severe and/or much more prolonged and/or relapsing disease. Our two patients were fortunate in that their Susac syndrome, though initially intense, subsided relatively quickly and followed a relatively mild and short course, such that they appeared to require no more than monthly IVIG, after the initial weeks of their illness. Other patients need much more aggressive and much more sustained immunosuppression. For example, it is sometimes difficult to control extremely intense/aggressive Susac syndrome even with а combination of cyclophosphamide, high dose coritcosteroid, IVIG, plasma exchange, and rituximab (20).

The long term outcome of Susac syndrome has not yet been well studied (13-,15). Some patients, like our two patients, have an excellent outcome; others experience a devastating outcome, with severe disability and severely diminished quality of life. Much depends on where a given patient's Susac syndrome falls along the spectrum of disease severity. Much depends on how well and how promptly a given patient's treatment needs are recognized and appropriately met. We considered these cases valuable to present because they were male patients, had somewhat unusual presenting symptoms, had no accompanying hearing loss, required relatively less aggressive and less sustained immunosuppression than usual, and have followed a relatively short and benign course, without relapse and with excellent outcome. They also serve as good examples of the apparent efficacy and sufficiency (in their cases) of IVIG.

### **Conflict of Interest**

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. The first case was presented as poster presentation at the 16th Congress of the European Federation of Neurological Societies, 8-11 September 2012, Stockholm, Sweden

#### References

- Barker RA, Anderson JR, Meyer P, Dick DJ, Scolding NJ. Microangiopathy of the brain and retina with hearing loss in a 50 year old woman: extending the spectrum of Susac'syndrome. Journal of Nurology, Neurosurgery and Psychiatry 1999;66:641-3
- O'Halloran HS, Pearson PA, Lee WB, Susac JO, Berger JR. Microangiopathy of the brain, retine and cochlea (Susac syndrome). A report of five cases and a review of the literature. Ophthalmology 1998;105:1038-42
- Schwitter J, Agosti R, Ott P, Kalman A, Waespe W. Small infarctions of cochlear, retinal and encephalic tissue in young women. Stroke 1992;23:903-7
- 4. Susac JO. Susac's Syndrome. Neurology 1994;44:591-3
- Susac JO, Hardman MD, Selhorst JB. Microangiopathy of the brain and retina. Neurology 1979;29:313-6
- 6. Bitra RK, Eggenberger E. Review of Susac syndrome. Curr Opin Ophthalmol.2011;22(6):472-6
- Greco A, De Virgilio A, Gallo A, Fusconi M, Turchetta R, Tombolini M, Rizzo MI, de Vincentiis M. Susac's syndrome--pathogenesis, clinical variants and treatment approaches. Autoimmun Rev. 2014;13(8):814-21
- Kleffner I, Duning T, Lohmann H, Deppe M, Basel T, Promesberger J, et al. A brief review of Susac syndrome. J Neurol Sci. 2012;322(1-2):35-40
- 9. Saw VP, Canty PA, Green CM et al. : Susac Syndrome: microangiopathy of the retina, cochlea and brain. Clinical and Experimental Ophthalmology 2000;28:373-80
- Dörr J, Radbruch H, Bock M, Wuerfel J, Brüggemann A, Wandinger KP, et al. Encephalopathy, visual disturbance and hearing loss-recognizing the symptoms of Susac syndrome. Nat Rev Neurol. 2009;5(12):683-8
- Oliveira P, Mayeux J. Teaching neuroimages: snowballlike lesions with sudden hearing loss. Neurology. 2014;82(12):100

- 12. Papo T, Biousse V, Lehoang P et al. Susac Syndrome. Medicine(Baltimore) 1998;77:3-9
- Petty GW, Matteson EL, Younge BR, Mcdonald TJ, Wood CP. Recurrence of Susac syndrome (retinocochleocerebral vasculopathy) after remission of 18 years. Mayo Clinic Proceedings 2001;76:958-60
- Susac JO, Egan RA, Rennebohm RM, Lubow MJ. Susac's syndrome: 1975-2005 microangiopathy/autoimmune endotheliopathy. J Neurol Sci. 2007;257(1-2):270-2
- Feresiadou A, Eriksson U, Larsen HC, Raininko R, Nygren I, Melberg A. Recurrence of Susac Syndrome following 23 Years of Remission. Case Rep Neurol. 2014 May 21;6(2):171-5
- Mateen FJ, Zubkov AY, Muralidharan R, Fugate JE, Rodriguez FJ, Winters JL, et al. Susac syndrome: clinical characteristics and treatment in 29 new cases. Eur J Neurol. 2012;19(6):800-11

