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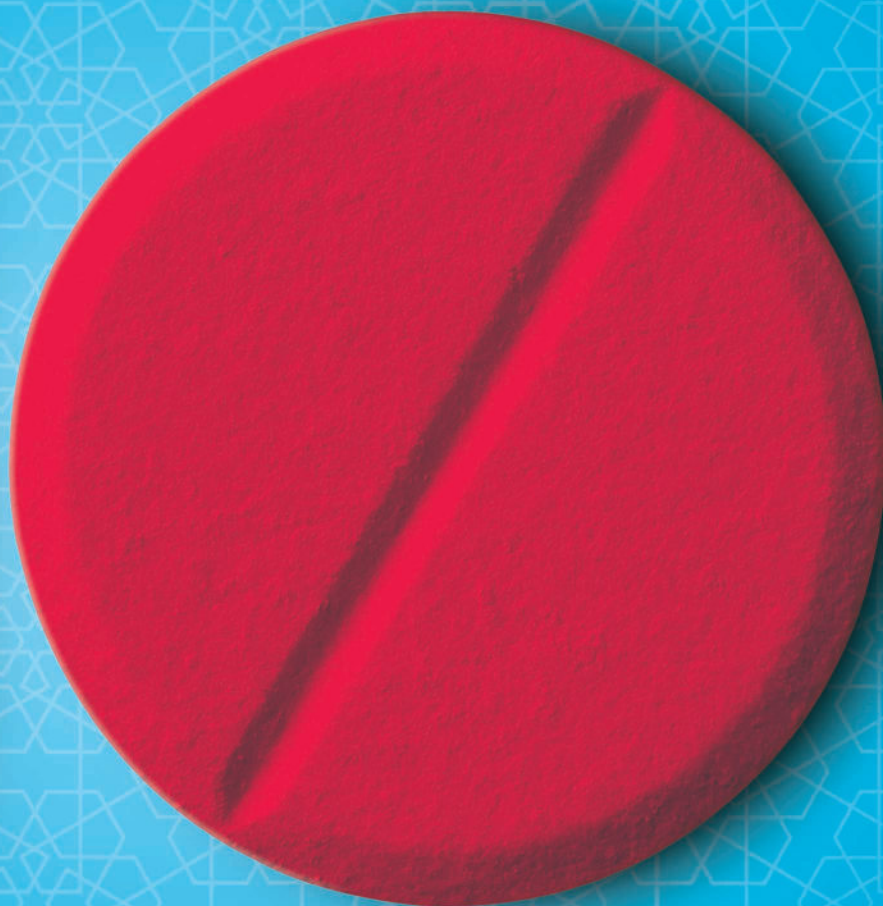
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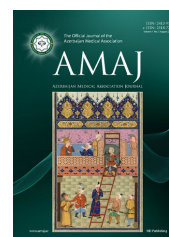
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Sotos Syndrome: A Case Report

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Sotos syndrome is an overgrowth condition accompanied by macrocephaly, distinctive facial appearance and mental retardation. It usually occurs sporadically.

The aim of the current case report was to emphasize the necessity of detailed evaluation, including genetic testing, in patients who have overgrowth combined with other dysmorphic features.

Keywords: overgrowth, mental retardation, Sotos syndrome

Introduction

The syndrome which includes overgrowth, acromegaly and mild mental disabilities was described in 5 patients by Sotos, in 1964 [1]. Although, there had been reported six generation family members with Sotos syndrome inherited by autosomal dominant type, most cases of disorder are sporadic. Its estimated incidence is 1:10000-1:50000.

Diagnostic criteria of Sotos syndrome are receding forehead hairline, macrocephaly, frontal bossing, long narrow face, sparse hair, characteristic chin, distinctive facial appearance, advanced bone age, and mental retardation [2,3]. Patients usually have excessive occipitofrontal circumference. Eye symptoms are often presented by nystagmus and strabismus. Anomalies of skeletal, cardiovascular, central nervous and urogenital systems can be also detected. The skeletal features include scoliosis, large hands and feet. There are numerous cardiovascular findings which may be presented in form of hypotonia, triventricular septal defect, ventricular septal defect, patent ductus anterior, and microvalval prolapses in tricuspid valve in 8 % of patients [4].

The morphological changes of the brain are detectable on MRI and include ventriculomegaly in 60-80% of patients and dysgenesis of corpus callosum. Impairment of cog-

nitive functions such as learning disabilities and speech problems may also be observed.

Case Report

A 13 year and 8 month old male patients were admitted to the Department of Pediatric Endocrinology of 19 Mayis University for overgrowth. The state of excessive growth had been observed for the last two years. Increase in body weight was not observed. No other complaints like headache, nausea, vomiting, fatigue, joint pains, excessive perspiration, thirst, urination, vision impairment or smell disturbances were found.

There was no history of drug intake, specific infections or prenatal trauma. Patient's weight at birth was 4800 g; however the height was not recorded. He began to speak when he was 1 year old and walk when he was 4 years old. He underwent appendectomy, tonsillectomy and adenoidectomy. He has been under neurologist's control since school years because of difficulties in learning which was diagnosed as a slight mental retardation.

The family history revealed that the patient's uncles were quite tall. The heights of the parents were normal and recorded as 152.1 cm for mother and 182.3 cm for father.

Physical examination revealed dysmor-

phic features, including sparse hair, long narrow face, large nose and chin, prominent forehead, and large sized head. In addition to these, prognathism and high arched palate were found (Fig. 1). According to the patients' parents all these features has been observed since his birth.

His anthropometric values were as following: height 178 cm (<97th percentile) with z-score 2, and body weight 75.7 kg (<90th percentile), occipitofrontal circumference 57 cm (<97th percentile), calculated target height 173.7 cm with z-score -0.37, height age 18 years and skeletal age 14 years.

The results of biochemical blood tests, including levels of Na, K, Cl, glucose, creatinine, Ca, P, ALT and AST (aspartate and alanine transaminases), triglyceride, cholesterol, HDL and LDL (high and low density lipoproteins), thyroid hormones, prolactin, total testosterone, FSH (follicle-stimulating hormone), cortisol and ACTH (adrenocorticotrophic hormone) were normal. However, growth hormones levels showed slight elevation: GH (growth hormone) 4.27* (range 0-3) ng/mL, GH (after 30 minutes) 5.64* (range 0-3) ng/mL, IGFBP-3 (insulin-like growth factor-binding protein) 7.25* (range 2.4-7.0) mg/mL, IGF (insulin-like growth factor) 1537 (range 183-850) ng/mL.

Genetic analysis using FISH (fluorescence in situ hybridization) method has revealed mutation of NSD-1 gene, located on chromosome 5, q35. Due to results of genetic analysis and characteristic clinic findings, i.e. dysmorphic features, mental retardation, high anthropometric values and exclusion of other causes of overgrowth, the patient was diagnosed to have Sotos syndrome.

Discussion

Abnormal growth and dysmorphic signs such as macrocephaly, prominent forehead and chin can be signs of a genetic syndrome. Examples of common overgrowth syndromes, among some with rare conditions, include Beckwith-Wiedemann syndrome, Marfan syndrome, fragile X syndrome, homocystinuria and Sotos syndrome.

Beckwith-Wiedemann syndrome is characterized by macrosomia, macroglossia, abdominal wall defects and hepatosplenomegaly.

Fragile X syndrome is associated with macrocephaly, moderate to severe mental retardation and delayed development.

Marfan syndrome is associated with connective tissue dysplasia and characterized by blue sclera, ocular lens dislocation, heart defects, pulmonary, skin signs, dural ectasia at lumbosacral level of spinal column.

Homocystinuria is a disorder of methionin metabolism characterized by mental retardation and recurrent venous thrombosis.

In case of absence of dysmorphic features, one has to differentiate between familial tall stature, precocious puberty, excess of gonadotropin hormone, hyperthyroidism and some other rare conditions.

In our case parents' height was normal. Values of the sex hormones and results of thyroid function tests were in acceptable ranges. The presence of dysmorphic features allowed us to suspect genetic disorder, which was further confirmed by the results of genetic test. The mutation of NSD-1 gene located at 5q35

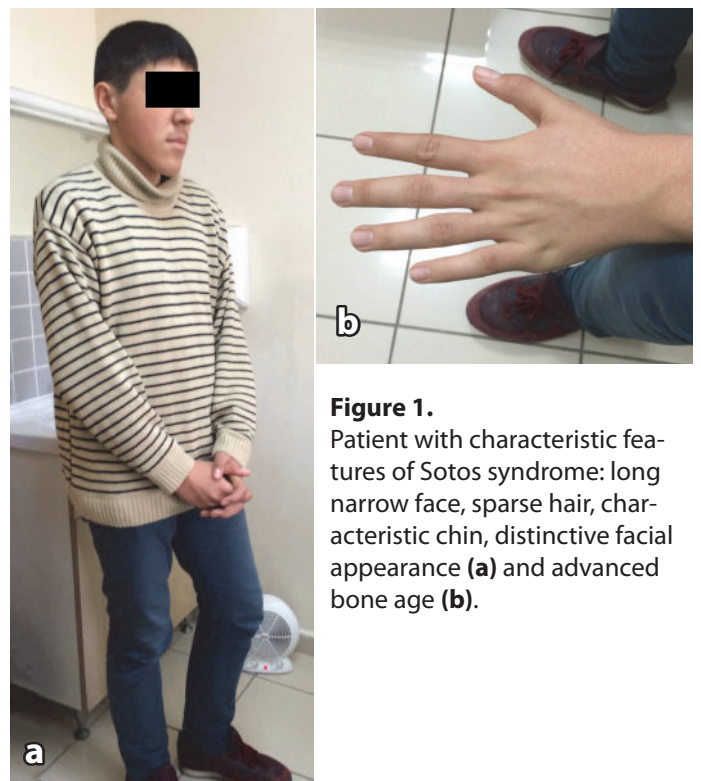


Figure 1. Patient with characteristic features of Sotos syndrome: long narrow face, sparse hair, characteristic chin, distinctive facial appearance (a) and advanced bone age (b).

locus was discovered. NSD-1 gene encodes histone methyltransferase. Deletions of this gene, which is located at 5q35 locus, are responsible for 75% of cases of Sotos syndrome [5]. However in 55% of Asians (especially in Japanese) and 10 % of Europeans, the negative results of genetic analysis don't deny the diagnosis.

Conclusion

Patients with increased height especially if this is combined with dysmorphic features and abnormal growth rates should be carefully investigated. It must be particularly emphasized, that in case of overgrowth combined with macrocephaly, dysmorphic features and mental retardations an investigation for Sotos syndrome is necessary for establishing proper diagnosis.

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Hemarthrosis: Concurrent acute presentation of pyrophosphate dehydrate and uric acid crystals in an elderly patient with a history of rheumatoid arthritis diagnosed with septic arthritis

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Concomitant septic arthritis in the presence of crystalline disease is a rare presentation of acute hemarthrosis and knee pain. Literature review showed that co-occurrence of these entities is an infrequent phenomenon but it needs to be acknowledged that these studies are few in number and were done on small patient population. This case challenges the notion that presence of crystals in the synovial fluid rules out septic arthritis even in the setting of low synovial WBC count. Additionally, the presence of pseudogout in patients suffering from gout is a rare entity as well. These findings in literature are described in case reports dispersed over the past three decades. We present a case where concurrent treatment of gout, pseudogout, and septic arthritis in a patient who presented with acute hemarthrosis.

Keywords: gout, pseudogout, rheumatoid arthritis, infection

Introduction

Age related crystal induced arthropathies are a common phenomenon presenting to the primary care office or the emergency department [1,2]. The two most common pathologies that are encountered are pseudogout (Calcium Pyrophosphate Deposition Disease - CPPD) and gout. Gout classically presents with podagra and/or severe pain involving the joints of the upper digits. With poor management of the disease, it can progress to multi-joint involvement and subcutaneous depositions known as tophi. CPPD on the other hand involves the knee joints at the onset with later progression to other areas. The management of these entities include acute pain control, chronic suppressive therapy, and avoiding dietary and/or social triggers. The concurrent presentation of these diseases can affect chronic management and lead to increased patient discomfort. However, the co-occurrence of these entities are rare [2,3].

Patients suffering from rheumatoid arthritis usually require long term steroid and possible immunosuppressive therapy. This leads to increased susceptibility towards life threatening or limb endangering infec-

tions [1]. Vigilance is needed to have a low threshold for ruling out septic arthritis in this patient population. This is usually done via synovial fluid analysis but as demonstrated in this case report, it does not necessarily provide a clear answer to the problem, especially in patients with multiple comorbidities. Even if the diagnosis is not clear, appropriate antibiotic therapy is necessary if suspicion remains. Physicians should use their clinical judgment based on patient presentation in order to arrive at proper diagnosis and initiate appropriate care plan.

