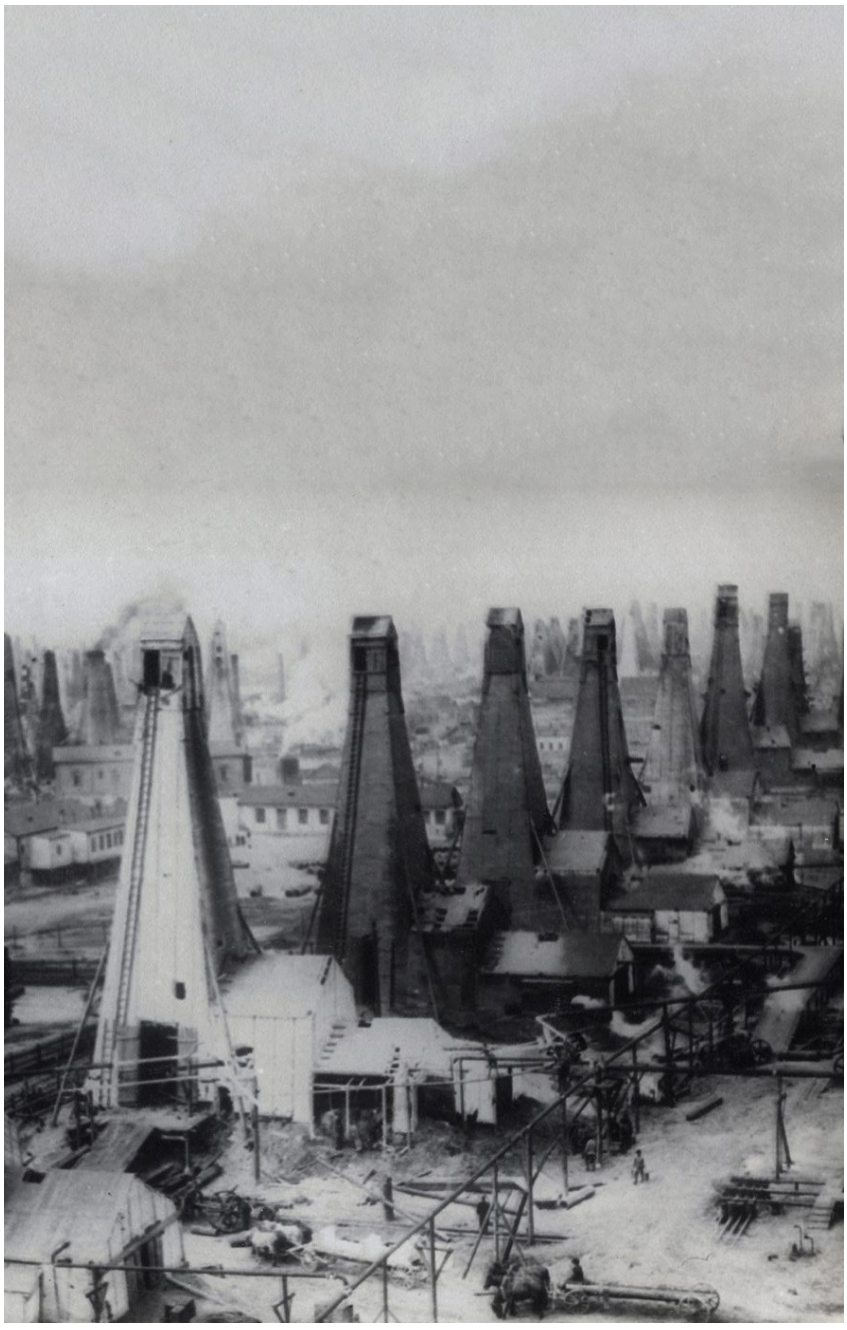
ISSN: 2413-9122
e-ISSN: 2518-7295
Volume 2 No. 1 April 2017

The Official Journal of the
Azerbaijan Medical Association

AMAJ

Azerbaijan Medical Association Journal



www.amaj.az

MD Publishing

AMAJ Azerbaijan Medical Association Journal

Official Journal of the Azerbaijan Medical Association

Volume 2 • No. 1 • April 2017

DOI

For information about DOIs and to resolve them, please visit *www.doi.org*

The Cover: The Nobel Brothers' oil wells in Baku.

In 1846, the world's first oil well was mechanically drilled in the Bibi-Heybat suburb of Baku city.

In 1878, the Nobel Brothers Petroleum Production Company (Branobel Oil Company) was established in Baku, Azerbaijan. Ludwig and Robert Nobel, Alfred Nobel's brothers, built the largest oil company the world had seen. In fact, when the Nobel Prizes were established more than a century ago (1901), roughly 12 percent of the prize money was drawn from Alfred's shares in the Nobel Brothers' Petroleum Company in Baku. This money is still being used to honor Nobel Laureates and their great contributions to modern society.

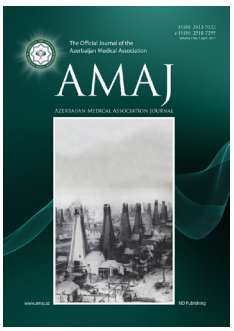


Photo courtesy Behruz Huseynzade.

AMAJ Azerbaijan Medical Association Journal

Official Journal of the Azerbaijan Medical Association

Volume 2 • No. 1 • April 2017

Call for papers

Azerbaijan Medical Association invites authors to submit their papers to Azerbaijan Medical Association Journal - **AMAJ**.

Authors preparing manuscipts for submission to **AMAJ** should consult Information for Authors available from journal site: *www.amaj.az*

Adherence to the instructions will prevent delays both in acceptance and in publication. Please, submit your publications to: *http://my.ejmanager.com/amaj/*

• • •

The **AMAJ** staff continually seeks to expand our list of highly qualified reviewers. Reviewers recieve manuscripts electronically and are asked to review them and return comments within 3 weeks. All reviews must be completed online. Guidelines for reviewers are available at *www.amaj.az*

• • •

All articles published, including editorials, letters, and book reviews, represent the opinions of authors and do not reflect the policy of Azeraijan Medical Association, the Editorial Board, or the institution with which the author is affliated, unless this is clearly specified.

• • •

For futher information please contact:
MD Publishing House, Istiqlaliyyet 37/2,
AZ1000, Baku, Azerbaijan
Tel: (+99455) 328 1888, email: editorial@amaj.az

Case Report
DOI: 10.5455/amaj.2017.01.028

Recurrent Dermatofibrosarcoma Protuberans of the Scalp Reconstructed by Visor Flap

Ilyas S. Akhund-zada, MD[1]
Rauf I. Karimov , MD, PhD[1]
Rauf Sadiqov, MD[2]
Rafael Bayramov, MD[2]

[1] Department of ENT Head and Neck Surgery, Central Hospital of Oil-workers, Baku, Azerbaijan.

[2] Republic Neurosurgery Hospital, Baku, Azerbaijan.

Correspondence:
Ilyas S. Akhund-zada, MD,
Specialist of Plastic, Reconstructive and Aesthetic Surgery; Fellow of EBOPRAS,
Department of ENT Head and Neck Surgery, Central Hospital of Oil-workers
Khatai, Yusif Safarov, 21
Baku, Azerbaijan.
email: ilyas.akhundzada@gmail.com
Phone: (+99470) 663 06 71

Dermatofibrosarcoma protuberans (DFSP) is a locally aggressive skin tumor. It usually occurs on the trunk and extremities and, only about 10% of cases DFSP are found on head and neck region. Presence of the tumor on the head challenges surgical treatment because of need in wide resections of limited scalp tissue. Recurrent cases of DFSP often possess possibility of bone and intracranial involvement that dictates even greater resection with simultaneous closure to prevent meningitis and other fatal complications. Control of the wound margins either by means of frozen sections or by Moh's technique is a keystone of successful treatment.

Here we present a case of recurrent DFSP of the fronto-parietal area. The case is remarkable for intracranial extension of the tumor. Also we describe a rare use of bipedicle a. temporalis superficialis (visor) flap for reconstruction of composite defects of forehead resulted from wide resection of large recurrent DFSP lesion.

Keywords: dermatofibrosarcoma protuberans, surgical flaps, bipedicle a.temporalis superficialis flap, scalp

Introduction

Dermatofibrosarcoma protuberans (DFSP) is a locally aggressive skin tumor of presumably mesenchymal origin. It is the most common type of skin sarcomas. The tumor usually affects people between 20-60 years; men are slightly more predisposed to it than women. Despite of locally aggressive behavior of classical dermatofibrosarcoma protuberans distant metastasis usually occurs only in 0.5% of cases and it most commonly affects the lungs. [1, 7]

Usual sites of occurrence include trunk and extremities and in only about 10% of cases DFSP occur in head and neck area. [1, 2]

Aberrations in 17th and 22nd chromosomes and previous local trauma are suggested to contribute to pathogenesis of DFSP. [3]

According to histological findings the tumor can be plaque, nodular, pigmented, fibrosarcomatous or juvenile type. Immunohistochemistry reveals CD34 antigen and absence of S-100 marker, which is necessary for differentiation from other similar tumors. [1-6]

Due to highly invasive nature DFSP can affect different underlying structures (fat tissue, fascia, muscle, bone, etc.) and cause various correspondent symptoms. However, main invasion occurs horizontally, i.e. in the adjacent skin. Because of tentacle-like centrifugal growth, nests of the tumoral cells can microscopically be found in normally appearing skin as far as 9-15 cm away from the main lesion.

Typically the tumor has irregular borders and composed of firm subcutaneous (sometimes ulcerated) nodules of fleshy or reddish color. Less common presentation of DFSP is morphea-like, atrophic patch, which is prone to further ulceration.

Treatment modalities classically consist of wide resection with 2-4 cm margins of visibly unchanged skin. Chemotherapy with imatinib and radiotherapy can be used as adjuvant measures in selected cases. [1-7, 9,

11]

Although, DFSP rarely affects the scalp the localization itself challenges surgical treatment. This is particularly true in case of recurrent lesions, which invade bone and intracranial structures. Presence of intracranial extension requires more aggressive resection, including bone, dura mater and even brain structures apart from skin.

According to review of available literature several options of reconstruction can be exploited after classical wide excision or Moh's chemosurgery, including; a) wound grafting, b) reconstruction with local scalp flap(s), c) usage of tissue expanders prior to the main surgery, d) reconstruction with free flap, e) usage of tissue expanders, after the main surgery. [2, 4, 6, 11]

In case of intracranial involvement with invasion into dura and more deep structures resection must be followed by simultaneous reconstruction combined with one of the method of soft tissue coverage. This is necessary to prevent liquor leakage as it may have fatal consequences.

Here we present a case of recurrent DFSP of the frontal region with intracranial extension, which has been treated successfully by wide excision (including bone and intracranial structures), cranioplasty and transposition of bipedicle a. temporalis superficialis (visor) flap.