- Dörr J, Ringelstein M, Duning T, Kleffner I. Update on Susac Syndrome: New Insights in Brain and Retinal Imaging and Treatment Options. J Alzheimers Dis. 2014; 42(3):99-108
- Erer S, Boz M, Taskapilioglu O, Hakyemez B, Kaderli B, Zarifoglu M. The first Susac's syndrome case in Turkey. J Neurol Sci. 2006 Dec 21;251(1-2):134-7
- Ozturk A, Degirmenci Y, Tunc M, Kececi H. Susac's syndrome: a case of simultaneous development of all three components of the triad. J Neurol Sci. 2013 Jan 15;324(1-2):187-9
- Rennebohm RM, Lubow M, Rusin J, Martin L, Grzybowski DM, Susac JO. Aggressive immunosuppressive treatment of Susac's syndrome in an adolescent: using treatment of dermatomyositis as a model. Pediatr Rheumatol Online J. 2008 Jan 29;6:3.

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# A dangerous joke: Colon perforation by an air compressor

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#### Abstract

**Objectives:** The most dangerous complication of air entry into rectum at a high volume and velocity in a short time are sigmoid colon rupture and pneumoperitoneum

**Case:** We present a 28-year-old male was brought to emergency with abdominal nausea, vomiting, pain, and abdominal swelling for 4 hours. Based on physical examination and laboratory data the patient was taken to operating theatre for suspected acute appendicitis. On exploration an excessive amount of gas distention was observed in whole colon with caecum being more distended. There were serosal linear defects that were bleeding, with the largest being 3-4 cm, mainly in rectum. Larger defects were primarily closed. A more detailed patient history was taken and it was found out that he was pumped high-pressure air by an air compressor into his anus while joking with his friends.

**Conclusion:** Air compressor abuse is one of the non-iatrogenic causes of barotrauma to colon. It usually occurs while working mates are joking with each other. In the rare cases where the cause of acute abdomen remains unclear it would be prudent to question the trauma history and use of an air compressor

Key words: Air compressor, Colon perforation, Emergency

#### Introduction

Colonic barotraumas are caused by pressure increase inside the intestinal lumen (1). The two probable result of colonic barotrauma are colonic mucosal injury and colon perforation (2). The most dangerous complication of air entry into rectum at a high volume and velocity in a short time are sigmoid colon rupture and pneumoperitoneum (3). In this paper we discussed a rare case colonic perforation that was caused by an air compressor.

#### Case

A 28-year-old male was brought to emergency with abdominal nausea, vomiting, pain, and abdominal swelling for 4 hours. Abdominal examination revealed a diffuse tenderness on palpation, guarding, and rebound tenderness that was more prominent at the right lower quadrant. Based on physical examination and laboratory data the patient was taken to operating theatre for suspected acute appendicitis. A Mc-Burney laparotomy was made under general anesthesia. On exploration appendix appeared normal whereas there was approximately 300 cc blood in the abdominal cavity and thus a midline incision was done. On exploration an excessive amount of gas distention was observed in whole colon with cecum being more distended.

There were serosal linear defects that were bleeding, with the largest being 3-4 cm, mainly in rectum and also in sigmoid colon, descending colon, and transverse colon (Figure 1). Larger defects were primarily closed, abdominal cavity was irrigated, a drainage catheter was placed in Douglas pouch, and the cavity was closed. A more detailed patient history was taken after the patient was woke up and it was found out that he was pumped high-pressure air by an air compressor into his anus while joking with her friends

#### Discussion

Colonic trauma due to iatrogenic highpressure air flow most commonly occurs during colonoscopy (2). Woltjen reported a caecal perforation count of 4 among 3000 colonoscopy procedures (4). Since cecum is the widest portion of colon, it is the most commonly and severely affected part of colon in colonoscopy (4-7).

Air compressor abuse is one of the noniatrogenic causes of barotrauma to colon. Air compressors are generally used in various industrial fields, factories, and cleaning machines.

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Their unsuitable and unconscious use may lead to serious consequences such as colonic perforation. It usually occurs while working mates are joking with each other (3, 7). Our patient's history was similar to the literature.

There are some cases in the literature that were managed by primary closure and loop colostomy (7, 8). Our case was also managed by primary closure and abdominal drainage. Air compressor abuse usually causes perforation of rectosigmoid colon while cecum is most commonly affected site in colonoscopy (2). Since the rectosigmoid region has a limited mobility than other segments, it is more susceptible to barotrauma (7). Our patient had also transverse colon lesions in addition to rectum and sigmoid colon segments. With respect to the trauma site, our case was similar to other previous reports.

The diagnosis of this condition is not very difficult to make when patient reveal their history of air compressor-induced injury. In some instances when history cannot be taken or verified, a trauma history and the profession of the patient should be questioned (2). Intraperitoneal free air on direct X-ray or computed tomography (CT) may suggest colonic perforation. Our patient was taken into operation with suspected acute appendicitis without X-ray and CT examinations. Primary repair or segmentary resection is the recommended approaches in the treatment of colonic perforations (8). Hence, our patient was treated by primary suturing of the defects and resection was not required since he had colonic perforation and he applied earlier at the course. He experienced no complications after the surgery.