Case Presentation

62-year-old Caucasian male with a history of gout (currently on allopurinol and colchicine), rheumatoid arthritis (on chronic methotrexate suppression therapy), multiple arthroplasties in the span of last 30 years due to complications of osteoarthritis presented to the ED with acute onset right knee pain and swelling status post exiting his vehicle. He did not endorse popping sensation or pain at that time but reported worsening pain within four to five hours with subjective fevers and chills. Patient was

in moderate distress due to pain in the emergency department and had limited mobility in his right leg as well. Patient was hemodynamically stable except for elevated blood pressure of 190/95 and low grade fever of 38.00 C, which at the time was attributed to severity of the pain.

Physical exam was remarkable for tenderness to palpation of the affected knee, significant swelling of the right knee with negative anterior or posterior drawer test. Initial labs showed elevated white blood cell count of 11,000 causing concern for possible septic arthritis, which led to the ED staff to perform arthrocentesis of the affected joint. Forty milliliters of serosanguineous fluid was extracted. Fluid profile showed 40,149 WBC and crystals. Patient noted partial relief from pain. Of note, coagulation studies were unremarkable but there was a mild elevation in CRP. Broad-spectrum antibiotics were initiated due to equivocal fluid analysis and patient presentation. Within the next two hours, return of erythema and swelling was noted on the affected knee. Hydromorphone PCA was initiated for the patient. MRI and x-ray was unchanged from previous studies done 5 years ago, which showed osteopenia, chondrocalcinosis and severe cartilage loss on the patella (Figure 1).

Following day, another arthrocentesis was performed by orthopedic surgery. Fluid showed serosanguineous profile as well. Analysis showed 17,420 WBC, 17% hematocrit, monosodium urate, and pyrophosphate dehydrate crystals. No bacterial, fungal elements, or AFB were seen in the initial culture analysis but cultures three days later showed *S.aureus* that was resistant to methicillin. Patient was started on naproxen 500 mg twice daily

for acute gout flare, 1.2 mg of colchicine initially then reduced to 0.6 mg daily. Broad spectrum antibiotic coverage was continued for the next three days and was switched to oral treatment regimen for fourteen days at the time of discharge. Patient noted significant relief two days later and was able to ambulate.

Discussion

Simultaneous presentations of pseudogout, gout with concomitant septic arthritis have been described in handful of case reports in patients suffering from chronic arthritic disease [2,3]. There have been handful reports describing these diseases occurring together in the last three decades. According to Stockman et al, 5.8% of patients (8/138) who suffered from gout had concomitant pseudogout [10]. Imaging findings show concomitant erosions and chondrocalcinosis associated with both types of crystalline disease, which is rarely seen on conventional imaging (Figure 2).

Diagnosis in this patient was relatively straightforward since both of the crystalline diseases were identified under synovial fluid analysis using careful polarized light microscopy. Furthermore, it is important to rule out serious pathology such aseptic arthritis by obtaining synovial fluid culture before starting treatment [6]. Our patient has a history of RA, which according to some studies indicate 15 times increased risk of septic arthritis, especially given the current disease-modifying anti-rheumatic drug (DMARD) therapy [1,4]. Given the patient had an elevated white count with mono-articular presentation, septic arthritis was higher on the differential versus crystal deposition dis-

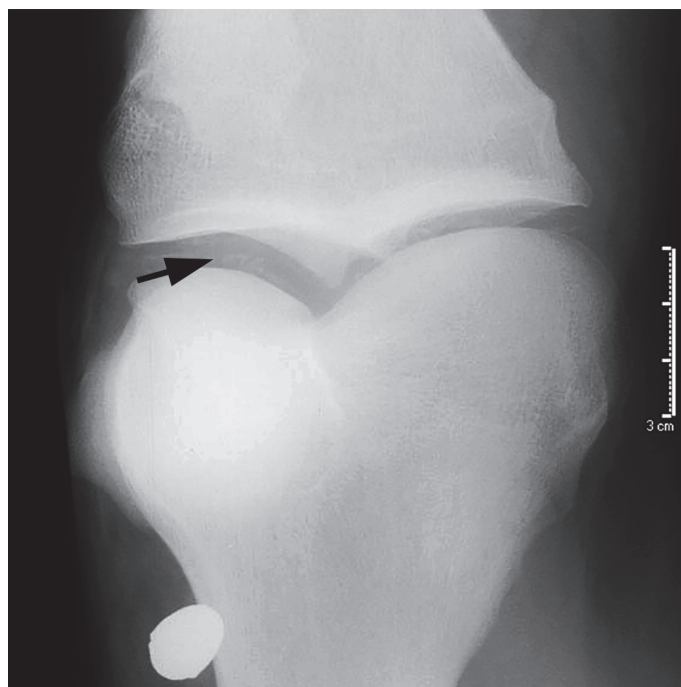


Figure 1.

Knee X-ray performed in the emergency department shows chondrocalcinosis (arrow) suggestive of severe long standing pseudogout as well as severe cartilage loss consistent with patient's history.



Figure 2.

Rare radiological coexistence of CPPD and gout in a patient with poor management of underlying disease. Classic punched-out lesions (black arrow) are seen with interspersed joint space chondrocalcinosis (white arrow).

ease. Nevertheless, synovial fluid analysis revealed the causative bacterial organism and the treatment was initiated accordingly.

This patient had extensive history of osteoarthritis with surgical interventions dating back to his early 20's as well as history of RA. Thus it would not be unusual for this patient to present with hemarthrosis after a minor trauma causing ACL or PCL tear [9]. This was ruled out from MRI even though it was of limited value due to patient's inability to fully comply due to pain. Hemarthrosis due to RA is not unusual, and was considered here as it was observed that a patient had a significant increase in synovial WBC's [9]. Conversely, RA induced flare usually have WBC count of above 50,000 with significantly elevated CRP and ESR. This patient's CRP level was not impressive at the time of presentation and marker levels decreased as expected with the initiation of therapy. It is also unusual to have an acute RA flare from minor trauma in a patient who has been compliant with therapy.

Spontaneous hemarthrosis of the knee should also raise suspicion for possible hemophilia but negative past history, family history, age of presentation and normal coagulation studies makes this unlikely at this stage. Regardless of etiology, proper acquisition and interpretation of synovial fluid is paramount for proper diagnosis and treatment of acute monoarthritis with presumed concomitant septic arthritis [5,6,10]. Shah et al showed that presence of either urate or CPPD crystals does not exclude septic arthritis with certainty. Their patient database showed 4/267 (1.5%) of their patients with the similar diagnosis [8]. Of note, all 4 patients had elevated WBC count above 50,000. Nevertheless, in a series patients followed by Weng et al, they found synovial count varying from 11,610 to 85,000 with the mean value of $44,102 \pm 30,306$ [8].

Treatment of simultaneous gout and pseudogout utilize same treatment regimen. Colchicine 1.2mg BID initially the first day and 0.6 mg BID thereafter is initiated with high dose twice daily NSAID (naproxen or indomethacin) [1]. However, in this case, due to suspicion of septic arthritis, the infectious disease team was consulted. Upon discussion of this case, the patient was started on broad spectrum antibiotic coverage as consequences for adverse outcomes would be significant. The regimen should be tailored to synovial fluid culture results.

Additionally, due to significant discomfort induced by the crystalline deposition, proper analgesic treatment with opioids may be necessary depending on the patient's pain tolerance levels [7]. If the patient does not show rapid improvement or is unable to ambulate, corticosteroid injection can also be implemented but septic arthritis needs to be ruled out prior to initiation. This was not the case with our patient as he was able to walk around using crutches within 48 hours of treatment initiation.

Conclusion

It is important to emphasize that utmost attention is needed when evaluating patients for septic arthritis with acute knee swelling. This is especially true in patients with history of rheumatoid arthritis treated with immunomodulatory drug regimens. In these patients, presence of crystals in the synovial fluid does not rule out septic arthritis. Correlating clinical and laboratory patient presentation can aid in rapid initiation of treatment.

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Free floating thrombus in right heart associated with pulmonary embolism: The effect of streptokinase

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Floating right heart thrombus in -transit originate from the deep veins of lower limbs, travel to the pulmonary arteries and is uncommon in patients with pulmonary embolism. This condition is a medical emergency due to the high risk of mortality. However, the most appropriate treatment is yet to be determined and there is no clear consensus in the literature on therapeutic management.

We have described two cases of deep vein thrombosis complicated with pulmonary embolism and right heart thrombus, which were treated with streptokinase and had diverse outcomes.

Keywords: pulmonary embolism, floating thrombus, deep vein thrombosis, streptokinase

Introduction

Free floating right heart thrombus-in-transit is a rare condition, which is commonly present with acute pulmonary embolism picture, including dyspnea (91,66%), chest pain (41,66%), syncope during exertion (16,66%), etc [1]. The large use of echocardiography has induced the distinguishing of free floating right heart thrombi [2]. Right heart thrombus is considered to be an extreme therapeutic emergency that can result in fatal pulmonary embolism (PE). The overall mortality rate in patients with right heart thrombus has been reported as 28% and as high as 100% in untreated patients [3]. We report two cases of pulmonary embolism that were found to have free-floating right atrial thrombi on echocardiography. In both patients, PE was confirmed by computer tomography (CT) scan with contrast and thrombolytic therapy initiated with streptokinase. These cases illustrate well the potential life threatening nature of free floating right heart thrombus and suggest that prompt intervention is necessary.

Case Presentation

Case 1. A 65-year-old man was referred

to our emergency room because of seven-day history of sudden onset and gradually worsening dyspnea. There was neither chest pain nor syncope. His past medical history was remarkable with left sided lower limb edema. His peripheral oxygen saturations fell to 85% on room air. During physical examination, his blood pressure was 110/70 mmHg with pulse 95 beats per minute. A transthoracic echocardiography was performed immediately and showed moderate enlargement of the right ventricle with systolic impairment and two free-floating thrombi in right atrium sized 27×23 mm and 33×22 mm consequently. This patient also had pulmonary hypertension with peak pulmonary pressure 70 mmHg. CT of the chest with contrast revealed thrombi in the distal branches of right and left pulmonary arteries, right-sided pulmonary effusion and “Hampton hump” sign in superior and posterior basal segments of the right lung which implies lung infarction. Meanwhile, thrombus in right atrium was also confirmed (fig. 1). Patient was admitted to intensive care unit and anticoagulation therapy with heparin was started. Laboratory studies showed D-dimer 2.16 mcg/mL, Troponin I 0.01 ng/mL, Creatine Ki-

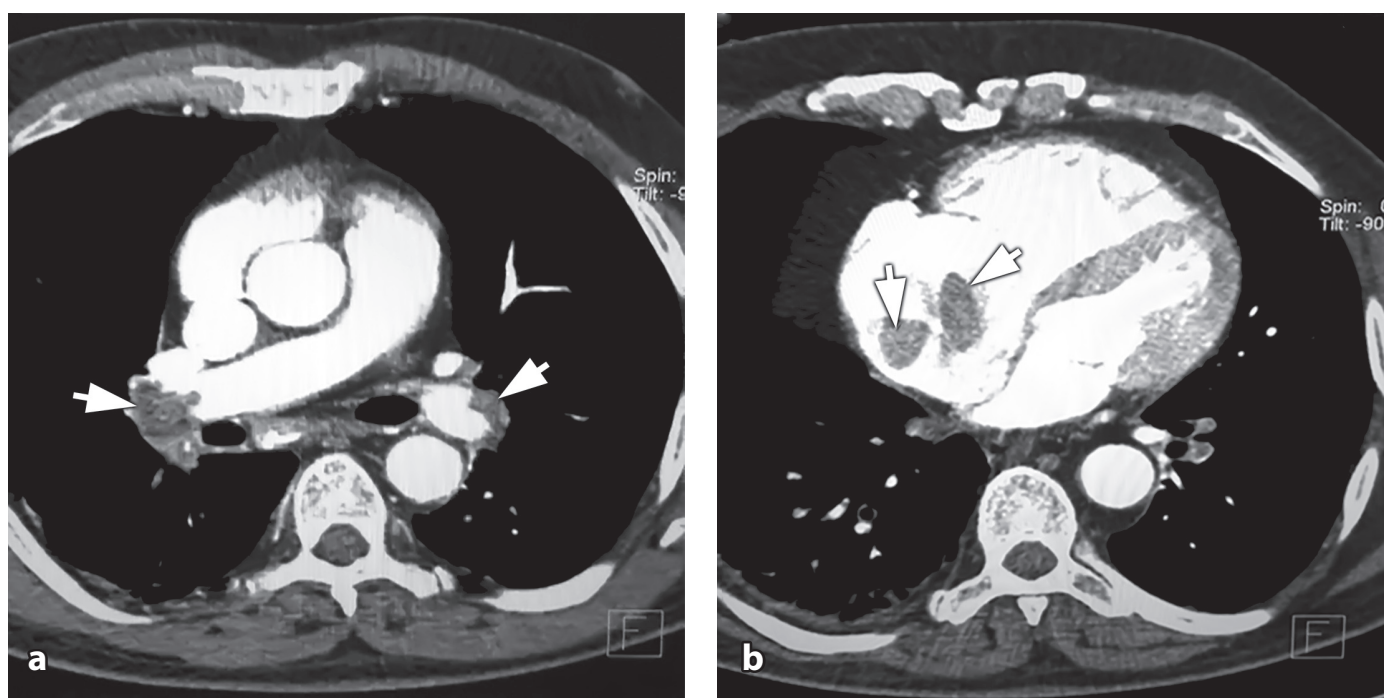


Figure 1. Pulmonary computed tomography angiogram showing (a) pulmonary embolism and (b) thrombus in right atrium (arrows).