Case report

A 36 years old female applied to neurosurgeon because of tumoral growth on the head, at locus of previous operation. Her disease began 7 years before a small nodule on frontal area of the scalp. It was excised at provincial healthcare unit under the local anesthesia without further pathologic examination. The tumor reoccurred next year. Totally, before current application she was operated 6 times, because of almost annual recurrence of the neoplasm. Pathological diagnosis of the tumor was established to be dermatofibrosarcoma protuberance.

In a last few months she had developed a headache, dizziness, general malaise and nausea. She was very anxious and depressed concerning course of her disease. Other findings of routine physical examination were in the normal range.

Local examination revealed scarred hairless oval-shaped area measured approximately 7x8 cm occupying center of frontoparietal area. Its borders were marked by postoperative scars, whereas the center contained crust covered nodularities and left lower quadrant was occupied by typical pinkish hard subcutaneous tumoral nodule. (Fig. 1)

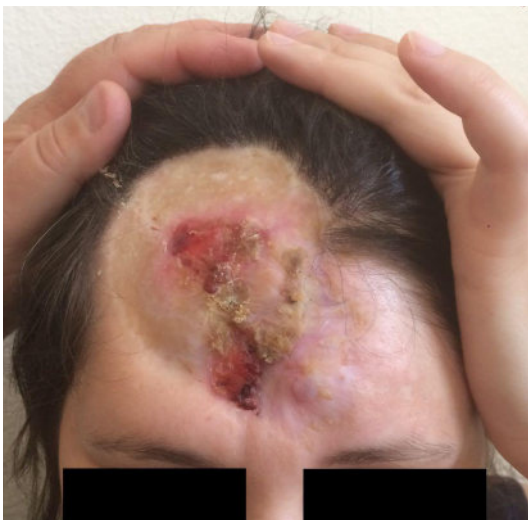
Magnetic resonance imaging of the brain revealed well-defined, 60x52 mm sized lesion, which continued from subcutaneous level to the extra-axial space through postoperative bone defect in the left half of frontal bone. The lesion homogeneously absorbed contrast agent being hypointense on T1-weighted and slightly hyperintense on T2-wighted images. Right-sided dislocation of the anterior median structures up to 8-10 mm, as well as diffuse edema of the left hemisphere was also noted. (Fig. 2)

After explanation of the details of treatment and taking written consent, the patient undergone operation. Because of nature of the post-resection defect, reconstruction initially included using of free radial forearm flap along with transposition of local scalp. However, the patient insisted on procedure with no risk of total flap loss. So, our final surgical plan included wide excision of the tumor within 3 cm of the healthy skin, frozen section biopsies from margins of the skin wound, en bloc resection of tumor bearing scalp along with underlying portions of frontal and parietal bones, resection of the intracranial part of the tumor with surrounding dura, closure of the dural defect by graft of fascia lata, closure of the bony defect by acrylic allotransplant and closure of the skin defect by bipedicle a.temporalis superficialis (visor) flap with subsequent skin grafting of the donor area (Fig. 3 a-e).

The operation proceeded according to plan described above; definitive closure began after 8 frozen biopsies taken from the edges of skin wound (2 pieces from each quadrant) had been reported to be tumor-negative.

The postoperative course was free of major complications. The patient was signed out at 10th postoperative day. As of the last control, which was 6 month after the last operation, she was satisfied with results of treatment and showed improved mood. No signs of recurrence were found. Deformities in form of "dog ears" at the edges of visor flap resolved simultaneously after 5th postoperative month.

Surprisingly, the patient is not bothered by hair on the forehead region. She prefers carrying head covering veils and refuses further reconstruction to achieve hairless skin or to use any oth-



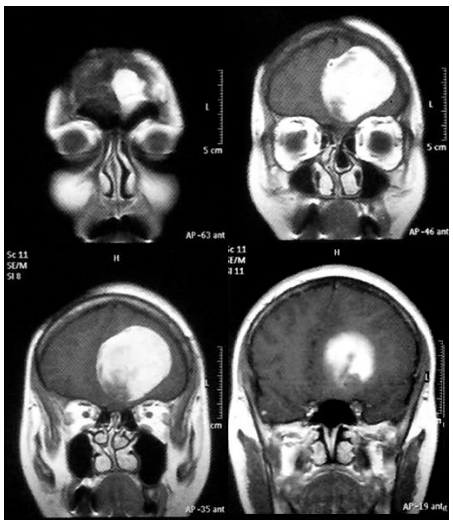


Figure 1. (left)
Pre-operative appearance of the tumor.

Figure 2. (right)
Pre-operative MR imaging (T2-weighted, coronal) shows intracranial extension of the tumor.

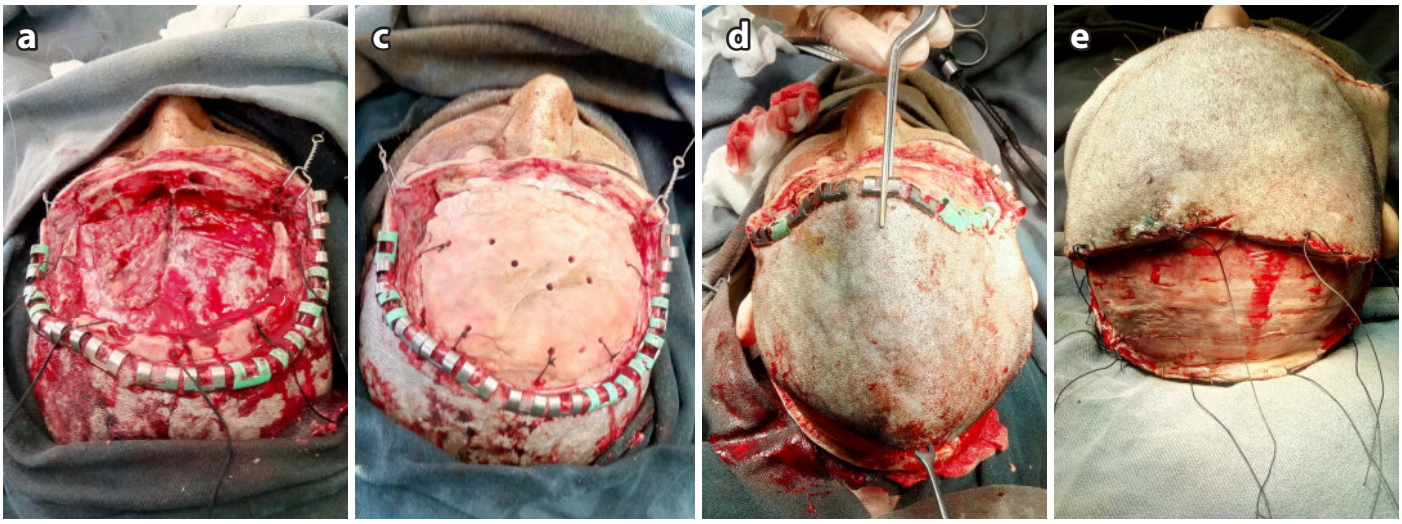


Figure 3. (a, b, c , d, e)
Intraoperative view: en bloc resection and duroplasty, cranioplasty, bipedicle temporalis (visor) flap, graft on donor area.

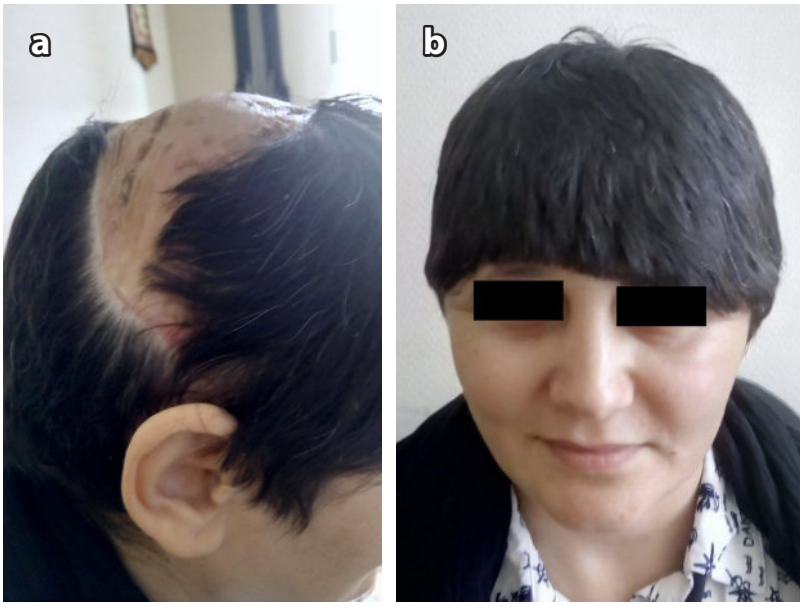


Figure 4. (a, b)
Post-operative view of the frontal area and donor site of the visor flap.

er method of hair removal. (Fig. 4 a-b)

Discussion

Dermatofibrosarcoma protuberans (DFSP) is a rare skin tumor with locally aggressive behavior. Spread of tumoral cells far from the primary lesion is commonly known histological and clinical feature of the tumor. Therefore wide excision is keystone of the successful treatment. [1, 3-5]

Although DFSP of the head and neck constitutes less than 10% of cases, treatment of such entity is challenged by some anatomic and physiologic features. First, only limited amount of hair bearing scalp is available, which leads to an aesthetic problem in case of wide excision and skin grafting. However, this issue can be addressed by preoperative scalp expansion in case of small, non-recurring and slowly growing lesions. Otherwise, postoperative scalp expansion may also be an option. Second, wide excision may exceed borders of scalp, requiring excision part of the ear, forehead or brow. In these cases additional aesthetic problems are to be considered. Third, cases where lesions invaded cranial bones and intracranial space require immediate reconstruction of multiple tissue layers to prevent meningitis and similar fatal complications. [8] The dura is classically repaired by fascil graft from fascia lata, temporal fascia etc. Alloplastic materials as well as biological glues may also be used for this purpose. [1] Bone grafts (from iliac crest or ribs), alloplastic materials (acrylic derivatives) or even autologous bone processed by autoclaving or irradiation can be employed to reconstruct defects of cranial

vault. Alternatively, the defect can be left open. Skin closure over cranial defect (either open or reconstructed) must be in the form of flap. [2, 4] Small to average wounds can be closed by transposition of the rest of the scalp with primary closure or grafting of the donor site. However, repair of large scalp defects, which exceeding 9 cm in diameter, often require free tissue transfer. Here muscle flap with overlying split thickness skin graft remains classical option, whereas fasciocutaneous flaps (like anterolateral thigh flap, transverse thoracodorsal artery flap etc.) can also be used in suitable cases.

In our case, we used classical dural repair by graft of fascia lata. Taking into a consideration large size of cranial defect (occupying large part of parietal and almost all frontal area) we decided on alloplastic bone replacement with methylmethacrilate material. Reconstruction of soft tissue coverage was done by large bipedicle a. temporalis flap (visor flap). The choice of this flap was directed by need in reconstruction that posesses low morbidity and no risk of total flap failure. Visor flap is widely known for its application in reconstruction of hear-bearing segments of upper and lower lips, and check in male patients, particularly after burn injuries. It provides ample amount of thick scalp skin and have robust circulation through both a. temporalis and depending on design can also include branches of retroauricular arteries. Here, we used the flap to cover almost entire parieto-frontal area over alloplastic material. This is, for our knowledge is a rare, "off label" use of visor scalp flap.