In conclusion, one of the rare causes of colonic barotrauma is the exposure to an air compressor. Patients presenting with such an exposure are reluctant to give an accurate history due to social, religious, or psychological reasons. In the rare cases where the cause of acute abdomen remains unclear it would be prudent to question the trauma history and use of an air compressor. Exposure to an air compressor should be remembered as a possible cause of acute abdomen.

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Figure 1. Serosal linear defects

#### References

- Mulkipatil SY, Shirbur SN, Shankar gururaj. Rare presentation of colonic perforation due to severe barotrauma. Medica. 2014; 3: 140-145.
- Choi JY, Park KS, Park TW, Koh WJ, Kim HM. Colon barotrauma caused by compressed air. Intest Res. 2013; 11: 213-216.
- Kim SJ, Ahn SI, Hong KC, Kim JS, Shin SH, Woo ZH. Pneumatic colonic rupture accompanied by tension pneumoperitoneum. Yonsei Med J. 2000; 41: 533-5.
- 4. Woltjen JA. A retrospective analysis of cecal barotrauma caused by colonoscope airflow and pressure. Gastrointest Endosc. 2005; 61: 37-45.
- Orsoni P, Berdah S, Verrier C, et al. Colonic perforation due to colonoscopy: a retrospective study of 48 cases. Endoscopy. 1997; 29: 160-164.
- Anderson ML, Pasha TM, Leighton JA. Endoscopic perforation of the colon: lessons from a 10-year study. Am J Gastroenterol. 2000; 95: 3418-3422.
- 7. Suh HH, Kim YJ, Kim SK. Colorectal injury by compressed air-a report of 2 cases. J Korean Med Sci. 1996; 11: 179-182.
- Coffey JC, Winter DC, Sookhai S, Cusack SP, Kirwan WO. Non- iatrogenic perforation of the colon due to acute barotrauma. Int J Colorectal Dis. 2007; 22: 561-562

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# Nephrotic syndrome associated with breast cancer; when should it be treated?

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#### Abstract

**Objectives:** We hereby describe a 46-year woman with nephrotic syndrome and new-onset breast cancer. **Case:** The patient was admitted to our hospital with peripheral edema and a mass in her right breast. Membranous glomerulonephritis (MGN) was the etiology of nephrotic syndrome. MGN has been reported in association with a wide variety of solid neoplastic diseases, although rarely occurs as a complication of breast carcinoma. We report a rare and a resistant case of MGN, who got breast cancer diagnosis concomitantly. **Conclusion:** Patient had no improvement in proteinuria although breast cancer was under-controlled with chemotherapy and surgery

Key words: Breast cancer; glomerulonephritis; proteinuria; nephrotic syndrome

#### Introduction

Although nephrotic syndrome caused by membranous glomerulonephritis (MGN) has been reported in association with a wide variety of solid neoplastic diseases, it rarely occurs as a complication of breast carcinoma. We report a rare case of nephrotic syndrome caused by MGN, who got breast cancer diagnosis concomitantly and had persistent proteinuria after the treatment of malignancy. We review the literature on this unusual association.

#### Case

A 46-year woman was admitted to our department with symptoms of peripheral edema and a mass in right breast for a month. The patient had not been receiving any medications and systemic disease to cause the nephrotic syndrome. There was no history of fever, oral ulcers, joint pains, edema or rash. On physical examination, her blood pressure was 120/80 mmHg, pulse rate was 82/min, and body temperature was 36.5 °C. She had pretibial edema and masses in right breast and right axillary while the examinations of other systems proved normal. Laboratory evaluation revealed serum urea 53 mg/dl (10-50), creatinine 0.87 mg/dl (0.6-1.3), total serum protein of 33 g/L (64-83), serum albumin 13 g/l (35-54), 24-hour excretion was protein 5.1 gr, ervthrocvtes sedimentation rate 81 mm/h, hemoglobin: 12.4 g/dl, white blood cell count of 9.2 K/ul, other hematologic and biochemical parameters were normal. Automatic full urine testing showed +4 proteinuria. Urine microscopy revealed epithelial cells.