nase - MB 1,7 ng/mL. A Doppler ultrasound of the lower limbs demonstrated right sided deep vein thrombosis of popliteal and femoral veins. After consultation with cardiothoracic surgeon, thrombolytic therapy with Streptokinase was initiated with loading dose 250000 IU in 30 minutes then with 100000 IU per

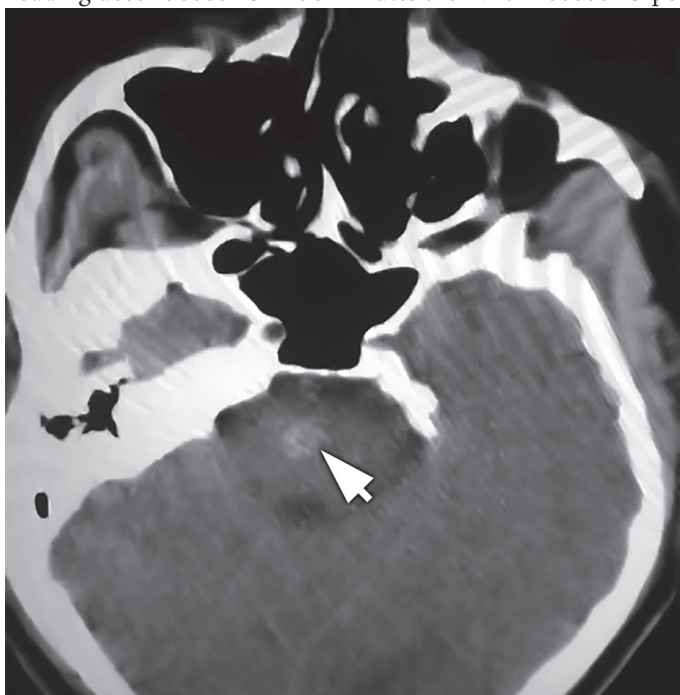


Figure 2. Computed tomography imaging of brain showing hyperdense region in brainstem associated with hemorrhage.

hour as continuous infusion. During the first minutes of infusion, patient suddenly worsened. Patient was sedated, intubated and connected to ventilator. High inotropic support was started. After 24 hour of thrombolytic therapy, repeated echocardiography showed lysis of one of the thrombi inside the right atrium. Streptokinase infusion continued for the next three days. Last echocardiography showed that right heart was free of thrombus and systolic pulmonary artery pressure dropped into the normal values. On the same day sedation was stopped and the patient underwent CT imaging of brain because of the questionable neurological status. CT confirmed small bleeding in brain stem (fig. 2).

Case 2. A 42-year-old man with past medical history of PE was admitted to emergency department with shortness of breathing, palpitations and syncope. He had a history of Diabetes Mellitus type 1. Patient started receiving anticoagulation therapy with warfarin one month prior to admission in our hospital. His physical examination was remarkable for tachycardia 110 bpm and tachypnea 32 rpm with a blood pressure 90/60 mmHg. Echocardiography showed mobile large thrombus in right atrium and relatively enlarged right ventricle with systolic dysfunction. It was not possible to measure pulmonary pressure. Laboratory studies showed an elevated D-dimer 121.5 ng/mL, INR 1.4 and troponin I 0.07 ng/mL. After initial stabilization and invasive monitoring, urgent CT of the chest was performed and revealed filling defects in both the right and left pulmonary arteries. Meanwhile, there was hypodensity inside the vena cava superior and filling defect in the right atrium. All the radiological findings were in favor of thrombi (fig. 3).

The patient suddenly developed cardiac arrest while diagnostic work-up and cardiothoracic surgeon consultation was going

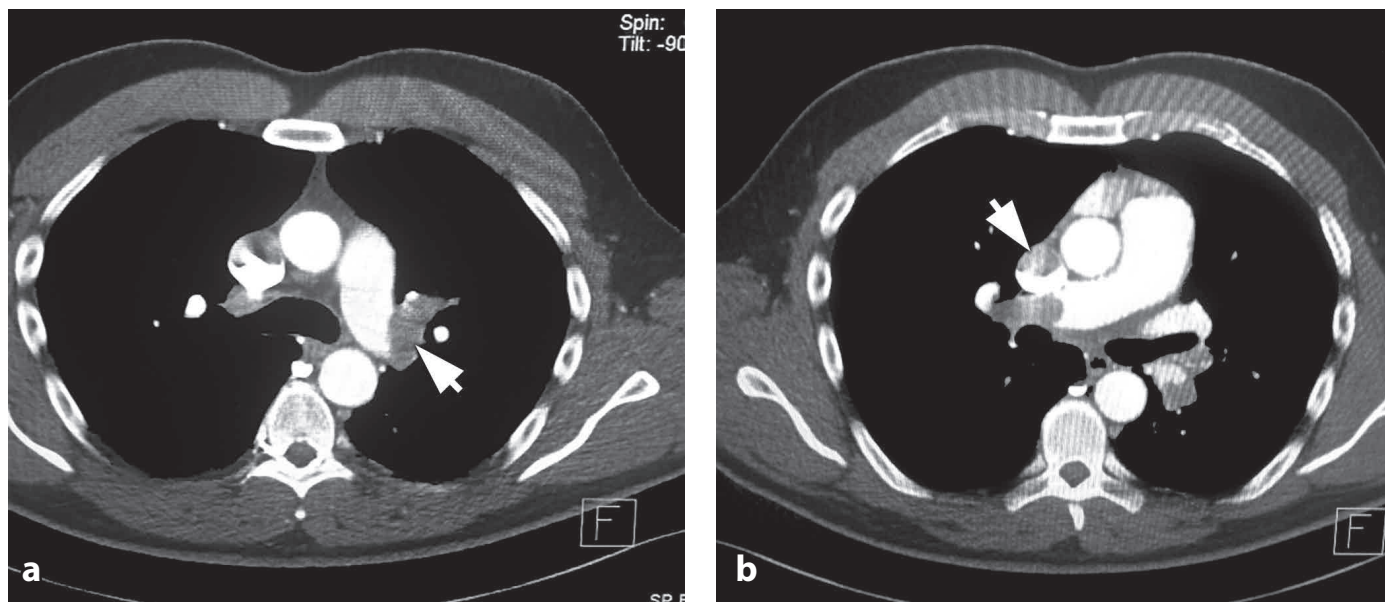


Figure 3. Pulmonary computed tomography angiogram showing filling defects in (a) left pulmonary artery and in (b) inferior vena cava (arrows).

on. During CPR streptokinase 1.5 million IU intravenous bolus dose was given as a step of despair to save patient. Despite of one hour of vigorous resuscitation the patient did not survive.

Discussion

Mobile right heart thrombi are quite uncommon and there are two morphological types of thrombi: type A and type B. there might be peripheral venous clots which accidentally lodge in the right heart on their way to the lungs (type A) or they may develop within the right heart chambers (type B). Type A thrombi are worm like shape and are extremely mobile. Type B thrombi are less mobile, attach to the right atrial or ventricular wall and are morphologically similar to left heart thrombi [4]. Our patient had mobile thrombi moving to and from into right ventricle which resembles type A thrombi. These are very dangerous and can critically worsen the hemodynamic. For this reason, free floating thrombi are an extreme therapeutic emergency and any delay to diagnosis and treatment could be lethal. From this point of view, transthoracic echocardiography is essential for diagnosis and must be performed systematically as soon as PE is suspected. This is an essential investigation that can be performed at bedside to directly visualize the thrombi, asses and monitor right ventricle function, and help making treatment decision. [5]. Despite, this is one of the therapeutic emergencies, but there is no clear consensus on how to manage it. Three options are possible: 1) single intravenous bolus heparin, with further supporting dose, while keeping the activated partial thromboplastin time at 2-2,5 times normal; 2) surgical embolectomy with exploration of pulmonary arteries and right atrium; 3) thrombolysis. Anticoagulation with heparin is reserved for haemodynamically stable patients who are not candidates for surgery/thrombolysis. In our patients, we had a therapeutic dilemma: surgery or

thrombolysis. Some studies reported that there was no expressive contrast between these therapeutic modalities in terms of in-hospital mortality. However, recent data indicate better outcomes with thrombolysis [5]. Cardiac surgery is preferred for very large RHT, tricuspid occlusion, associated paradoxical embolism via patent foramen ovale transit, thrombolytic failure or contraindications to thrombolytic therapy. In addition the theoretical advantages of thrombolysis are numerous. It accelerates lysis of thrombi and pulmonary reperfusion, minimizes pulmonary hypertension and raises right ventricular function. Moreover, it may destroy the clot at three locations at the same time: intracardiac, pulmonary and venous [5]. In our first case, thrombolysis worked very well, however there was side effect, i.e. intracranial bleeding. Besides the risk of major bleeding, thrombolytic therapy may be associated with a hypothesized risk of clot fragmentation and migration, complete pulmonary embolization or recurrent PE following partial dissolution of the venous thrombus. The occurrence of such an event in a hemodynamically unstable patient can lead to a catastrophic clinical course with severe hemodynamic compromise. However, there are numerous survivors who underwent thrombolytic therapy during cardiac arrest caused by PE [6]. Our second patient was diagnosed to have thrombi in right ventricle and proximal parts of pulmonary arteries. Such condition implies urgent surgical intervention. Unfortunately, patient had arrest during necessary diagnostic work-up. Bolus streptokinase infusion was tried as a step of despair, but failed. Nevertheless, we would like to emphasize necessity of early and aggressive use of thrombolytic therapy in patients suspected to have PE according to clinical findings. Further clinical trials are needed to elucidate all pros and cons of usage of streptokinase in similar cases.

Conclusion

Free floating thrombi in the right heart are rare and usually indicate travelling clots from the legs to the lungs. Echocardiography is main diagnostic tool in identifying the problem. However the option of optimal therapy for patients with PE with mobile right heart thrombi is still open to debate, but thrombolysis is readily available and effective.

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Supraclavicular Flap for Reconstruction of the Face

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The aesthetic and functional role of the human face can not be overemphasized. It is keystone in perception of self-identity and represents the most striking features of an individual's being. Being a place of concentration of major perceptive organs, like eyes, ears and nose, the face also has direct involvement in emotional and social communication.

Facial disfigurements may present in different forms, varying from minor nuisances to severe debilitating problems. The main goals in reconstruction of severely deformed face include restoration of function, comfort and appearance. Nowadays we have plenty of surgical modalities to fulfill these tasks, including cadaveric face transplantation. However, neither of the procedures can be considered as fully consistent in terms of achievable results.

Here we describe reconstruction of face by expanded supraclavicular flap. Two clinical cases are presented.

We performed a three-stage reconstruction, which included implantation of tissue expander in supraclavicular area, subsequent transfer of a fasciocutaneous flap onto the face, and finally, pedicle division of the flap with additional scar revision. A satisfactory fascial shape has been achieved.

We believe that supraclavicular flap, prefabricated by expansion is a powerful tool for autologous reconstruction of face and can be successfully used in selected cases.

Keywords: face, burn, reconstruction, expander, supraclavicular, flap

Introduction

Face has a great importance, both from aesthetic and functional point of view in a human life. It is keystone in perception of self-identity and represents the most striking features of an individual's being. Being a place of concentration of major perceptive organs, like eyes, ears and nose, the face also has direct involvement in emotional and social communication [1, 2].

Disruptions of face features vary in their severity from minor nuisances to severe debilitating problems. There are many causes which may make patient seek facial reconstruction. Mechanical traumas, burns and tumors are among major causes [2]. As final result the patient gets a conglomerate of scarred tissues which can interfere with

mouth, nose and eyes functions, cause painful or unpleasant sensation (including intense itching) and often burden the affected person with unbearable psychosocial problems [3].

The goals of reconstruction of severely deformed face include therefore, restoration of function, comfort and appearance. Nowadays, there are many methods of reconstruction of severely scarred facial skin. Small lesions can be directly excised and closed primarily either by linear suture or by adding Z-plastics if necessary. More large areas require local flaps, which are particularly usable in reconstructions around natural orifices. Burn scars, particularly those including 1 or 2 aesthetic areas can be covered by suitable skin grafts, i.e. from preauricular, mastoid or clavicular area [1, 3]. However,

this technique doesn't give predictable results in many cases. Complex problems, such as central facial tissue defects usually require free transfers of autologous tissues. Almost complete scarring of facial skin remains challenge for reconstruction. Face allotransplantation was offered as a solution in such cases since 2005. Although, preliminary results were promising there are still many issues related to donor selection, graft failure (rejection), comorbidities induced by immunosuppressive drugs, graft availability and ethical considerations [2].