The drawbacks of our operation include translocation of hear-bearing skin on the forehead and creation of alopecia at the donor site of visor flap. Our patient refused further reconstruction. However, one can speculate on that further refinement can be done by laser hair removal, resurfacing of the forehead area by non-hear-bearing skin graft, reposition of the visor flap back to donor area and transfer of a free fasciocutaneous flap onto forehead area. Alopecia on the donor site can also be addressed by usage of tissue expanders.

By presenting this case we would like to raise awareness of DSFP in case of nodular lesions of scalp. Wide excision with subsequent control (either by frozen sections or by Moh`s method) is sine qua non of successful treatment, especially in case of recurrent lesions. It has to be emphasized that recurrent DFSP of scalp has potential for intracranial extension, which necessitates even larger resection. According to our experience, visor scalp flap is a valuable option for reconstruction of composite defects resulted from such resections.

References

1. Carloni R, Herlin C, Chaput B, De Runz A, Watier E, Bertheuil N. Scalp Tissue Expansion Above a Custom-Made Hydroxyapatite Cranial Implant to Correct Sequelar Alopecia on a Transposition Flap. World Neurosurgery. 2016 Nov 30;95:616-e1.
2. Maguire R, Nikolorakos D, Lam A. Dermatofibrosarcoma Protuberans: The role of tissue expansion in reconstructive surgery of the scalp. International journal of surgery case reports. 2016 Dec 31;19:137-9.
3. Liansheng L, Xialiang L, Yaodong Z, Yajun X, Meiqing L. Report of two cases of recurrent scalp dermatofibrosarcoma protuberans and literature review. Indian journal of dermatology. 2014 Nov;59(6):602.
4. Mori S, Di Monta G, Marone U, Chiofalo MG, Caracò C. Half forehead reconstruction with a single rotational scalp flap for dermatofibrosarcoma protuberans treatment. World journal of surgical oncology. 2012 May 6;10(1):78.
5. Eguzo K, Camazine B, Milner D. Giant dermatofibrosarcoma protuberans of the face and scalp: a case report. International journal of dermatology. 2014 Jun 1;53(6):767-72.
6. Badeau AM, Granick M, Deleyiannis FW. Considerations for tissue expansion in the management of massive dermatofibrosarcoma protuberans of the head and neck. Eplasty. 2013;13.
7. Gatlin JL, Hosch R, Khan M. Dermatofibrosarcoma protuberans of the scalp with fibrosarcomatous degeneration and pulmonary metastasis. Journal of clinical imaging science. 2011 Jan 1;1(1):55.
8. Abe T, Kamida T, Goda M, Inoue R, Fujiki M, Kobayashi H, Hatano Y, Shibuya H, Fujiwara S, Terashi H, Mori T. Intracranial infiltration by recurrent scalp dermatofibrosarcoma protuberans. Journal of Clinical Neuroscience. 2009 Oct 31;16(10):1358-60.
9. Mattox AK, Mehta AI, Grossi PM, Cummings TJ, Adamson DC. Response of malignant scalp dermatofibrosarcoma to presurgical targeted growth factor inhibition: Case report. Journal of neurosurgery. 2010 May;112(5):965-77.
10. Lemm D, Muegge LO, Hoeffken K, Aklan T, Mentzel T, Thorwarth M, Schultze-Mosgau S. Remission with Imatinib mesylate treatment in a patient with initially unresectable dermatofibrosarcoma protuberans—a case report. Oral and maxillofacial surgery. 2008 Dec 1;12(4):209-13.
11. Parker TL, Zitelli JA. Surgical margins for excision of dermatofibrosarcoma protuberans. Journal of the American Academy of Dermatology. 1995 Feb 1;32(2):233-6.

Case Report
DOI: 10.5455/amaj.2017.01.029

Infant Mortality Due to the Fall of Television: A Presentation of Two Cases

Semih Petekkaya, MD, PhD[1]
Zerrin Erkol, MD[1]
Osman Celbis, MD[2]
Bedirhan Sezer Oner, MD[3]
Turgay Bork, MD[2]
Bora Buken, MD[4]

[1] Abant Izzet Baysal University, Faculty of Medicine, Department of Forensic Medicine, Bolu, Turkey.

[2] Inonu University, Faculty of Medicine, Department of Forensic Medicine, Malatya, Turkey.

[3] Amasya University, Faculty of Medicine, Department of Forensic Medicine, Amasya, Turkey.

[4] Duzce University, Faculty of Medicine, Department of Forensic Medicine, Düzce, Turkey.

Correspondence:
Semih Petekkaya, MD, PhD
Abant Izzet Baysal University, Faculty of Medicine, Department of Forensic Medicine, Bolu, Turkey.
email: semihpetekkaya@gmail.com
Phone: +90 537 958 6888

Television, which emerged in parallel with the scientific developments in the last century, is the most common and effective mass media component, and became an indispensible part of the daily life of the modern societies. Because of its widespread use, television, especially as the result of falling on someone in the house, can cause injuries even death.

In this study, 8-month and 1-year old male infants who died after the fall of television and forensic autopsy were evaluated. In the first case, the television fell on the 8-month-old male infant as the result of the incident of two siblings of the subject (ages: 2 and 3) were playing with television stand, while the mother was folding the clothes next to him. In the second case, television and television stand fell on the unaccompanied subject while he was playing with them. In both cases, forensic autopsy revealed skull fractures, brain hemorrhage and lung damage.

It is important to raise the awareness of the families, and to take the simple and adequate precautions in the home environment in order to protect the children as the future of the society from the domestic accidents.

Keywords: television, accidental falls, infant mortality, safety

Introduction

Television, emerged in parallel with the scientific developments in the last century, is the most common and effective mass media component, and became an indispensable part of the daily life of the modern societies. Social communication, information exchange, education and entertainment are some of the most important purposes of the daily use of television. [1-3] There is at least one television in the 95% of houses in United States, and in Canada, there is at least one in the 95% and are at least two televisions in the 60% of the houses. [4,5] The intensive use of television in daily life can affect the family members differently. Especially, the visually moving scenes, colorful and illuminated images catch attract the kids and babies' attention and lead them to head towards the television. While the motor development of the infants shows differences between the individuals, a two-year-old infant is mature enough to turn on and off the television (3).

According to the data from 2015 from World Health Organization, one of the five most common reasons of mortality in the infants under 5 years is the injuries. [6] Injuries are considered as the one of the main causes of the deaths and disability caused by the accidents during the childhood in most of the developed countries. [7] Because of its widespread use, television can cause an injury, even death as a result of the fall on the person. The most frequent type of the injury caused by the fall of the television is the blunt head trauma. [8-10] Considering the mechanical properties and weight of the television, the risk of mortality and morbidity are higher in the accidents where it falls on the infants whose physical and motor coordination are poorly developed.

In our study, it was aimed to discuss the precautions which can be taken in order to prevent the infant mortality by taking two cases of infant death as the example.

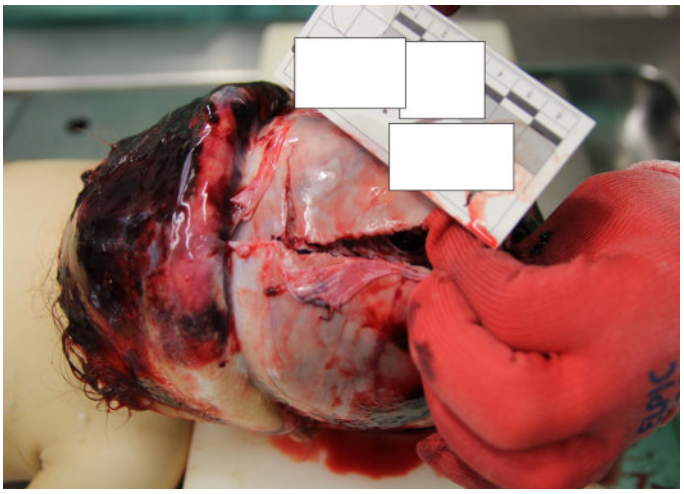


Figure 1.

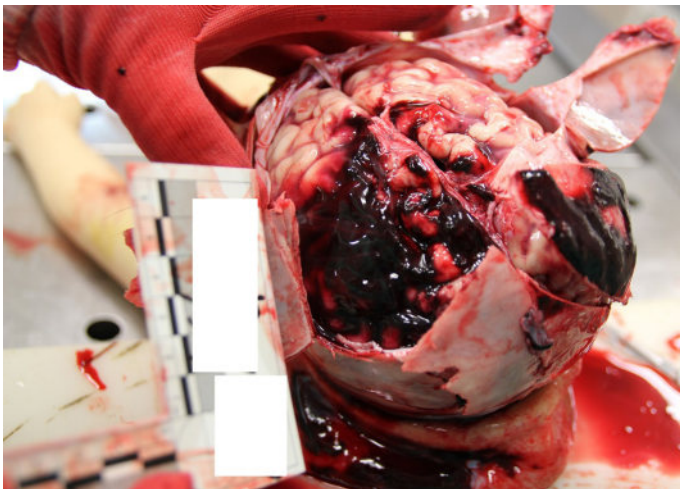


Figure 2.

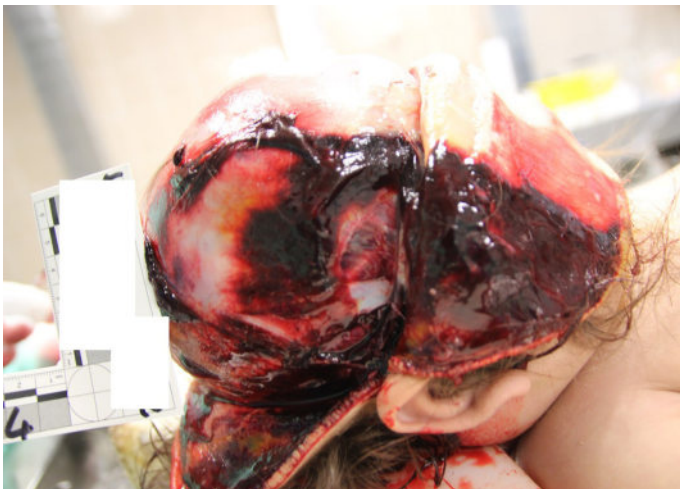


Figure 3.