HbsAg, HBeAg, anti-Hbs, anti-Hbe, anti-HCV and HIV were all negative. CRP, C3, C4, IgG, IgA and IgM levels were normal. Antinuclear antibody, peripheral and cytoplasmic antineutrophil cytoplasmic antibody, antidouble-stranded DNA antibodies, anti-glomerular basal membrane antibody, rheumatoid factor and cryoglobulins were negative. Abdominal ultrasonography was normal. The patient underwent a renal biopsy for assessment of the histological features of the nephrotic syndrome and breast biopsy; diagnosed and membranous glomerulonephritis and invasive ductal carcinoma respectively. There was no evidence of distance metastasis. The patient was started on diuretic and neoadjuvant chemotherapy and performed a modified radical mastectomy two months later. Postoperatively taken adjuvant chemotherapy she had with cyclophosphamide 600 g/m2, adriamycin 60 mg/m2, 5-fluorouracil 600 mg/m2 and radiotherapy. Breast cancer was accepted as under control by ten months after the diagnosis. In terms of MGN, she was followed up with RAS blockade and diuretic treatment. The patient had no evidence of recurrence of renal disease for 16 months after diagnosis. After 16 months, laboratory tests revealed proteinuria (4 gr/24 h) and hypoalbuminemia (22 g/l) so we started prednisone as 1mg/kg/day dosage and cyclosporine as 4mg/kg/day dosage. At the third month of the therapy, proteinuria remarkable decreased to 1 gr/24 h, and the patient felt well

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#### Discussion

Nephrotic syndrome can be due to secondary causes. Depending on the age of the patient, cancer may be an important cause of nephrotic syndrome, as has been observed in as many as 22% of patients over 60 years presenting with MGN (1). A review of the literature shows that nephrotic syndrome is a rare complication of breast carcinoma (4%), although mostly occur in the carcinomas of lungs (49%) and gastrointestinal tract (23%) (2). The relationship between nephrotic syndrome and malignancy is inconsistent. According to literature, 40-50% of patients have symptomatic nephrotic syndrome before the diagnosis of cancer. In 15-20% of patients cancer precedes the diagnosis of nephrotic syndrome and in the remaining patients there is simultaneous presentation of both diseases (3). The nephrotic syndrome often constitutes the prodromal stage of neoplastic disease.

Pathogenesis of paraneoplastic glomerulopathies remains controversial and mainly two mechanisms were suggested. First, tumors are important sources of antigens that may induce the production of specific antibodies and cause the formation of immune complexes in blood, which can subsequently deposit in renal tissue. Second, antigens with high affinity for basement membrane can settle directly in renal tissue and induce in situ formation of immune complexes with circulating antibodies (4). Both mechanisms would explain the role of chemotherapy or radical surgery in the resolution of the paraneoplastic nephrosis.

Removal or irradiation of the tumor is usually associated with dramatic decrease of the proteinuria whereas recurrence of the neoplasm is followed by increased proteinuria (5). Although the diagnosis of paraneoplastic glomerulopathy should theoretically be sustained by clinical and histological remission after radical surgical removal or chemotherapy-induced complete remission of the tumor, only few papers have reported this (3). Besides, some authors have suggested that nephrosis could be due to derangements in cell-mediated immunity in general and not necessarily to tumorassociated antigens that directly damage renal tissue. For this reason, it is possible that such a syndrome responds to prednisone, which is a "symptomatic" rather than "etiological" therapy (5).

In this case, while cancer was controlled with chemotherapy and surgery, there was no improvement in proteinuria. We did not find a definitive recommendation for the time required to wait after curative tumor treatment in the literature for glomerulonephritis. We waited spontaneous remission of the disease for about 18 months after treatment. Remission of glomerulopathy was failed so we planned to immunosuppressive therapy. In view of the patient's good response to immunosuppressive therapy, our case report seems to support the second hypothesis. We therefore suggest that, despite the paraneoplastic origin of MGN, in these patients it is useful to administer prednisone and other antiproteinuric agent in addition to adequate treatment for the tumor itself.

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#### References

- Valcamonico F, Ferrari V, Simoncini E, Gregorini G, Vassalli L, Amoroso V., et al. Paraneoplastic Nephrotic Syndrome in Advanced Breast Cancer Patient. A Case Report. Tumori 2004; 90 (1): 154-156.
- 2. Eagen JW, Lewis EJ. Glomerulopathies of neoplasia. Kidney Int 1977; 11:297-306.
- 3. Pai P, Bone JM, McDicken I, Bell GM. Solid tumour and glomerulopathy. Q J Med 1996; 89:361-367.
- 4. Ozawa T, Pluss R, Lacher J, Boadecker E, Guggenheim S, Hammond W., et al. Endogenous immune complex nephropathy associated with malignancy: studies on the nature and immunopathogenic significance of glomerular bound antigen and antibody, isolation and characterisation of tumour specific antigen and antibody and circulating immune complexes. Q J Med 1975; 44:523-541.
- Gagliano RG, Costanzi JJ, Beathard GA, Sarles HE, Bell JD. The Nephrotic Syndrome Associated with Neoplasia: An Unusual Paraneoplastic Syndrome Report of a Case and Review of the Literature. Am J Med 1976; 60:1026-1031.

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