Because of unique quality and quite a large area of the face many conventional techniques fall short in results of reconstruction of large facial defects. The supraclavicular area represents most suitable donor area for substitution of the skin of the face, being closer to face skin both in sense of texture and pliability. There are different methods of utilizing supraclavicular skin, including prefabrication by transferring different vascular pedicles in this area and usage of tissue expanders [4, 5, 6].

The usage of free flap for prefabrication definitely adds donor site morbidity and prolongs time of operation. Insufficiency of insurance cover and all consequences that follows should also been taken in consideration. Although we perform all kinds of microsurgical procedures we decided to use more simple technique on our patients, as their family members insisted on the use of procedures with no risk of total flap failure.

Supraclavicular flap is a fasciocutaneous flap based on branch of transverse cervical artery. It can be used as pedicled, free or even as perforator flap. As a regional flap it can be used for closure of defects in lower neck and lateral areas of the face [7].

Here we present 2 patients with face disfigurement, one after

burn and other after necrosis of vascular malformation. In both patients we used expansion of supraclavicular flap with subsequent transfer of the expanded flap to scarred area and pedicle division after 2 weeks from transfer.

Case Presentation

Case 1

A 15-year-old female was referred to our clinic for the treatment of severe face disfigurement. She was burned at age 5 having fell when jumping over a traditional fire place used on Novruz celebration.

Physical examination showed coarse scarring involving almost all the face, except the perioral area. There were pigmented patches of abdominal skin grafts on forehead and both cheeks. Both lower lids were pulled down by the scar tissues which caused moderate to severe ectropions. Moderate contractures were found on anterior cervical skin and submental area. (Fig. 1a).

We planned the operation in three stages. The first stage included subcutaneous implantation of 400 cc rectangular tissue expander into supraclavicular area. The expander was filled intraoperatively by injection of 10 cc of saline with subsequent regular filling 2 times a week in 20-25 cc amounts. After achieving overexpansion with 620 cc volume the filling stopped for 1 month. The second stage was then performed and consisted of removing of the expander and dissection of supraclavicular flap. After mobilization of the flap the reach of it was checked and scarred area of the cheek was excised. The flap was sewn on do-



Figure 1. a - Pre-op face appearance, note bilateral ectropion; b - Supraclavicular flap markings; c - Supraclavicular flap after expansion; d - Cheek after removing scars and raised supraclavicular flap; e - Supraclavicular flap transferred to the cheek with pedicle preservation.

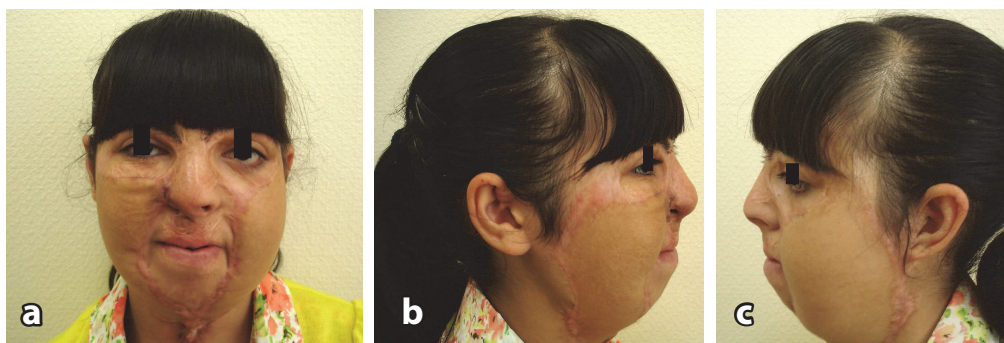


Figure 2. **a** - Final result after scar revisions, flap debulking and canthopexies, note normal lower lids; **b** - Final result after scar revisions, flap debulking and canthopexies, right side; **c** - Final result after scar revisions, flap debulking and canthopexies, left side.

nor area without disturbing pedicle. The donor site was closed primarily. On the 15th day after second stage the pedicle of the flap was constricted by elastic drainage. After confirmation of adequacy of the circulation the pedicle was divided and final scar revision was performed (Fig. 1 b-e). The same procedure was subsequently performed on the contralateral side.

The postoperative course was even. Hypertrophic scarring on some parts of suture line occurred what required additional scar revision procedures as well as conservative therapy in forms of intralesional steroids and silicone sheets wearing.

Additional partial debulking of the left flap and bilateral canthopexies were performed (Fig. 2 a-c).

As of the last control, the patient had more acceptable appearance. She reported increase in self confidence and social activity. She also developed very good tactile sensation on areas covered by the flap. The ectropion was corrected bilaterally. She continues conservative therapy against scar hypertrophy.

Case 2

A 9 year old female patient referred to us for treatment of face disfigurement. As a child, she got sclerotherapy for large lesion, (presumably large vascular malformation or hemangioma) which occupied major part of her left hemiface. This resulted in fulminant tissue necrosis with secondary infection and consequent scarring. Physical examination showed atrophic scarring involving left cheek, insufficiency of lower lip and loss of the lower 2/3 of the left external ear. We planned reconstruction with expanded supraclavicular flap. First, 320 cc rectangular expander was placed under the left supraclavicular area and gradually expanded twice a week until final size of 400 cc. Then, the expanded flap was transferred to surface of the left cheek after excising the scars. The pedicle of the flap left undisturbed. On the 15th day after second stage the pedicle of the flap was constricted by elastic drainage. After confirmation of adequacy of the circulation the pedicle was divided and final scar revision was performed (Fig 3 a-d).



Figure 3. **a** - Pre-op face appearance, note insufficiency of lower lip; **b** - Suprascapular flap markings; **c** - Suprascapular flap after expansion; **d** - Suprascapular flap transferred to the cheek with pedicle preservation.

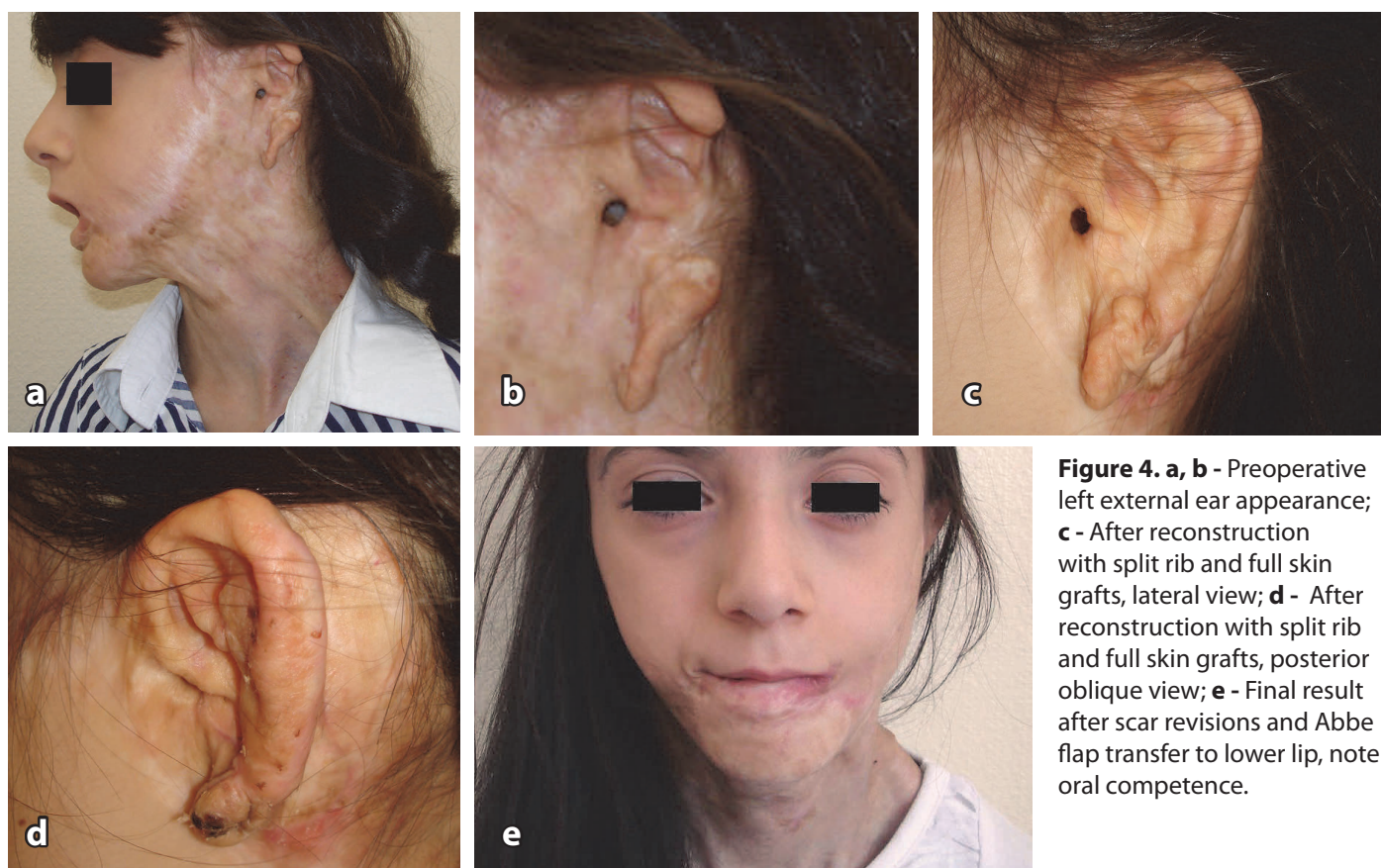


Figure 4. a, b - Preoperative left external ear appearance; c - After reconstruction with split rib and full skin grafts, lateral view; d - After reconstruction with split rib and full skin grafts, posterior oblique view; e - Final result after scar revisions and Abbe flap transfer to lower lip, note oral competence.

Additionally she underwent left ear reconstruction with rib cartilage (Brent's procedure), lower lip reconstruction with Abbe flap and mucosal advancement and suspension of the left corner of mouth to the zygomatic arch by prolene suture (Fig. 4 a-e).

As of the last control, the patient had more acceptable appearance. Her parents reported increase in self confidence and social activity. The skin sensation on the flap area was satisfactory. She has got more symmetry of the position of the lips in static state.

Discussion

Restoration of severely disfigured face still constitutes a challenge. The main goals of facial reconstruction include restoration of acceptable appearance to achieve positive impact on self-confidence and social communication, as well as augmentation of impaired functions. Main causes of fascial disfigurement include mechanical and burn trauma and defects after tumor resection [1, 2, 4].

Nowadays the large spectrum of reconstructive techniques of face can be viewed as repair with either autologous or alien tissues. The wide use of facial allotransplantation is still hindered by availability of transplants, need in life-long immunosuppressive therapy, ethical issues etc. Small defects of the face can be readily repaired by using conventional techniques like skin grafting or local flaps with sufficiently good results whereas gross disfigurements require transfer of ample amount of tissues, number of

operations and still fall short from the ideal [4, 5].

The use of supraclavicular skin for substitution of the face skin has been proposed long ago. However this area is limited in quantity of material. Numerous methods have been proposed to overcome this shortage, among which prefabrication of flaps by implantation of vascular pedicle and consequent expansion has gained popularity [3, 4]. Nevertheless, this adds technical challenges, prolongs the operational time and creates additional donor site morbidity. A different approach is provided by idea of suprascapular flap, which has been largely investigated in N. Pallua's works [7, 8, 9, 10]. This fasciocutaneous flap is based on branch (the supraclavicular artery) of transverse cervical artery. The vasculature of the flap is located at a point which is approximately 3 cm above the clavicle, 8.2 cm lateral to sternoclavicular joint and 2.1 cm dorsal to the lateral edge of sternocleidomastoid muscle. The size of the native flap can vary from 10x20 to 16x30 square centimeters. The dimensions of the flap may be extended far beyond cited ones by using of tissue expanders. The flap can be used as pedicled by tunneling it under cervical skin to the face, or as a free flap.

Because of safety reasons, to exclude the chance of pedicle incarceration we used three-stage transfer. The first stage included incision along clavicular lower border, development of subfacial pocket and placement of expander. After the period of expansion the next stage was performed. It consisted of remove of expander and full mobilization of the flap, which was transferred

to the face area without disturbing the pedicle. After flap “take”, the pedicle was divided on 15th postoperative day with simultaneous additional scar revisions.