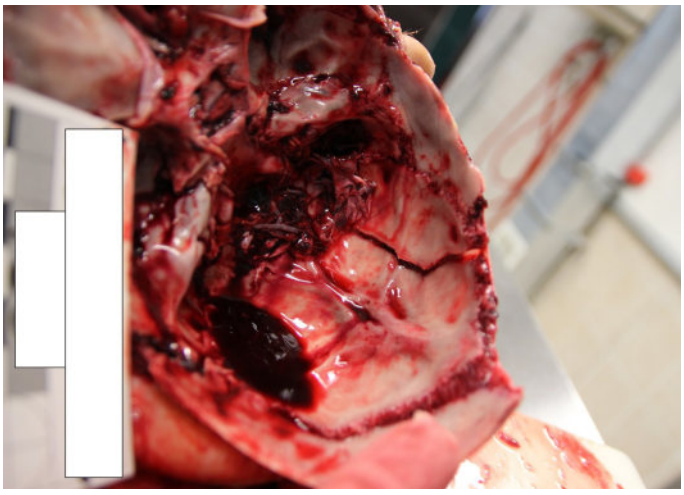


Figure 4.

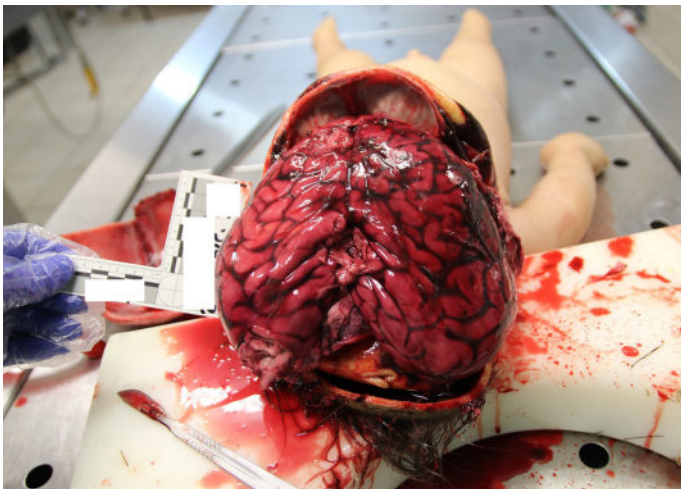


Figure 5.

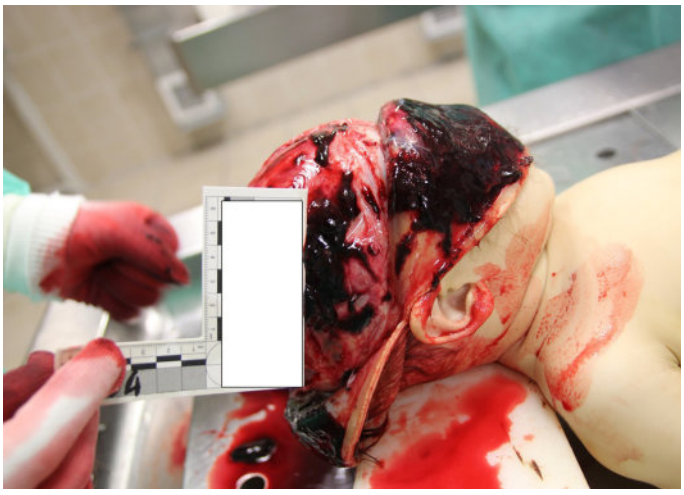


Figure 6.

Case Reports

Case 1: In the story of the incident, mother placed an 8-month-old male infant next to her on the floor, while she was folding the clothes. In the meanwhile, other two siblings (ages: 2 and 3 years old) of the infant started to play with the television stand. It was learnt that after a while, the television tilted over from the stand and hit the back of the mother, then fell on the baby, who was then immediately taken to the hospital and could not be saved despite all the interventions. It was understood that the television stand was unbalanced and could easily be shaken, and same television fell on the baby at the age of two months but was sent home without being able to detect any serious condition during the examinations at that time.

During the autopsy, the 8-month-old, 72 cm in height and 8.2 kg in weight, external examination of male infant revealed ecchymotic scratch areas on the forehead and nose, 2 cm long suture wound on the inside of the left elbow, needle marks on the inside of the right elbow, on the inside of the right and left wrists, on the back of both hands and both inguinal regions. During the internal examination, diffuse bleeding under the scalp (Fig. 1), fractures on the frontal-parietal-occipital bones (Fig. 2), epidural hematoma (4×2 and 3×1.5 cm in size and 0.3 cm thickness) on the frontal region, subarachnoid hemorrhage areas on the left parietal lobe (10×8 cm) and right parietal lobe (4×3 cm), focal subarachnoid hemorrhage on the other brain regions (Fig. 3), pneumothorax in the right chest, pink, foamy liquid in the trachea and bronchi, hemorrhage areas in the left lung, and collapsed right lung were observed. Alcohol, hypnotic-narcotic-stimulant substances and other toxic substances were not detected by the systematic toxicological analyses. The histopathological examination on internal organ samples taken during the autopsy revealed fresh subarachnoidhemorrhage in the brain, edema in the lungs, intraalveolar fresh bleeding and emphysematous changes, congestion findings in the other internal organ samples. It was concluded that the causes of death of the infant were skull fractures and cerebral hemorrhage due to the blunt head trauma and pneumothorax and lung injury due to the chest trauma.

Case 2: In the story of the incident, television and the television stand fell on an unaccompanied one-year-old male infant in the house. As the result of this, infant hit his head on the ground and television stand remained on the body. First examination in the hospital revealed an ecchymosis (2×3 cm) and hematoma on the left frontal region, an ecchymosis (3×3 cm) and hematoma on the right temporal region, an active bleeding in the right ear, reception of light reflex. Pupils were isochoric, midline and mildly myotic. An ecchymosis and edema was observed in the right eye. Fracture lines on the right fronto-parietal, the left fronto-temporal, the bilateral zygomatic, the bilateral occipital bones (more obvious on the right) and extending to the mastoid cells on the right were observed on the computer tomography. It is reported that subdural hemorrhage foci (4.5 mm) in the right fronto-temporo-parietal region, parenchymal hemorrhage foci in the right occipital and temporal regions and subarachnoid hemorrhage foci in the basal cisterns were observed. The subject was followed and treated in the pediatric intensive care unit was lost after 15 days in spite of all interventions.

During the autopsy, a 1-year-old, 80 cm in height and 13 kg in weight, external examination of the male infant revealed that edemas in both periorbital regions, ecchymosis in the right periorbital region, abrasion thought to be connected to a medical application, on the right corner of the mouth (3×0.5 cm), healing wound on the back of left foot (3×1 cm). Body looked swollen and there were needle marks on the back of the both hands, wrists, inside of the elbows, hells and toes. Internal examination revealed the hemorrhage on the right fronto-temporo-parietal region and in the inner surface of the heart. Intramuscularhemorrhage areas were observed in both temporal muscles (Fig. 4), as well as linear fracture lines on the right fronto-temporal and parietal occipital bone (Fig. 5) and epidural and subdural hemorrhage regions. Brain was spongy and in dark red colour (Fig. 6). Serous fluid was present on both rib cages, and severe edema was observed in the lungs, as well as petechiae. There were hemorrhage areas (2×1 cm) on the lower lobe under pleura of the right lung, and petechiae on the surface of the heart. Hemorrhage areas (2×0.5 cm) on the upper part of the esophagus were observed. Serous fluid was seen in the abdominal cavity. Alcohol, hypnotic-narcotic-stimulant substances and other toxic substances were not detected by the systematic toxicological analyses. The histopathological examination on internal organ samples taken during the autopsy revealed fresh subendocardial bleeding areas in the myocardium, widespread atelectasis in the lungs, fresh lobular pneumonia, indications of lung damage, presence of fibrin micro-thrombi in the vessel lumen, edema, hyperemia, fresh hemorrhage areas in the parenchyma of the brain tissue, as well as edema and severe hyperemia, and acute suppurative pancreatitis and hyperemia findings. The causes of death of the infant were concluded as skull fractures and cerebral hemorrhage due to the blunt head trauma and complications caused by lung injury due to the chest trauma.

Discussion

Rate of injury, due to the fall of television during childhood, increases all over the world because of the lack of information and the precautions about the subject. [10, 11] In a survey by the U.S. Consumer Product Safety Commission, it was reported that annual average of the individuals visited emergency service due to injuries caused by the fall of television were 15,400 between 2011 and 2013. [12] 64% of the individuals were between the ages 0 and 9. When compared the injuries caused by the television fall in the infants between the ages of 0 and seventeen, 19% of them had contusion and abrasion, 12% of them had internal organ injury, 7% of them had bone fractures and 11% of them had other diagnoses. [12] Also, most affected parts of the body were reported as head, arms and feet, and highest death ratio (74%) was seen in the childhood. [12]

Old tube televisions are heavier and have sharper angular, and their center of gravity is located in the front section. The center of gravity of the newly produced flat screen televisions is more balanced, yet, as the screen size increases, the center of gravity shifts and this makes them unbalanced. Nowadays, tube televisions are not sold in the electronic markets anymore.

However, new flat screen televisions are located more central locations at houses, while the old tube televisions are located in the rooms that are rarely controlled. Because of that, control of the parents over the child declines. [4, 10] In both cases, injuries were caused by the tube televisions located in the central rooms. In our second case, the accident happened because the infant was playing with the television unaccompanied. Because of that, parents should either check the safety of the infant more frequently or not leave them alone in the room.

In a study conducted in the University of Pittsburgh where the injuries caused by the fall of television during childhood, 52 cases of victim infant, youngest infant was 13 months old and mean age was 36 months, were detected. [7] All injuries occurred were reported to happen in the house environment and 83% of the cases happened without any eye-witnesses and the most common type of injury was blunt head trauma (83%). [7] In another study, conducted in Canada in 1997-2011, 179 cases where the patients visited the hospital because of the injuries caused by television fall were analyzed. [5] The analyses revealed that 81% of the cases were younger than 5 years old and 51% of the cases included the cases of babies who just started to walk. [5] Most of the injuries occurred at home on the weekends and most common type was the blunt head trauma. [5] Similar results were also obtained in other studies. [4, 10, and 13] Another study conducted in Elazig reported the age range of four male infant case of television fall as 15-36 months. [8] Two cases presented in our study had the ages of 8 months and 1 year. While 8 months of age is considered as young according to the literature, other two siblings (ages 2 and 3 years old) have the influence on the fall of the television and the stand on the case by playing with them. The other case, who was 1 year old, was thought to be vulnerable to the accidents due to reasons such as being at the initial stage of the walking, having undeveloped judgment and motor coordination, being unaccompanied and having curiosity for colorful and moving images on the television, having tendency to recognize the environment by biting and touching and not taking the necessary precautions against the accidents. In accordance with the literature, the primary injury leading to death is blunt head trauma in both cases.

In the first case presented, it was learnt that the television stand was unbalanced and was shaken easily even with the slightest touch. Because of the above mentioned reason, television had fallen on the subject when he was 2 months old, but luckily it did not cause any injury. However, the fact of that family did not have the television stand repaired to stabilize it or replace it with a new and firm-stable stand, and did not take the lesson from the first accident and take the necessary precautions clearly led to the second accident that caused the death.