Our patients had major disfigurement face, which prevented them greatly from social interactions, impaired self-confidence as well as created some functional problems like ectropion, nasal obstruction and insufficiency of the oral sphincter. Thus, there was need in both functional and aesthetic restoration. After the discussion of the available options we chose the usage of the pre expanded supraclavicular flap. As a result, we achieved quite acceptable external appearance of the face together with fixing of functional problems.

The pre expanded supraclavicular flap provides ample amount of similar skin for substitution of large scarred areas on the face. Presence of vascular pedicle allows the use of flap as a regional or even distant (free), without additional microsurgical prefabrication [9, 10]. To be sure of proper flap circulation the pedicle can be left over the neck skin until flap “take” on the donor area, which was done in our cases. We believe that usage of suprascapular flap, being both simple and effective, should be a method of choice in treating cases with major disfigurement of the face.

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The Role of Dermatologist in the Diagnosis of Systemic Langerhans Cell Histiocytosis in Adult Patient.

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Langerhans cell histiocytosis (LCH) is a malign proliferation of dendritic cells which are able to infiltrate any organ and system. LCH could be restricted to a single organ. In adults, LCH is usually restricted to the lungs. Skin involvement of LCH in adults is rare. However, in pediatric group, skin is a one of the predominant organ which LCH involves solely or as a part of systemic disease. Pediatric cutaneous LCH demonstrates more clear clinical view compared to adult cutaneous LCH. Skin lesions in pediatric cutaneous LCH usually affects the seborrheic regions of the skin, therefore to recognize and diagnose cutaneous LCH in children is unchallenging compared to adult LCH.

In present article, a 33-year old patient with polydipsia and polyuria has been diagnosed as a central diabetes insipidus due to tumoral infiltration of sella turcica. The nature of the tumoral lesion has been revealed by histopathologic examination of single skin lesion which has been detected and sampled by dermatologist. In this case, diagnosis of adult type of LCH was challenging. However, detailed and total skin examination paved the way toward the correct diagnoses and avoided from the transsphenoidal intervention.

Since the skin biopsy is a low risk diagnostic tool, detailed skin examination should be implemented and unusual skin lesion should be excised for histopathologic examination in a challenging adult LCH cases.

Keywords: langerhans cell histiocytosis, adult, Hand-Schüller-Christian disease.

Introduction

Langerhans cell histiocytosis (LCH) is a proliferative disorder of dendritic cells with a capacity to infiltrate any organ and system. Three clinical subtypes of LCH were described up to now: localized, chronic disseminated and acute disseminated. In terms of organ involvement, LCH is classified as: single system (SS) and multisystem (MS). SS LCH could be unifocal and multifocal. Its equivalent to eosinophilic granuloma, localized form of LCH. MS LCH without risk organ (RO) is considered as a chronic disseminated (Hand-Schüller-Christian disease), however, MS LCH with RO are equivalent to acute disseminated form (Litter-Siwe disease) [1]. Bone and skin involvement is more common for SS in children, whereas lung infiltration of LCH is more common

in adults [2,3]. Skin as a SS involvement of LCH is rare in adults [2,3]. According to International Histiocyte Society Registry, skin infiltration of LCH as a part of MS occurs frequently in both, adults and children [2,3]. However, German registry demonstrates that skin involvement of LCH in adults is not common (17%) [4]. In children, LCH is usually limited to the seborrheic areas. But skin lesions of LCH in adult patients are not well defined [5]. So, diagnostic value of dermatologist in pediatric LCH cases is more prominent than in adulthood. Here, we report a case of LCH with hypophysis infiltration that was diagnosed by single skin lesion.

Case Presentation

A 33-year-old male patient referred to

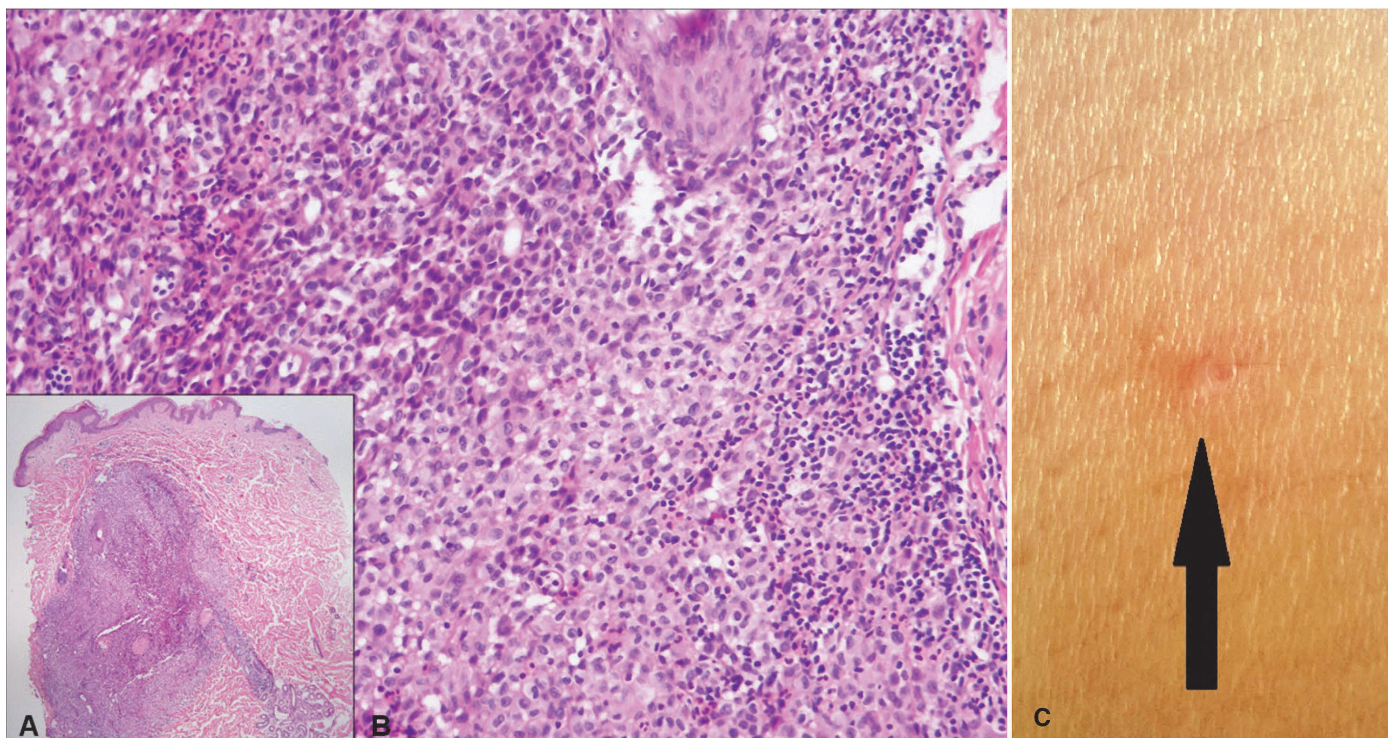


Figure 1. **A.** Nodular infiltration in the middle and deep dermis H&E x 40. **B.** Large, eosinophilic cytoplasm of Langerhans cells is evident H&E x 200. **C.** Single pink papule at the proximity of axillary fold.

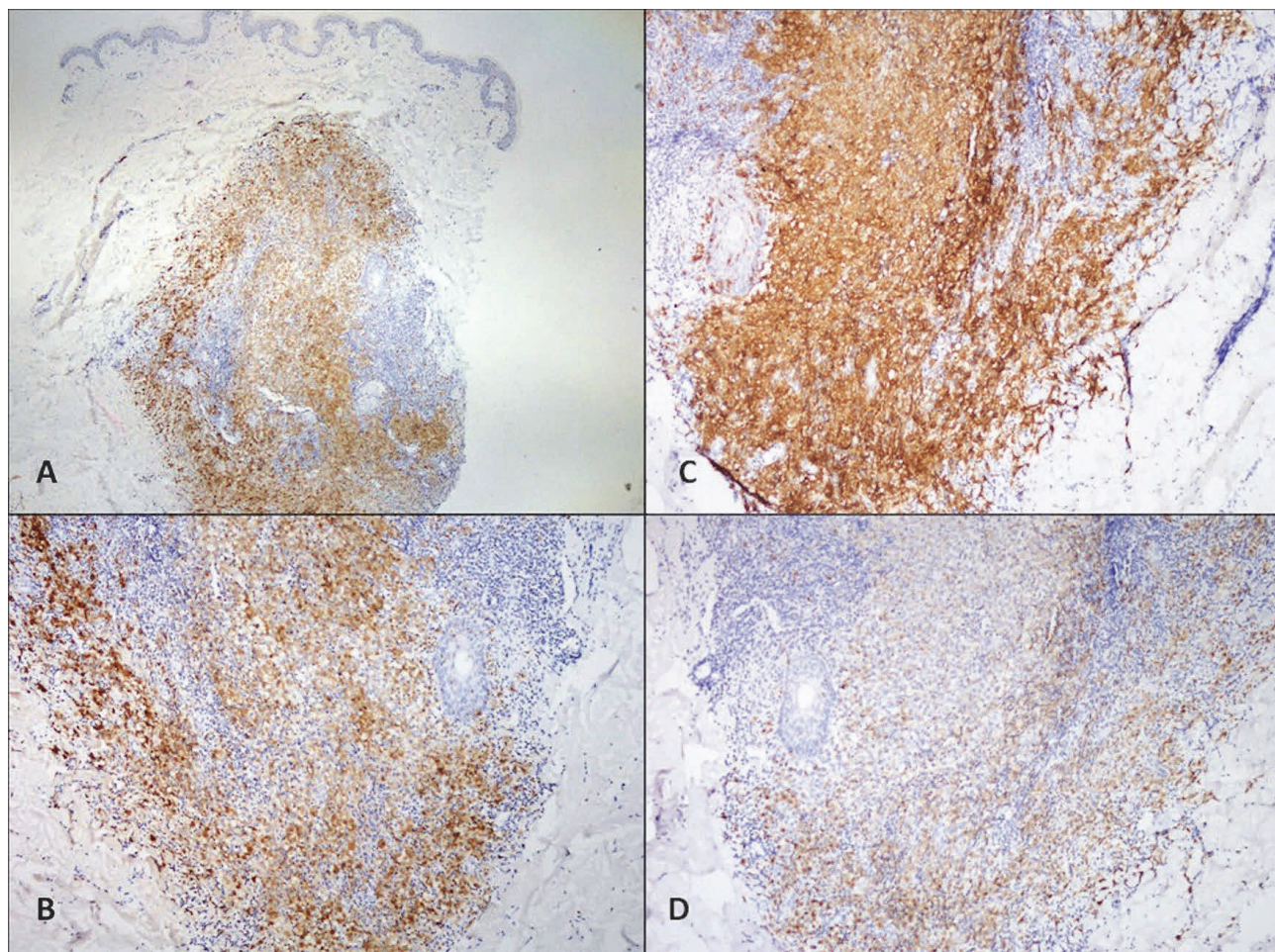


Figure 2. **A.** S100 positivity x 40. **B, C.** CD1 positivity x 100. **D.** Langerin positivity x 100.

dermatology department from endocrinology department to evaluate the patient in terms of skin involvement of LCH. He was hospitalized in endocrinology department due to polydipsia and polyuria that lasted for 6 month. After series examinations, patient was diagnosed with a central diabetes insipidus. Pituitary MRI revealed tumoral infiltration of sella turcica of unknown origin. LCH and sarcoidosis were the preliminary diagnosis. Systemic evaluation of the patient in terms of LCH and sarcoidosis revealed negative results. So, patient was referred to dermatology department for diagnostic purposes. Patient was thoroughly evaluated in terms of sarcoidosis and LCH. Single pink papula near the axillar region was found (Figure 1). "Apple jelly" sign was negative. It didn't look like folliculitis, hemangioma or melanocytic nevus. Seborrheic localization and atypical view of the lesion gave an idea to think about cutaneous LCH. The lesion was excised and histopathologically evaluated (Figure 1, 2). The nature of the hypophyseal infiltration was diagnosed by single LCH skin lesion in this patient. Ophthalmologic examination revealed minimal exophthalmos of the left eye. Macroscopic exophthalmos was absent. Lytic lesions were absent on bone survey. Lung and visceral organ involvement of LCH was negative.

Discussion

The role of dermatologist in the diagnosis of systemic LCH is very crucial, especially in adult cases. Therefore, in these group of patients, total skin examination should be performed thoroughly. Intertriginous regions should be carefully evaluated since LCH is prone to infiltrate seborrheic areas of the skin. Any atypical skin lesion should be sampled for histopathologic (HP) evaluation. In this case, systemic LCH was confirmed by single papula which was located at the proximity of seborrheic area. Because of detailed skin examination and HP evaluation patient escaped from invasive transsphenoidal intervention. Moreover, patient evaluated also in terms of chronic disseminated form of LCH, Hand-Schüller-Christian disease (HSCD). HSCD is more common in pediatric group. Exophthalmos, lytic bone lesions usually on skull, and diabetes insipidus due to pituitary stalk infiltration by LCH are classic triad of HSCD. However, classic triad presents only 25% of cases.[6] Probably, this case is an incomplete HSCD of adulthood which demonstrates skin involvement additionally.

Conclusion

In conclusion, total skin examination should be implemented thoroughly, in adults with systemic LCH suspicion. Skin lesions with an atypical view should be sampled for HP evaluation. Moreover, invasive diagnostic procedures such as transsphenoidal intervention bypassed in this patient due to skin biopsy technique which is a low risk diagnostic method. By presenting this article, it is emphasized once more that, the role of the dermatologists in the diagnosis of systemic LCH might be crucial.

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Deep infiltrating endometriosis surrounding T-shape copper IUD displaced into the lower anterior abdominal wall

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IUD insertion may cause potential dangerous consequences. A case of its migration to the lower anterior abdominal wall in a 36 year old patient was demonstrated. The T-shaped IUD was removed laparoscopically with surrounded tissue. Pathohistological examination of the removed tissue mass found out deep infiltrating endometriosis.

Keywords: Intrauterine contraceptive device, uterine perforation, migration, laparoscopy, endometriosis.

Introduction

Complications associated with the use of intrauterine contraceptive devices (IUDs) are the object of intent observation of gynecologists since they are implemented worldwide. Although, the method is considered relatively safe, it may cause some serious and potential dangerous consequences such as migration to abdominal cavity and adjacent organs due to the perforation of the uterus, which can lead to significant clinical problems [1,2]. Rare cases of abdominal wall swelling and abscess associated with unusual location for a displaced IUD are known due to the appropriate publications [3,4].

Case Presentation

A 36 year old patient (gravida 3, para 1) was admitted to our department after she underwent hysterosalpingography. She was a patient of infertility clinic following an incident of early miscarriage in 2012 with suspicion of IUD expulsion which was inserted 9 years ago. The patient was almost symptom free excluding mild pain in lower abdomen on the first day of her menses and missed threads of the coil. Abdominopelvic radiograph showed wandering IUD at right lower abdominal wall quadrant, approximately at the limits of the small pelvis (fig. 1). Sonography confirmed that the uterine cavity was empty.

She was operated by laparoscopic route. It was revealed that the IUD was migrated into the abdominal wall close to the bladder. Its location was marked by dense adhesions (fig. 2). Having freed from them and following the opening of retroperitoneum the target surrounded by infiltrated tissue was detected (fig. 3). En bloc dissection was impossible without getting into the bladder completely (fig. 4). After removal of the coil with tissue mass the hole in the bladder was sutured in two layers (fig. 5 and 6). Pathohistological examination of the removed tissue mass found out deep infiltrating endometriosis.

Discussion

According to the publications perforation of the uterus with IUDs occurs in about 0.12 to 0.68 per 1000 insertions [5]. The true incidence is likely higher because of the asymptomatic nature of the perforation [6]. The misplacement areas include the pelvis, peritoneal cavity and adjacent organs. Although, the majority of uterine perforations do not affect other organs, there are about 110 case reports about the migration of IUDs outside the uterine cavity and, in more than 2/3 of these cases they were located in the bladder with or without they being calcified [6,7]. Nevertheless displaced IUDs were also



Figure 1. Hysterosalpingography film which detected the coil outside the uterus.

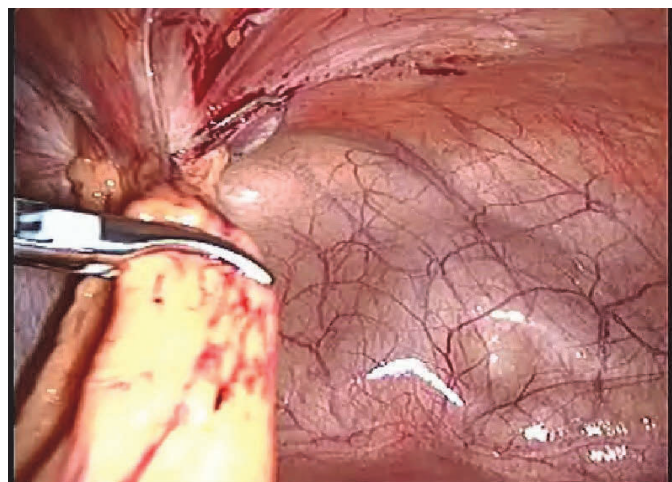


Figure 2. First look by entering into abdominal cavity.

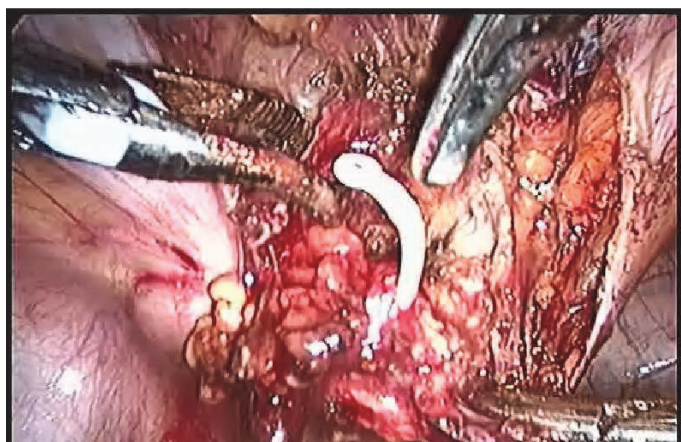


Figure 3. Detection of the IUD surrounded by dense fibrotic tissue in the abdominal wall.

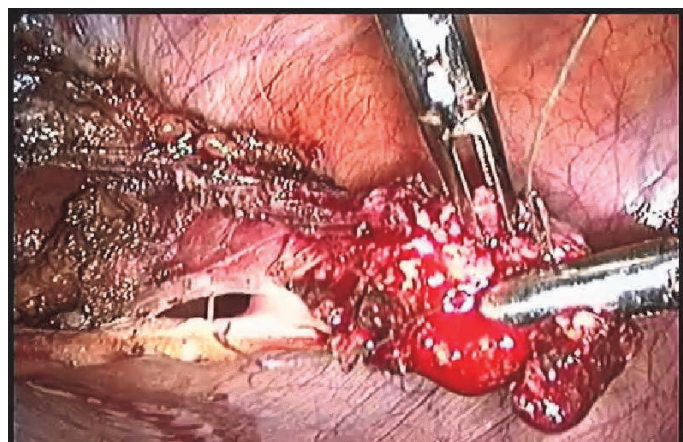


Figure 4. Opening of the bladder by en bloc dissection of the IUD with surrounded tissue.

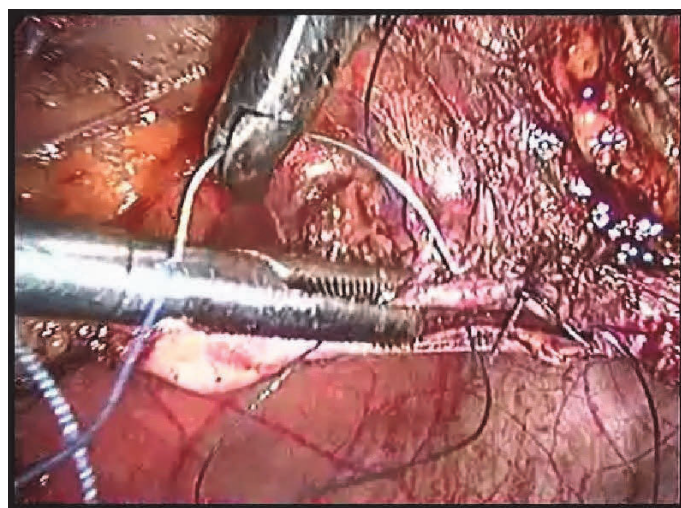


Figure 5. Sutured hole in the bladder

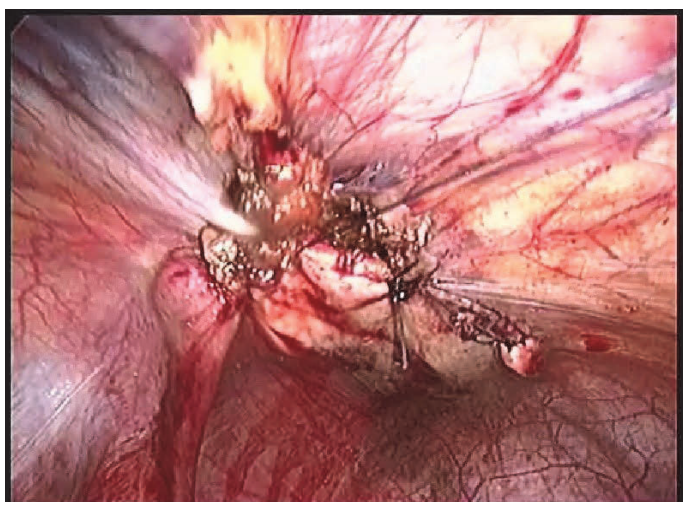


Figure 6. The last suture: peritonization

found in rectosigmoid colon [1, 8], loops of mid ileum [2], mesentery [9], omentum [1,2], and even gastric serosa [10].

The exact mechanism that causes uterine perforation and migration of the IUD is not entirely known. There are some predisposing factors discussed in the literature, such as the uterine size, position, timing of the insertion, congenital uterine anomalies and former operations like previous Cesarean section [3].

A translocated IUD induces a dense fibroblastic reaction, granuloma development, cystic lesions and even abscess formation [3,4].

Revealed deep infiltrating endometriosis (DIE) in surrounding the IUD tissue in our case is an interesting finding which by, all means, deserves attention and requires further considerations concerning the questions 'how' and 'why'. No previously published report concerning such association was detected by Pubmed searching.

Missing strings during vaginal examination or unexpected pregnancy in patients with IUDs suppose its expulsion, though clinicians should assume that it is either dislocated or migrated until it is documented by visualization. [1,3,5] Even the presence of an IUD string visible through the cervical os is insufficient to exclude the possibility of a dislocated IUD [1,3,6].

The current guidance is that all misplaced IUDs should be surgically removed [1,5]. Therefore, the value of preoperational diagnostics cannot be underestimated.

To evaluate whether an IUD is within the patient (inside the uterus or dislocated) or expelled, a plain X-ray film is the first diagnostic procedure [4]. Transvaginal sonography should be combined with abdominal X-ray to reach a definitive diagnosis [5].

However, sonography cannot accurately demonstrate the extent of myometrial or bladder or intestinal wall perforation, especially when the IUD has completely migrated outside of the uterus [5,7]. El-Hefnawy et al suggested that noncontrast CT be included in the differential diagnosis to depict the site of the dislocated IUD, anatomic relation between it and organs involved, and the extent of bladder injury [7].

As a majority of surgeons we have chosen laparoscopic route to remove the IUD. Based on personal experience, we can state that, to make the surgery more convenient and to avoid intra-operative 'surprises' the necessity of combination with hysteroscopy, cystoscopy and colonoscopy depending on situation is to be discussed.

Conclusion

Regardless of the fact that IUD insertion is a relevant and relatively safe method of contraception close follow up is needed to detect complications and subsequent management. DIE in tissue masses around the migrated to the abdominal wall coil is another attention deserving issue.

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Whole body hypothermia treatment and results in newborns with perinatal asphyxia: a case series.

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Hypoxic ischemic encephalopathy (HIE) secondary to asphyxia clinically manifests as acute or sub-acute brain injury. Out of all newborns with HIE, 15-20% die in the postnatal period, while 25% sustain severe and permanent neurological damage (cognitive delay, cerebral palsy and epilepsy). Early application of whole body hypothermia therapy (the lowering of body temperature to 33.5-34°C) has been shown to lower disability and mortality rates in newborns with HIE. To date, we have used therapeutic hypothermia in 7 neonates born in our clinic with severe asphyxia. One of the patients died; 6 have been discharged and are showing normal neurological, mental and physical development.

Keywords: newborn, perinatal asphyxia, hypoxic ischemic encephalopathy, therapeutic hypothermia

Introduction

Perinatal hypoxic ischemic encephalopathy (HIE) is seen in every 3-5 full-term live births out of 1000 [1]. HIE secondary to asphyxia clinically manifests as acute or sub-acute brain injury. The main causes of HIE are systemic hypoxemia and low brain blood flow [2-4]. Out of all newborns with HIE, 15-20% die in the postnatal period, while 25% develop severe and permanent neurological deficit (cognitive delay, cerebral palsy and epilepsy). Early application of whole body hypothermia therapy (the lowering of body temperature to 33.5-34°C) has been shown to lower disability and mortality rates in newborns with HIE. ILCOR has recommended therapeutic hypothermia as first line treatment for moderate and severe HIE in full-term or late preterm neonates since 2010 [6, 7]. Experimental studies have demonstrated that lowering the brain temperature by 2-3°C after hypoxic ischemic damage slows down the energy metabolism and reduces neuronal death [8]. As far as our knowledge goes, whole body hypothermia has never been used in our country before. The aim of this report is to present the results of our application of this method to treat newborns with severe and moderate HIE since November 2015.