Traumatic findings in the areas exposed to the trauma during the course of the fall of the television may also suggest the presence of physical abuse. Therefore, this kind of cases should also be considered as child abuse during the process of judicial investigation. The findings in the crime scene such as the type, location, weight of the television, as well as the properties of stand and altitude of fall, should be recorded, and physical-psychological development should be questioned.

Ota and colleagues investigated the danger of television in the house and level of knowledge of the parents about it in a study, and reported that the 85% of the parents included in the study were ignorant about the danger of fall of the television. [14] In another study, it was stated that because the novice parents know less than 50% of the present risks, and the situation is independent of the educational level unless the parents are pre-educated about how to raise a child. [15] In order to protect the children as the future of the society, it is important to raise awareness of the parents. Also, simple and enough precautions, such as using special television stands produced by the television manufacturers, stabilizing the television stands to the ground and the wall, placing the television cables out of reach of children to prevent them to pull them, not placing the items (toys, remote control, food etc.) that attracts the kids, not using the television stands with drawers, should be taken in the home environment.

References

1. Büyükbaykal G. Televizyonun Çocuklar Üzerindeki Etkileri. İletişim Fakültesi Dergisi. 2007;28:31-44.
2. Aral N, Aktaş Y. Çocukların Televizyon Ve Diğer Etkinliklere Harcadıkları Sürenin İncelenmesi. Hacettepe Üniversitesi Eğitim Fakültesi Dergisi. 1997;13: 99-105.
3. Çakır V. Bir Sosyal Etkinlik Olarak Eğlence ve Televizyon (Konya Örneği). S.Ü. Sosyal Bilimler Enstitüsü Dergisi. 2005;13:123-42.
4. Bernard PA, Johnston C, Curtis SE, King WD. Toppled Television Sets Cause Significant Pediatric Morbidity and Mortality. Pediatrics. 1998:102(3);1-4.
5. Mills J, Grushka J, Butterworth S. Television-Related Injuries In Children-The British Columbia Experience. Journal Of Pediatric Surgery. 2012;47:991-5.
6. World Health Organisation. Causes of Child Mortality. http://who.int/gho/child_health/mortality/causes/en/ (01.12.2016)
7. Rutkoski JD, Sippey M, Gaines BA. Traumatic Television Tip-Overs in the Pediatric Patient Population. Journal Of Surgical Research. 2011;166(2):199-204.
8. Türkoğlu A, Tokdemir M, Yaprak B, Börk T, et al. Üzerine Televizyon Düşmesi Sonucu Meydana Gelen Çocuk Ölümleri: Olgu Sunumu. F.Ü. Sağ. Bil. Tıp Derg. 2014;28(1): 35-7.
9. Turan Y, Uysal C, Korkmaz M, Yılmaz T, Göçmez C, Özevren H, Gören S, Ceviz A. Kaza ile üzerine cisim düşmesine bağlı hastaneye başvuran hastaların değerlendirilmesi. Dicle Tıp Dergisi. 2015;42(1): 51-4.
10. Platt MS, Stanley C. TV Tip-Over Morbidity and Mortality in Children. J Forensic Sci. 2011;56(5):1364-7.
11. Pakiş I, Karapirli M, Yaycı N. Üzerine Kaza ile Cisim Düşmesine Bağlı Çocuk Ölümleri. Acıbadem Üniversitesi Sağlık Bilimleri Dergisi. 2010;1(2):81-4.
12. United State of America Constumer Product Safety Commision. Product İnstabilityor Tip-Over İnjuries and Fatalities Associated with Televisions, Furniture, and Appliances: 2014 Report. Erişim adresi: https://www.cpsc.gov/s3fs-public/pdfs/InstabilityorTipoverReport2014Stamped.pdf. Erişim Tarihi: 15.12.2016.
13. Deisch J, Quinton R, Gruszecki AC. Craniocerebral Trauma Inflicted by Television Falls. J Forensic Sci. 2011;56(4):1049-53.
14. Ota FS, Maxson RT, Okada PJ. Childhood İnjuries Caused By Falling Televisions. Acad Emerg Med. 2006;13:700-3.
15. Gaines J. Recognition Of Home İnjury Risks By Novice Parents Of Toddlers. Accident Anal Prev. 2009;41(5):1070-4.

Case Report
DOI: 10.5455/amaj.2017.01.030

Cardiac Dysrhythmia During Superficial Parotidectomy

Lala Aliyeva, MD[1]
Qulam Rustamzade, MD, PhD[2]
Araz Aliyev, MD[3]

[1] Anaesthesia and Intensive Care Department, Central Hospital of Oil-workers, Baku, Azerbaijan.

[2] Head of Anaesthesia and Intensive Care Department, Central Hospital of Oil-workers, Baku, Azerbaijan.

[3] Department of ENT Head and Neck Surgery, Central Hospital of Oil-workers, Baku, Azerbaijan.

Correspondence:
Lala Aliyeva, MD,
Anaesthesia and Intensive Care Department, Central Hospital of Oil-workers, Khatai, Yusif Safarov, 21
Baku, Azerbaijan.
email: anestlaliyeva@gmail.com

Bradycardia and asystole due to the trigeminocardiac reflex can occur during several maxillofacial, skull base and ophthalmic procedures.

We describe a case where severe bradycardia occurred during superficial parotidectomy. The case report is presented to show a possible existence and importance of reflex bradycardic responses that may occur during surgical procedures involving the parotid gland and their management.

Keywords: trigeminocardiac reflex, anesthesia, bradycardia, parotidectomy

Introduction

The trigeminal nerve is the largest of the cranial nerves, and it provides sensory supply to the face, scalp, and mucosa of the nose and mouth. Bradycardia and asystole due to the trigeminocardiac reflex (TCR) can occur during several maxillofacial, skull base and ophthalmic procedures. [1] Stimulation of any sensory branch of the trigeminal nerve is thought to provide an important stimuli for the initiation of the trigeminorespiratory reflex, parasympathetic dysrrhythmias, sympathetic hypotension, apnea, gastric hypermotility etc. [2] In fact, the term TCR was coined by the anesthetists Shelly and Church. They also described the first occurrence of central TCR in humans during the surgery of cerebellopontine angle and brainstem. According to Schaller's experience, the TCR occurs in 10-18% of the patients undergoing maxillofacial, skull base and ophthalmic surgery. [3]

Presented case highlights the importance of reflex bradycardic responses, which may occur during parotidectomy, and the significance of perioperative management of TCR.

Case report

A 44 years old heavy smoker normotensive male, weighed 85 kg and with height 185 cm, was scheduled for left superficial parotidectomy. His medical history and physical examination were unremarkable. Results of the routine clinical investigations, including electrocardiogram (ECG), were within the normal limits. Premedication included 5mg of intravenous dormikum. His baseline heart rate (HR) was 85 beats per min and his blood pressure (BP) measured 128/80 mmHg. The anesthesia was induced with intravenous injection of propofol (160 mg) and fentanyl. [100 μg] Orotracheal intubation was facilitated with suxamethonium. The anesthesia was maintained with sevoflurane (2.5%) and oxygen, and controlled ventilation. The HR and BP remained stable at induction (75-85 beats per min and 140/80 - 110/70 mmHg, respectively). The surgeon proceeded with left superficial parotidectomy (facial nerve was preserved). For the initial 50 minutes, the patient's condition was stable, with ETCO2 (end tidal carbon dioxide) ranging between 28 and 35 mmHg, SpO2 (peripheral capillary oxygen saturation) 98-100%, BP between 120/80 and 100/60 mmHg, and sinus rhythm of 70-80 beats per min. During dissection of the superficial lobe for the mobilization of facial nerve, there was a little rise in BP up to 145/100 mmHg and in HR up to 90 beats per min. A dose of fentanyl (100 μg intravenously) was administered and concentration of sevoflurane was increased up to 4%. About 30 minutes later, sudden bradycardia occurred. The

BP and HR records were 80/46 mmHg and 42 beats per min correspondently (Fig. 1). The surgery was immediately halted, oxygenation and normocapnia were confirmed, and inhalational anesthetic agents were discontinued. A dose of atropine (1 mg intravenously) was administered and repeated after two minutes, as HR decreased further to 39 beats per min. Because HR remained unresponsive to atropine, a dose of dopamine (10 μg/kg/min intravenously) was administered. Then HR promptly increased to 94 beats per min with normal sinus rhythm, BP and SpO2 became 110/76 mmHg and 99% respectively. 5 min following administration of dopamine, BP constituted 120/81 mmHg and HR was 80-90 beats per min. In order to stop transmission of the afferent signals topical lidocaine (2%) was applied around the gland.

The entire episode lasted 10 min; throughout it systolic BP (recorded every 1 min) and SpO2 remained >90 mmHg and >99% respectively. Then the anesthetics were re-instituted and the surgery was continued. During the surgery there were several episodes of bradycardia, which reversed after withdrawal of the mechanical stimuli and a dose of dopamine.

The total duration of surgery was 3 hours. The patient made safe recovery with no untoward events in the postoperative period. His postoperative serial 12 lead ECG, serum electrolytes and other common biochemical parameters were within normal limits. The patient was discharged on postoperative day 2 to continue follow up an outpatient basis.

Discussion

The TCR has been described during many neurosurgical, ophthalmologic and maxillofacial procedures. It is defined as the sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, apnea, or gastric hyper-motility during stimulation of any sensory branch of trigeminal nerve. The proposed mechanism for the development of the TCR is that the sensory endings of trigeminal nerve send neuronal signals via the Gasserian ganglion to the sensory nucleus of the trigeminal nerve, forming the afferent pathway of the reflex arc. This afferent pathway continues along the short internuncial nerve fibers in the reticular formation to connect with the efferent pathway in the motor nucleus of the vagus nerve (Diagram 1). [4] Several experiments on animal models demonstrate that trigeminally induced cardiovascular reflexes could be mediated initially in the trigeminal nucleus caudalis and subsequently in the parabrachial nucleus, the rostral ventrolateral medulla oblongata, the dorsal medullar reticular field, and the paratrigeminal nucleus.

There has been a lot of discussion about the best and more effective management of TCR. Without any doubt, one can say that application of atropine is not the only modality of the treatment. To the authors' opinion, the first and the most important management option for the TCR is to be aware of its existence and to minimize any mechanical stimulation of the nerve that leads to its occurrence.

According to the clinical experience on this topic [5], the management of patients with TCR can be classified into the following categories which are illustrated in the flow-chart (Diagram 2):

1. Risk factor identification and modification.
2. Prophylactic treatment with either vagolytic agents or peripheral nerve blocks in case of peripheral manipulations on trigeminal nerve.
3. Careful cardiovascular monitoring during anesthesia especially in those with risk factors for TCR.
4. Treatment of the condition when it occurs:
   a. cessation of the manipulation, and;
   b. administration of vagolytic agents or adrenaline.