Materials and Methods

Whole body hypothermia was used in 7 patients born at our clinic between November 2015 and June 2016. The treatment was administered with the non-invasive HI-CO-HYPOTHERM 550 temperature management system.

Treatment was started in patients based on the following AAP criteria: gestational age ≥ 36 weeks, age ≤ 6 hours, 10 minute Apgar score ≤ 5 , the need for resuscitation for 10 minutes after birth, cord blood pH < 7.0 or BE ≥ -16 mmol/l, clinical signs of moderate or severe encephalopathy, seizures or abnormal activity on EEG (9).

The patient group was comprised of newborns that fulfilled at least 3 of the above criteria. Treatment was started in all patients within the first hour of life; body temperature was lowered to 33.5-34°C and raised back to normal after 72 hours.

Patients had gestational ages of 38-40 weeks, 1 minute Apgar scores of 3-5 and 5 minute Apgar scores of 3-6. Acidosis was observed in blood samples of all patients (pH ≤ 7.0 ; BE > -16). Three patients had meconium-stained amniotic fluid. One patient had 3rd degree HIE, 4 - 2nd degree HIE, and 2 - 2-3rd degree (Table 1).

Table 2. Laboratory parameters of cases

New born #	Gestational Age, Weeks	Apgar score		Initial blood sample			Encephalopathy degree	Clinical seizures	Seizures on EEG
		1 min	5 min	pH	BE	Lac-tate			
1	40	1	5	6,8	-20	12	2-3	-	+
2	40	3	6	7,0	-17	10,8	2	+	+
3	39	2	4	6,9	-25	>15	2	-	-
4	38	1	5	7,0	-21	-	2-3	-	-
5	38	3	6	7,0	-18	13	2	+	+
6*	38	1	3	<6,8	-	>15	3	+	+
7	39	1	4	6,9	-23	>15	2	-	-

*This patient was transferred to another hospital on postnatal day 8 and died there during follow-up.

Results

All patients who underwent therapeutic hypothermia were born via natural labor. In all cases, initial resuscitation procedures were carried out and treatment was started within one hour. During the 72-hour treatment, ECG showed sinus bradycardia in 5 patients. Clinical seizures were observed in 3 patients, while 4 had abnormal activity on EEG. Neurosonographic examination revealed no abnormalities. Eye fundus examination showed subretinal hemorrhage in 4 patients. Based on the clinical tableau and EEG results, 4 patients were started on phenobarbital. Six patients were discharged following 72 hours of treatment with normal neurological outcome. Regular follow-up exams of all six continue to show normal physical, mental and neurological development. On postnatal day 8, the intubated patient with the poorest clinical tableau and blood test results was transferred to another clinic as per the wishes of the family, where he died during follow-up.

Discussion

Neonatal deaths represent 41% of all annual pediatric deaths under the age of 5. A quarter of these occur in the first week of life. Various birth complications are the main causes of neonatal death (10). Approximately 25% of neonates born with cord blood pH<7 suffer permanent neurological damage and death (11). The perinatal brain is particularly susceptible to hypoxic ischemia, cerebral palsy and the resulting permanent damage (12). Asphyxia has a major impact on neonatal morbidity and mortality and developmental prognosis (5). Therapeutic hypothermia is the only currently known treatment that has been shown to improve neurological outcomes and disability and mortality risks in neonates with hypoxic brain injury. Hypothermia treatment was first used in newborns in 1960, and research and trials in the following decades demonstrated the efficacy of hypothermia in protecting the brain from the effects of perinatal oxygen deprivation.

As far as knowledge goes, the neonatal intensive care unit at our clinic is the pioneer of this treatment method in the Azerbaijan Republic. Of the 7 patients who were treated, 1 died after being transferred to another hospital and 6 were discharged within a week with regular follow-up showing no mental or

physical delays. The EEGs of patients who initially showed abnormal activity have now reverted to normal and phenobarbital has been gradually reduced and stopped. All cases of subretinal hemorrhage in the neonatal period have now shown reabsorption.

In conclusion, therapeutic hypothermia in neonates with hypoxic ischemic damage is a demonstrably effective line of treatment that merits wider implementation.

Conclusion

Based on the results of hypothermia treatment trials run in our clinic and similar results reported worldwide, we can conclude that hypothermia is the most effective currently known method of treating neonates with hypoxic ischemic encephalopathy.

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Corneal Measurements in patients with Diabetes Mellitus

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Purpose: To estimate the corneal measurements using Scheimpflug camera in patients with diabetes mellitus.

Methods: Twenty five diabetic patients were prospectively recruited. Two groups were stratified, diabetic group and control group. Central corneal thickness (CCT), keratometry values (Kmean and Kmax), corneal volume (CV), anterior chamber depth (ACD), anterior chamber volume (ACV), Qvalue, frontal and back elevation, and the parameters of corneal variance indices, including minimum radius (Rmin) were measured using Pentacam Scheimpflug camera. Endothelial cell density (ECD) was also recorded. Findings were evaluated and compared between the 2 groups.

Results: Two groups were found to have different Kmax ($P = 0.03$, one-tailed) and Rmin ($P = 0.04$, one-tailed) parameters. There was no statistical significant difference between the 2 groups in CCT, Kmean, CV, ACD, ACV, frontal/back elevation, Qvalue, ECD, and parameters of corneal variance indices.

Conclusions: Diabetes mellitus affects keratometry and radius values of the human cornea based on the corneal measurements from Scheimpflug camera.

Keywords: diabetes mellitus, diabetic cornea, corneal measurements, Scheimpflug camera, Scheimpflug measurements.

Introduction

Corneal morphological evaluation is always very crucial in ophthalmologists' clinical practice. In fact, physicians rely on corneal parameters such as central corneal thickness, anterior and posterior corneal curvature, anterior chamber depth or endothelial cells counts to make diagnosis, to follow up or to plan treatments for refractive defects or diseases such as glaucoma, keratoconus, corneal ectasia or cataract [1-6]. Even if last developments have supplied ophthalmologist with very reliable devices, it is always important to pay attention to the limitations of these instruments and to some clinical situations that could bias their precision in corneal power evaluation [7-13].

Hyperglycemia has toxic effects on almost all cells in the body. [14] Ophthalmic complications of hyperglycemia are most remarkable in cornea and retina. Retinal

impairment accounts for the majority of visual loss of diabetic patients [14]. Diabetic retinopathy is the most common cause of blindness for people over the age of 50 [1].

Diabetes mellitus has a significant detrimental effect on the morphology, physiology, and clinical appearance of the cornea. The diabetic tear film is composed of a 4-fold higher glucose level than that of normal tears. Changes also manifest in the corneal epithelium, epithelial basement membrane complexes, stroma, and endothelium [15-18]. Studies show that the eyes of patients with diabetes have a greater central corneal thickness (CCT) and that there is a positive correlation between CCT and the degree of diabetic retinopathy [19-21]. Corneal hydration control also appears to be compromised in corneas of diabetic patients [22,23].

The purpose of this study is to analyze corneal morphological parameters

measured with Scheimpflug camera in DM type 2 patients and to compare them with those evaluated in healthy subjects (HS). According to our knowledge, this is one of the first papers about this topic.

Materials and Methods

This is a preliminary prospective study. It enrolled patients from 27 to 79 years of age, who visited the clinic from August 2014 to December 2014. Study population was divided into two groups: first group consisted of diabetes mellitus (type 2) patients and second group was considered as a control group of HS. Patients were excluded from the study if they had a history of corneal pathology or any ocular surgery. None of the diabetic patients had any symptoms of diabetic retinopathy. Both eyes were examined at the same time in both groups. A complete medical history was taken, complete ophthalmic exam and Scheimpflug Camera scan (Pentacam, Oculus, Wetzlar, Germany) were performed. The central corneal thickness (CCT, μm), keratometry values (Kmean and Kmax, D), corneal volume (CV), anterior chamber depth (ACD), anterior chamber volume (ACV), Qvalue, frontal and back elevation, and the parameters of corneal variance indices, such as Index of Surface Variance (ISV), Index of Vertical Asymmetry (IVA), Central keratoconus Index (CKI), Index of Height Asymmetry (IHA) and Index of Height Decentration (IHD), minimum radius (Rmin) were recorded and used for statistical analysis. Endothelial cell density (ECD) was also recorded using a noncontact specular microscope Topcon SP-3000P (Topcon Corp., Tokyo, Japan). Every participant underwent 3 measurements both with Pentacam and with Topcon SP-3000p and average values were taken for statistical analysis.

Every participant was informed about the purpose of the study and had to give informed consent before inclusion. The study was performed in adherence to the tenets of the declaration of Helsinki and Institutional Review Board approval was obtained.

The results were expressed as the mean \pm standard deviation (SD). The normality of the data was tested with the Shapiro-Wilk test. The difference between the 2 groups was assessed using an unpaired t test; if the data was not distributed normally, the Mann-Whitney U test was performed instead. All calculations was performed using IBM SPSS statistical software (version 20, SAS Institute, Inc.). The level for statistical significance was set at $P < 0.05$ for one-tailed t-test.

Results

Patient demographic data with some ocular parameters are presented in Table 1. A total of 50 subject eyes were included in the study: 25 eyes were in the diabetic and another 25 eyes were in the non-diabetic group. The mean age of the diabetic patients was 60.80 ± 10.07 year with a range from 28 to 79 years. There were 15 males and 10 females. The mean age of the control population was 51.6 ± 10.78 year with a range from 27 to 73 years. There were 12 males and 13 females.

No statistically significant difference in ECD, CV, ACD, CCT, and ACV was found between two groups ($p > 0.05$ for all parameters, Table 1). From the Pentacam parameters of corneal

Table 1. Patient characteristics and some ocular parameters for Control and Diabetic groups.

Parameters	Control group	Diabetic group	p value
n	25	25	
M:F	12:13	15:10	
Age, y.o	51.6 ± 10.78 (27 to 73)	60.80 ± 10.07 (28 to 79)	0.372
CCT, μm	532 ± 43.90 (458 to 637)	536 ± 33.69 (470 to 624)	0.261
Kmax, D	44.87 ± 2.09 (40.50 to 49.50)	45.00 ± 1.34 (42.20 to 47.70)	0.032
ECD	2454 ± 288.54 (1842.20 to 3146.80)	2486 ± 419.65 (1398.70 to 3150.60)	0.367*
CV	59.66 ± 4.83 (51.60 to 71.90)	60.39 ± 3.93 (54.90 to 69.40)	0.323
ACD	2.73 ± 0.40 (2.02 to 3.46)	2.58 ± 0.37 (1.69 to 3.22)	0.438
ACV	141.76 ± 39.43 (80.00 to 218.00)	122.84 ± 32.21 (75.00 to 202.00)	0.180

n - number; y.o. - years old; M - male; F - female; Kmax - maximal keratometry; ECD = endothelial cell density; CV - corneal volume; ACD - anterior chamber depth; ACV - anterior chamber volume; asterisk (*) – Mann-Whitney U test.

variance indices only Rmin and Kmax was found to be different between groups ($p < 0.05$, one-tailed t -test, Table 2).

Discussion

Corneal changes are diagnosed in about 70% of adult patients with diabetes (24, 25). The purpose of this study was to estimate the effect of DM on the corneal measurements. We compared the corneal parameters between patients with DM with those of healthy subjects. The effect of hyperglycemia on refraction was explained with several studies, but the exact cause of refractive change due to unstable diabetes is still under debate. The chronic DM causes the alterations in the lens what lead to the refractive changes in patients [13-18]. However, the exact impact of the cornea to these refractive changes is still unknown. Sonmez et al. evaluated the corneal topographic measurements in patients which were under intensive treatment of acute severe hyperglycemia [26]. It was concluded that knowledge of these changes in corneal topographic parameters is important, especially during the treatment period of acute hyperglycemia, as it may cause an error for refractive and cataract surgery.

Data of this preliminary study suggests that there are some differences in corneal parameters evaluated with Scheimpflug camera between diabetic and non-diabetic patients. According to these results, the eyes in the diabetic patients displayed higher keratometry readings than the eyes of the non-diabetic ones.

Many studies confirmed that diabetes causes abnormalities in morphology and functioning of corneal endothelial cells. Functional disturbances may lead to increased autofluorescence of the cornea and its increased penetrability [27,28].

Table 2. Scheimpflug camera parameters of corneal variance indices for diabetic and control groups of patients.