If a TCR is elicited, the surgeon must stop the stimulus and wait until the pulse recovers its normal rhythm. There is also important issue concerning choice of pharmacological agent. Prabhakar et al. reported a 48-year-old female who developed severe bradycardia and hypotension during craniotomy for parietal convexity meningioma; she was unresponsive to atropine and successfully managed with epinephrine. [6] This important case report underlines the fact that TCR may be refractory to atropine and other vagolytic agents should be considered instead.

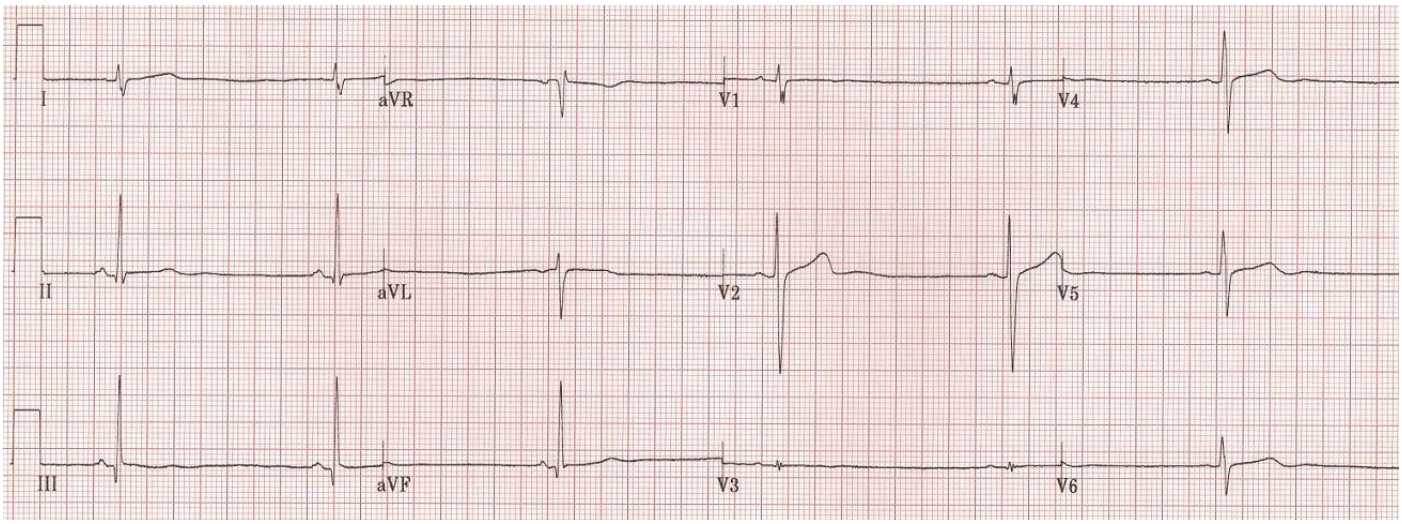
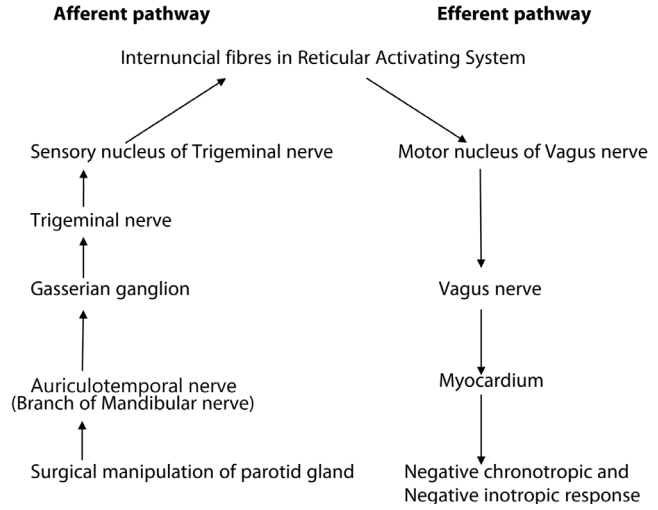


Figure 1.
Intraoperative ECG record of patient.

Diagram 1.
The trigeminocardiac reflex (TCR) [8]

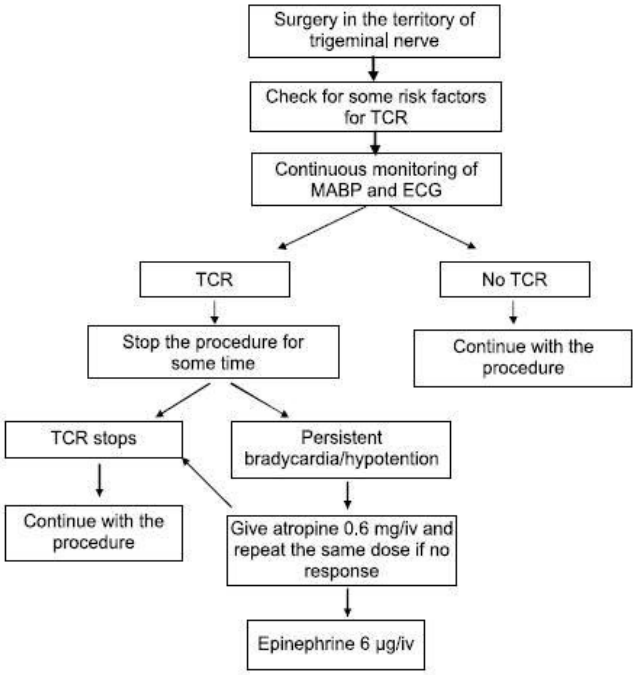


Our patient was not hypertensive and had no structural heart disease; however, his intraoperative blood pressure was labile. The patient initially developed hypertension (145/100 mmHg) and tachycardia (100 beats per min) which were considered due to pain and light anesthesia. So, this event was managed satisfactorily by increasing the concentration of sevoflurane (up to 4%), and administration of fentanyl. [100 μg] Dysrhythmias and severe bradycardia occurred suddenly in response to dissection, manipulation and traction of the superficial lobe of parotid gland. Cessation of the surgical stimuli and topical application of lidocaine did disrupt the dysrhythmia. A dose of atropine was administered but failed to control the bradycardia, which was subsequently corrected by dopamine.

The effect of dopamine can be explained by its indirect β- and α-adrenergic influences through stimulation of norepinephrine release. Dopamine is a second line drug for symptomatic bradycardia, and should be used after atropine. [7] At doses of 5 to 10 μg/kg/min, it shows positive inotrope and chronotrope effects. This dose of dopamine (a cardiac dose) acts on the sympathetic nervous system and increases cardiac output together with blood pressure.

Here we would like to underline the possibility of TCR during surgery, involving parotid gland and successful resolution of anticholinergic-resistant bradycardia with the use of appropriate dose of dopamine.

Diagram 2.
Common managment of trigeminocardiac reflex (TCR) [2]



References

1. Lübbers HT, Zweifel D, Grätz KW, Kruse A. Classification of potential risk factors for trigeminocardiac reflex in craniomaxillofacial surgery. Journal of Oral and Maxillofacial Surgery. 2010 Jun 30;68(6):1317-21.
2. Arasho B, Sandu N, Spiriev T, Prabhakar H, Schaller B. Management of the trigeminocardiac reflex: facts and own experience. Neurology India. 2009 Jul 1;57(4):375.
3. Schaller B, Probst R, Strebel S, Gratzl O. Trigeminocardiac reflex during surgery in the cerebellopontine angle. Journal of neurosurgery. 1999 Feb;90(2):215-20.
4. Precious DS, Skulsky FG. Cardiac dysrhythmias complicating maxillofacial surgery. International journal of oral and maxillofacial surgery. 1990 Oct 1;19(5):279-82.
5. Koerbel A, Gharabaghi A, Samii A, Gerganov V, Von Gösseln H, Tatagiba M, Samii M. Trigeminocardiac reflex during skull base surgery: mechanism and management. Acta neurochirurgica. 2005 Jul 1;147(7):727-33.
6. Prabhakar H, Ali Z, Rath GP. Trigemino-cardiac reflex may be refractory to conventional management in adults. Acta neurochirurgica. 2008 May 1;150(5):509-10.
7. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, Kudenchuk PJ, Ornato JP, McNally B, Silvers SM, Passman RS. Part 8: adult advanced cardiovascular life support. Circulation. 2010 Nov 2;122(18 suppl 3):S729-67.
8. Prakash S, Sahni A, Bamba C, Gogia AR. Cardiac dysrhythmia complicating total parotidectomy. The Journal of the Association of Physicians of India. 2013 Aug;61(8):569-71.

Case Report
DOI: 10.5455/amaj.2017.01.031

Huge leg hematoma due to vascular disruption following femur fracture: An industrial accident catastrophe

Hamit Serdar BASBUG, MD
Hakan GOCER, MD
Kanat OZISIK, MD

Department of Cardiovascular Surgery, Kafkas University Faculty of Medicine, Kars, TURKEY

Correspondence:
Hamit Serdar BASBUG, MD,
Department of Cardiovascular Surgery, Kafkas University Faculty of Medicine, Kars, TURKEY
email: s_basbug@hotmail.com
Phone: +90 505 261 2372

Lower extremity injuries represent one of the most common injury patterns observed in the daily practice of trauma surgery. Trauma to the long bones of the lower extremity may cause a vascular trauma involving either arterial or venous system or both. Additional vascular involvement worsens the clinical status and prognosis of the trauma patient with a potential limb loss. In this paper, an industry worker with a gross trauma causing proximal femur fracture with femoral artery and vein disruption and his surgical management was reported.

Keywords: femoral fractures, vascular system injuries, hematoma

Introduction

Limb loss after lower extremity long bone fractures has been generally ascribed to the extent of tissue damage, arterial involvement and the duration of ischemia before revascularization [1]. Associated venous injury and development of compartment syndrome are other factors affecting the prognosis [2,3]. Open femur fractures accompanied by limb-threatening vascular disruptions often present with complex management problems. Available literature about this issue is not satisfactory and affords insufficient assistance [4]. As most of the western world surgeons deal with relatively less number of these cases during their daily practice, management usually depends on the experience of others [5]. In this paper, the surgical experience of a massive industrial accident leading huge leg hematoma of the thigh and scrotum due to femoral artery disruption after femur shaft fracture was presented.

Case Report

A 29-year-old male construction worker arrived to the emergency department after an industrial accident with a huge right leg injury due to a femur shaft fracture. He was semiconscious with a blood pressure of 70/40 mmHg and heart rate of 121 bpm. He had a 3 cm skin wound of lateral thigh due to the laterally displaced fractured femur shaft perforation. According to his anamnesis, two tons of steel plate was dropped on his leg during working in the construction. Physical examination revealed a massively swollen right thigh with a huge scrotum indicating a massive bleeding and huge hematoma due to the vascular disruption (Fig. 1A). Laterally, there was a fractured bone tip exit skin lesion. Palpation of the peripheral pulses revealed absolute pulse deficit on his right leg over the popliteal artery as well as the distal posterior and anterior tibial arteries. The Mangled Extremity Severity Score (MESS) was determined as 9. Direct roentgenogram demonstrated laterally displaced proximal part of the fractured right femur shaft and a huge right leg diameter. (Fig. 1B). The Computerized Tomography (CT) Angiography of the right lower extremity demonstrated the femoral artery cut (Fig. 2A) and the presence of a massive scrotal hematoma with a contrast extravasation consistent with the vascular disruption (Fig. 2B). On the left leg, no abnormality was seen radiologically (Fig. 1B).

The patient was taken to the operating room for an emergency surgery. He was positioned in supine position under general anesthesia. Femoral sheet exploration was

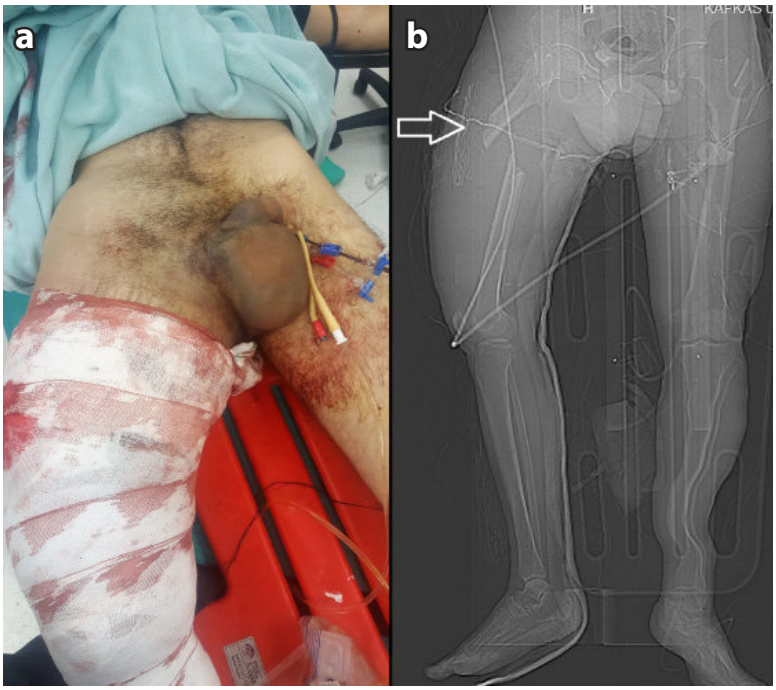


Figure 1.
Morphological view of the massive hematoma of the right thigh and scrotum. Note the thigh is extensively enlarged compared to the left side (a). Computerized Tomography (CT) topogram of the lower extremities demonstrates the presence of a huge right thigh. The arrow shows laterally displaced fractured proximal femur shaft (b).

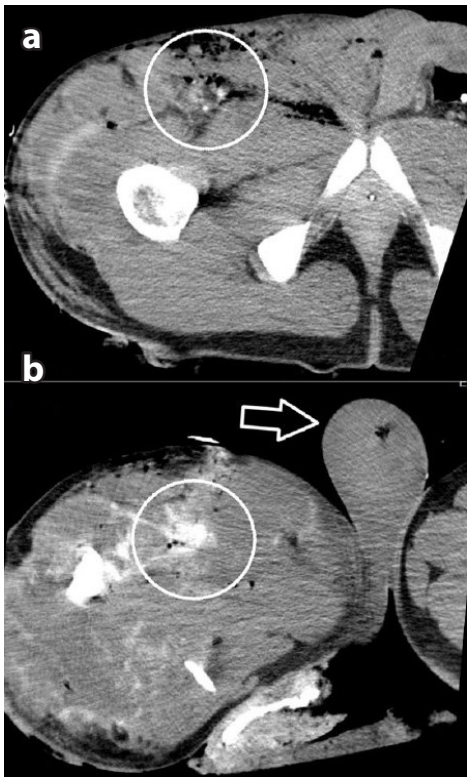


Figure 2.
CT angiography of the right lower extremity demonstrates the presence of a contrast extravasation (encircled) consistent with the vascular disruption (a) and massive hematoma disseminated through the scrotum (arrow) (b).

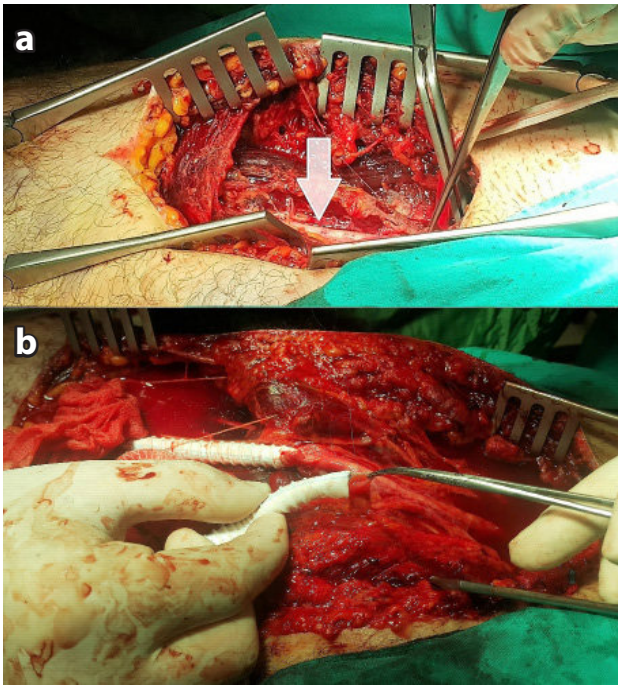


Figure 3.
The intraoperative image is demonstrating the cross-clamped intact femoral artery (arrow) at the level of inguinal ligament (a). Proximal end-to-end anastomosis of the femoral vein is being done with PTFE graft. Note that the femoral artery repair was initially done to restore limb perfusion rapidly (b).

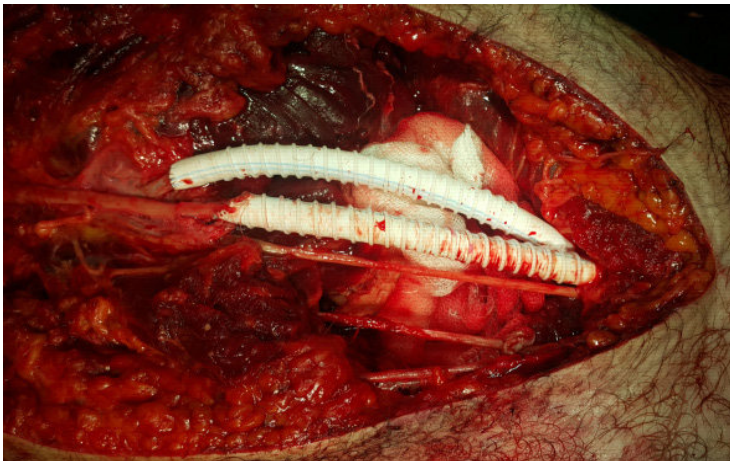


Figure 4.
The intraoperative image is demonstrating the final status of the revascularized femoral artery and femoral vein with ringed PTFE graft.

done at the level of inguinal ligament to explore the intact proximal part of the femoral artery and vein. Explored intact femoral artery was clamped (Fig. 3A). The skin incision was progressed distally to find out the disruption site of the femoral artery. Upon progression into Hunter's canal, the femoral artery and vein were found to be cut totally (Fig. 3B). Distal exploration was made to find the cut distal ends of the artery and vein. The femoral artery and vein were both repaired with 6 mm ringed polytetrafluoroethylene (PTFE) synthetic vascular graft (Fig. 4). Polypropylene 6-0 sutures with 13 mm needles were used for each anastomosis. The femur fracture stabilization and the muscular repair were made by the orthopedics. The patient was then closed with two hemovac drain tubes. The pulses were returned and the distal capillary perfusion was restored.

Discussion

Long bone fractures are considered as a heterogeneous group of traumas when associated with vascular disruptions [6]. Mangled extremities are vulnerable to infections and delayed wound healing due to the disrupted blood supply [7]. If the patient has a multisystem trauma, the prognosis gets even worse and the severe systemic problems may lead to limb amputation [8]. When the limb salvage is attempted, multiple factors affect the reconstruction process including the vascular and skeletal point of vision. These factors simply include the timing and priority of the injury [9]. There are two main factors in the determination of surgical sequence in fractures with vascular disruptions; ischemia time and fracture stability [10]. The reconstructive approach is planned mostly depending on these two main parameters. Prolonged ischemia time with absent collateral blood flow necessitates a rapid revascularization to protect the limb viability. Conversely, a grossly unstable fracture may need a fracture stabilization before the attempt for vascular repair. Temporary vascular shunts may be used to achieve a longer ischemia resistance during the skeletal rigid stabilization. Iatrogenic disruption of the temporary shunts or the permanent grafts may occur during the orthopedic procedures [7].

Although the expeditious surgical intervention is important for the successful prognosis in vascular traumas, imaging techniques should be obtained for accurate diagnosis. Arteriograms are performed either in the emergency department or in the operating room to avoid delays in sending the patient to radiology department [7]. Various methods may be used for vascular repair. Primary repair with end-to-end anastomosis is performed if there is no tissue loss. If there is gross vascular tissue loss that prevents the two ends to come together, graft interposition may be applied. These grafts include the synthetic grafts and reversed vein grafts [3].

In long bone fractures that accompany vascular injuries should initially be evaluated [7]. Despite there is no consensus about the optimum surgical sequence, the urgency and priority should be directed to the restoration of vascular integrity and reperfusion after providing the hemostasis [11]. The prevention of prolonged tissue ischemia should be the primary objective. Thus the treatment goal should be determined on the basis of the tenet that "a viable limb before a functional limb".

References

1. Andrikopoulos V, Antoniou I, Panoussis P. Arterial injuries associated with lower-extremity fractures. Cardiovasc Surg 1995;3(1):15-8.
2. Fields C, Senkowsky J, Hollier L, Kvamme P, Saroyan R, Rice J, et al. Fasciotomy in vascular trauma: is it too much, too often? Am Surg 1994;60(6):409-11.
3. Başbuğ HS, Özışık K. Tibioperoneal artery and vein reconstruction with small saphenous vein graft after gunshot injury: Original image. Turkiye Klinikleri J Cardiovasc Sci 2016;28(2):82-4
4. Lange RH, Bach AW, Hansen Jr ST, Johansen KH. Open tibial fractures with associated vascular injuries: prognosis for limb salvage. J Trauma Acute Care Surg 1985;25(3):203-8.
5. Hafez HM, Woolgar J, Robbs JV. Lower extremity arterial injury: results of 550 cases and review of risk factors associated with limb loss. J Vasc Surg 2001;33(6):1212-9.
6. Drost TF, Rosemurgy AS, Proctor D, Kearney RE. Outcome of treatment of combined orthopedic and arterial trauma to the lower extremity. J Trauma 1989;29:1331-4.
7. McHenry TP, Holcomb JB, Aoki N, Lindsey RW. Fractures with major vascular injuries from gunshot wounds: implications of surgical sequence. J Trauma 2002;53(4):717-21.
8. Bandyk DF. Vascular injury associated with extremity trauma. Clin Orthop 1995;318:117-24.
9. Lin CH, Wei FC, Levin LS, Su JI, Yeh WL. The function outcome of lower-extremity fractures with vascular injury. J Trauma 1997;43:480-5.
10. Moniz MP, Ombrellaro MP, Stevens SL, Freeman MB, Diamond DL, Goldman MH. Concomitant orthopedic and vascular injuries as predictors for limb loss in blunt lower extremity trauma. Am Surg 1997;63:24-8.
11. Kauvar DS, Sarfati MR, Kraiss LW. National trauma databank analysis of mortality and limb loss in isolated lower extremity vascular trauma. J Vasc Surg 2011;53(6):1598-603.