Parameters	Control group	Diabetic group	p value
Qvalue	-0.19 ± 0.13 (-0.45 to 0.01)	-0.26 ± 0.14 (-0.55 to 0.05)	0.784
ISV	14.28 ± 5.17 (7.00 to 26.00)	18.60 ± 8.87 (9.00 to 51.00)	0.058*
IVA	0.11 ± 0.06 (0.04 to 0.32)	0.15 ± 0.09 (0.05 to 0.46)	0.147*
IHA	2.92 ± 1.79 (0.30 to 6.00)	3.69 ± 3.11 (0.20 to 9.80)	0.741*
IHD	0.01 ± 0.004 (0.003 to 0.02)	0.01 ± 0.01 (0.002 to 0.04)	0.470*
Rmin	7.53 ± 0.35 (6.82 to 8.33)	6.52 ± 0.38 (6.08 to 8.00)	0.01
Qvalue	-0.19 ± 0.13 (-0.45 to 0.01)	-0.26 ± 0.14 (-0.55 to 0.05)	0.784
ISV	14.28 ± 5.17 (7.00 to 26.00)	18.60 ± 8.87 (9.00 to 51.00)	0.058*
IVA	0.11 ± 0.06 (0.04 to 0.32)	0.15 ± 0.09 (0.05 to 0.46)	0.147*

ISV - index of surface variance; IVA - index of vertical asymmetry; IHA - index of height asymmetry; IHD - index of height decentration; Rmin - radius of minimum; asterisk (*) - Mann-Whitney U test.

Morphological changes, recorded by contact specular microscope, may result in a high variability factor of the endothelial cell surface and decreased percentage of hexagonal cells in corneas in patients with diabetes compared to healthy patients [14]. However, our calculations didn't show any significant difference in ECD between diabetic and control groups. This is coincide with results published by Furuse et al. who could not demonstrate the significant changes in mean density of corneal endothelial cells in diabetic subjects of type 2 diabetes mellitus [25].

Although there is no overall concordance in the international literature, Lee et al. found that CCT was significantly increased ($p = 0.001$) in patients who had DM for 10 years (595.9 ± 6.4 mm) compared to healthy group (567.8 ± 6.3 mm) whereas, other studies concluded that CCT was not increased in DM type 1 or 2 [18, 29, 30].

Results of this study coincide with the those of Inoue et al, [31] who reported no significant differences in CCT between 99 subjects with DM type 2 and 97 healthy subjects. In smaller study groups, Keoleian et al. and Ziadi et al. also found no differences in CCT [29, 30].

In 81 subjects with DM type 1, Busted et al. no correlations were found between diabetes duration, blood glucose levels, use of insulin, and CCT, but an association between the level of retinopathy and CCT. [32] In DM patients with proliferative retinopathy, average CCT was 566 μ m as compared to 544 μ m and 527 μ m in subjects with diabetes without retinopathy and healthy subjects, respectively.

No diabetic retinopathy was observed in diabetic group of patients of our study.

No diabetic retinopathy was observed in diabetic group of patients of this study.

DM causes changes in corneal endothelial cell morphology similar to those induced by aging. [33,34] There is a hypothesis that DM causes premature aging of the eye what was determined by age dependence of corneal asphericity in healthy subjects [35].

Therefore in diabetic cornea the asphericity would be affected more than in healthy subjects. In our case, no significant changes were found in the asphericity of the anterior or the posterior corneal surface between groups. According to obtained results, we may consider influence of DM on the radius of the posterior corneal surface. This influence is too small to change the optical power of the diabetic cornea however, it may be clinically significant in patients with not well-compensated DM.

Conclusion

In conclusion, even if data of this study need to be confirmed in further ones with larger population, the observed results has shown a possible influence of diabetes on corneal parameters. Therefore one should exercise careful attention facing diabetic patients, in whom we need precise measurements of corneal curvature.

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Low Serum Vitamin D Levels and Post-Operative Outcomes

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Letter to the Editor

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Vitamin D (25-hydroxyvitamin D) deficiency is defined as <25 nmol/L or <10 ng/mL whereas insufficiency is 25–75 nmol/L or 10–30 ng/mL. Vitamin D deficiency is a common and ever-increasing health problem that approximately affects over one billion people worldwide probably due to the 21st century lifestyle since we are not getting as much sun exposure as we used to [1]. In the USA about 25 million adults are diagnosed with Vitamin D deficiency [2].

Vitamin D is known for numerous critical functions in human body and its main role is to sustain calcium and phosphate homeostasis, and to promote bone mineralization. However, lately many major roles of vitamin D have been recognized. Researchers found that vitamin D has significant impact on immune system function and essentially regulates pro-inflammatory pathways and cytokines which play vital role in the disease of several organ systems [3]. Recent data suggests that vitamin D deficiency or insufficiency status has been associated with pathogenesis of several diseases for example hypertension, type 2 diabetes mellitus, cancer, infections, and cardio-cerebrovascular disease [4,5]. Particularly during postoperative period, patients are vulnerable to serious infections and cardiovascular complications. Underlying mechanism shows that combination of vitamin D deficiency (common in patients undergoing surgery) and surgery appear to worsen the postoperative complications. Despite the high prevalence of hypovitaminosis D in this particular population and

substantial deteriorating of many outcomes with vitamin D deficiency, there is a paucity of data in the literature focusing on surgical population.

Recently, we published a large-scale retrospective cohort analysis of 3509 adult patients who had non-cardiac related surgery in our hospital between 2005 and 2011 [1]. The aim was to determine the relationship between serum vitamin D level and all of the causes in-hospital cardiovascular morbidity, and serious infections. Results showed that higher serum vitamin D levels were associated with decreased odds of in-hospital mortality or morbidity ($P = 0.003$) [1]. Furthermore, analysis showed that there was a linear reduction of severe in-hospital outcomes for each 5 ng/mL increase in vitamin D level over the range between 4 ng/mL and 44 ng/mL [1]. It is concluded that serum vitamin D levels were associated with a composite of in-hospital death, cardiovascular events and serious infections [1].

Historically, patients are always concerned about complications of anesthesia and surgery. Nowadays, anesthesia and surgery are safer and there is tremendous improvement in perioperative patient care. However, as healthcare the goal is to increase patient satisfaction and care, along with reducing postsurgical adverse outcomes to the minimum level. Worldwide, more than 234 million patients undergo major surgeries annually and most of them have vitamin D deficiency or insufficiency. Preoperatively, increasing the vitamin D level to the optimal concentration may decrease postoperative adverse outcomes and serious complica-

tions. Therefore, further well-designed large scale clinical trials are desired to determine the effect of vitamin D in patients undergoing surgery.

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WMA International Code of Medical Ethics

Adopted by the 3rd General Assembly of the World Medical Association, London, England, October 1949 and amended by the 22nd World Medical Assembly, Sydney, Australia, August 1968 and the 35th World Medical Assembly, Venice, Italy, October 1983 and the 57th WMA General Assembly, Pilanesberg, South Africa, October 2006.

DUTIES OF PHYSICIANS IN GENERAL

- A PHYSICIAN SHALL always exercise his/her independent professional judgment and maintain the highest standards of professional conduct.
- A PHYSICIAN SHALL respect a competent patient's right to accept or refuse treatment.
- A PHYSICIAN SHALL not allow his/her judgment to be influenced by personal profit or unfair discrimination.
- A PHYSICIAN SHALL be dedicated to providing competent medical service in full professional and moral independence, with compassion and respect for human dignity.
- A PHYSICIAN SHALL deal honestly with patients and colleagues, and report to the appropriate authorities those physicians who practice unethically or incompetently or who engage in fraud or deception.
- A PHYSICIAN SHALL not receive any financial benefits or other incentives solely for referring patients or prescribing specific products.
- A PHYSICIAN SHALL respect the rights and preferences of patients, colleagues, and other health professionals.
- A PHYSICIAN SHALL recognize his/her important role in educating the public but should use due caution in divulging discoveries or new techniques or treatment through non-professional channels.
- A PHYSICIAN SHALL certify only that which he/she has personally verified.
- A PHYSICIAN SHALL strive to use health care resources in the best way to benefit patients and their community.
- A PHYSICIAN SHALL seek appropriate care and attention if he/she suffers from mental or physical illness.
- A PHYSICIAN SHALL respect the local and national codes of ethics.

DUTIES OF PHYSICIANS TO PATIENTS

- A PHYSICIAN SHALL always bear in mind the obligation to respect human life.
- A PHYSICIAN SHALL act in the patient's best interest when providing medical care.
- A PHYSICIAN SHALL owe his/her patients complete loyalty and all the scientific resources available to him/her. Whenever an examination or treatment is beyond the physician's capacity, he/she should consult with or refer to another physician who has the necessary ability.
- A PHYSICIAN SHALL respect a patient's right to confidentiality. It is ethical to disclose confidential information when the patient consents to it or when there is a real and imminent threat of harm to the patient or to others and this threat can be only removed by a breach of confidentiality.
- A PHYSICIAN SHALL give emergency care as a humanitarian duty unless he/she is assured that others are willing and able to give such care.
- A PHYSICIAN SHALL in situations when he/she is acting for a third party, ensure that the patient has full knowledge of that situation.
- A PHYSICIAN SHALL not enter into a sexual relationship with his/her current patient or into any other abusive or exploitative relationship.

DUTIES OF PHYSICIANS TO COLLEAGUES

- A PHYSICIAN SHALL behave towards colleagues as he/she would have them behave towards him/her.
- A PHYSICIAN SHALL NOT undermine the patient-physician relationship of colleagues in order to attract patients.
- A PHYSICIAN SHALL when medically necessary, communicate with colleagues who are involved in the care of the same patient. This communication should respect patient confidentiality and be confined to necessary information.

Azerbaijan Medical Association

ABOUT

The Azerbaijan Medical Association (AzMA) is the country's leading voluntary, independent, non-governmental, professional membership medical organization for physicians, residents and medical students who represent all medical specialties in Azerbaijan.

Association was founded by Dr. Nariman Safarli and his colleagues in 1999. At the founding meeting, the physicians adopted the Statutes and Code of Ethics of the Association. The AzMA was officially registered by Ministry of Justice of Azerbaijan Republic in December 22, 1999.

Since its inception, the AzMA continues serving for a singular purpose: to advance healthcare in Azerbaijan.

- Founded in 1999, the AzMA provides a way for members of the medical profession to unite and act on matters affecting public health and the practice of medicine.
- We are the voice of physicians who support the need for organized medicine and want to be active within their profession.
- We are the representative for Azerbaijan doctors on the world-wide level and the voice of Azeri physicians throughout the world.

MISSION

The mission of the Azerbaijan Medical Association -is to unite all members of the medical profession, to serve as the premier advocate for its members and their patients, to promote the science of medicine and to advance healthcare in Azerbaijan.

GOALS

- Protect the integrity, independence, professional interests and rights of the members;
- Promote high standards in medical education and ethics;
- Promote laws and regulation that protect and enhance the physician-patient relationship;
- Improve access and delivery of quality medical care;
- Promote and advance ethical behavior by the medical profession;
- Support members in their scientific and public activities;
- Promote and coordinate the activity of member-specialty societies and sections;
- Represent members' professional interests at national and international level;
- Create relationship with other international medical associations;
- Increase health awareness of the population.

The association's vision for the future, and all its goals and objectives are intended to support the principles and ideals of the AzMA's mission.

INTERNATIONAL RELATIONSHIPS

Since its establishment, AzMA built close relationships with many international medical organizations and national medical associations of more than 80 countries. The following are the AzMA's international affiliations:

- Full membership in the World Medical Associations (WMA) (since 2002)
- Full membership in the European Forum of Medical Associations (EFMA) (since 2000)
- Full membership in the Federation of Islamic Medical Associations (FIMA) (since 2002)
- Associate membership in the European Union of Medical Specialists (UEMS) (since 2002)

Especially the year 2002 remained with memorable and historical events for AzMA such as membership to the World Medical Association (WMA). Today we are extremely pleased to represent our Association and to be a part of the WMA family.

MEMBERSHIP

A person with medical background, who accepts and follows the AzMA Statutes and AzMA Code of Ethics, may become a member of the Association. The Code of Ethics of the Association shall be the members' guide to professional conduct.

Membership in the AzMA is open to:

- Physicians residing and practicing in Azerbaijan and in abroad.
- Medical students enrolled at medical universities or schools
- Retired physicians

Members can access a special members only area of the AzMA website designed to provide the most up-to-date, and timely information about organized medicine in our country.

To the non-member, we hope you'll discover, through our website how valuable Azerbaijan Medical Association is to medicine in Azerbaijan and will join us.

MEDICINE'S VOICE IN AZERBAIJAN

As the largest physician membership organization in Azerbaijan the AzMA devotes itself to representing the interests of physicians, protecting the quality of patient care and as an indispensable association of busy professionals, speaks out with a clear and unified voice to inform the general public and be heard in the highest councils of government.

The AzMA strives to serve as the Medicine's Voice in Azerbaijan.

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For more information, please visit our website:
www.azmed.az

for notes

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**We work together for the
sake of healthy future of
Azerbaijan!**



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