Case Report
DOI: 10.5455/amaj.2017.01.032

Endovascular Treatment of Cerebral Aneurysm: Report of five cases and review of the literature

Aghakishi Yahyayev, MD
Ahmet Memish, MD, PhD

Republican Diagnostic Center,
Radiology Department.
Baku, Azerbaijan

Correspondence:
Aghakishi Yahyayev, MD.
Republican Diagnostic Center,
Radiology Department.
Tibilisi avenue 147,
Baku, Azerbaijan.
email: aghakishi@yahoo.com

In spite of latest modern medical development, subarachnoid hemorrhage due to aneurysm rupture remains devastating neurological problem with highest mortality rate. Unfortunately, classic surgical treatment of aneurysm carries out its own damage due to additional surgical access trauma especially in posterior circulation. For several years open surgery with clipping aneurysm`s neck was gold standard for the treatment of aneurysm. Over the two decades endovascular approach has taken over as an alternative treatment option. In this article we represent five cases with ruptured cerebral aneurysm and treated with endovascular coiling.

Keywords: intracranial aneurysm, subarachnoid hemorrhage, endovascular coiling

Introduction

Intracranial aneurysms are relatively common disease. Because of improvement of imaging techniques, asymptomatic aneurysms are being detected more frequently even in small size. [1] Despite of current medical, endovascular and surgical management subarachnoid hemorrhage, due to cerebral aneurysm rupture continues to have high rates of morbidity and mortality for patients. Unfortunately, most of the patients being diagnosed with aneurysms have complications and rupture is one of the catastrophic complications of the cerebral aneurysm. Endovascular therapy with few access complications has increasingly become an alternative option for treatment of ruptured aneurysms. [2] Improving device industries give us precious opportunity to treat aneurysm and avoid additional procedural trauma especially in injured brain.

Case reports

From November 2016 to January 2017 five patients were admitted to our hospital with headache lasting at least two or three days.

First female patient had mild headache with nausea, without episode of vomiting and with mild nuchal rigidity according to Hunt Hass classification grade-I. CT scan without contrast material injection demonstrates grade-I subarachnoid hemorrhage according to Fisher classification.

Two of the patients (both male) had mild to moderate headache with nausea, episode of vomiting and with full nuchal rigidity. Both of them were alert and oriented, with no neurological deficit according to Hunt Hass classification grade-II. CT scan without contrast material injection demonstrates grade-II subarachnoid hemorrhage according to Fisher classification.

Fourth patient's symptoms were a little bit awkward because the main symptom was severe nausea and multiple episodes of vomiting. Only mild headache with no nuchal rigidity was persisted. So the patient was misdiagnosed as food poisoning and came to our hospital only after five days. CT scan without contrast material injection demonstrates grade-II subarachnoid hemorrhage according to Fisher classification.

Last patient had severe headache with nausea and episode of vomiting. Full nuchal rigidity existed. She was confused and lethargic. Left side hemiparesis existed. Severity of patient was grade-III according to Hunt Hass classification. CT scan without

contrast material injection demonstrates grade-III subarachnoid hemorrhage according Fisher classification.

All patients underwent CT angiography and results were next: - In first case left middle cerebral artery, bifurcation saccular aneurysm. Two patients had anterior communicant artery aneurysm. Forth patient had internal carotid artery saccular aneurysm located in cavernous segment. And last patient had basilar tip aneurysm. All aneurysms were saccular in shape and with narrow neck. After neurosurgeon consultation all patient were thought as a good candidate for endovascular coiling. General anesthesia with endotracheal intubation and femoral approach was performed for all patients. Five or six F guiding catheters were used as support catheter. Pre-embolization angiograms were taken to figure out aneurysmal neck and relationship with nearby arterial branches. After micro-catheterization of the aneurysms 3D coiling was used for complete embolization. (Fig. 1) Control angiogram demonstrates no thrombosis in parent arteries and aneurysm occlusion. In one patient we had thromboembolic complication, embolic occlusion of right middle cerebral arteries superior branch, which is easily removed by intracranial stent retrieval device (Fig. 2). After the procedure, all patients were taken to intensive care unit to continue medical therapy for vascular vasospasm. Tree "H" therapy was used (Hypertension, Hemo-delusion, hypervolemia) as standard protocol. All patients were discharged after five to ten days without neurologic problems.

Discussion

Cerebral aneurysm and subarachnoid hemorrhage is one of the most dangerous entities among cerebral hemorrhage. It has the highest mortality rate about 65 %, where most death occurs in early clinical course. According to research about 10% of patients with aneurysmal subarachnoid hemorrhage (SAH) die before reaching medical attention, 25% die within 24 hours, and 40-49% die within 3 months.

The Fisher grading system is a radiologic classification based on amount of blood seen on non-contrasted CT scan within first five days of SAH. It has been used in clinical practice since 1980 with some changes and modifications. Based on this grading system we can predict the risk of cerebral vasospasm and general outcome of patients [3].

Although radiologic classification plays an important role in clinical practice but clinical classification remains as a leading tool to predict prognosis and outcome in patients with SAH. The Hunt and Hess scale is widely used for categorizing severity of SAH, was developed in 1968 as a clinical grading system based on symptoms of patient. [4] A higher grade predicts a poor outcome and lower likelihood of survival.

There are two type of treatment for patient with cerebral aneurysm and subarachnoid hemorrhage – endovascular coiling with or without balloon or stent assistance and open surgery with aneurysmal neck clipping.

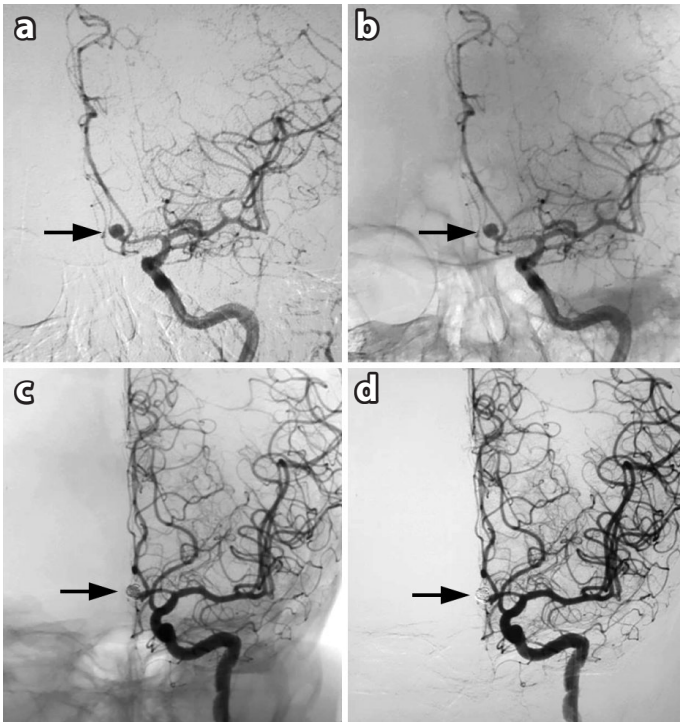


Figure 1.
Female patient with anterior communican artery aneurysm (arrow). Before (a, b) and after (c, d) embolization

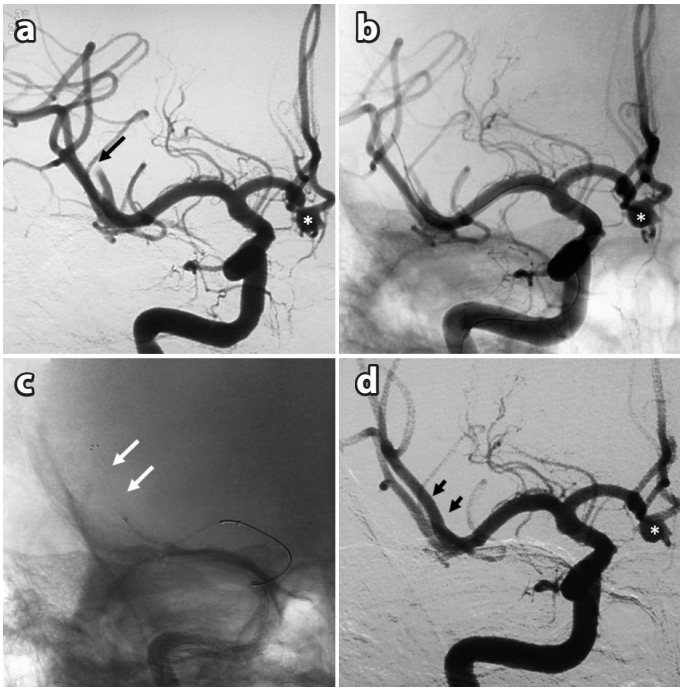


Figure 2.
a, b - Another female patient with anterior communicant artery aneurysm (asterisk). Small amount of thrombus in superior branch of right middle cerebral artery (long black arrow). **c -** Stent retrieval device inserted at same vessel (white arrows). **d -** Opened superior branch after thrombus extraction (small black arrows).

Open surgery has been broadly used during the last six-seven decades and it focuses on isolation of the vulnerable thin wall aneurysm from arterial circulation by clipping neck of the aneurysm.

Endovascular coiling is less invasive method which has been available for 20 years and getting better technical success with improving device industry. It focuses on filing of thin-walled aneurysmal cavity and excludes it from arterial flow while maintaining the normal patency of the parent artery and adjacent branches. Every year new coils come up with advanced stent and balloon development and their access devices. Because endovascular coiling has been increasingly used and taken over open surgery it became new topic for debate among physicians: - Surgical clipping versus endovascular coiling. [5,6] Not taking into account small researches there are several prospective, randomize, multicenter trails showing us their results. It has demonstrated that patient with intracranial aneurysm and subarachnoid hemorrhage, where both treatments are suitable, the outcome in terms of survival free of disability at 1 year is significantly better with endovascular coiling. [7,8].

Conclusion

Endovascular coil treatment of ruptured cerebral aneurysms are associated with significantly fewer complications and better outcomes than surgical clipping.

References

1. McCormack RF, Hutson A. Can Computed Tomography Angiography of the Brain Replace Lumbar Puncture in the Evaluation of Acute-onset Headache After a Negative Noncontrast Cranial Computed Tomography Scan?. Academic Emergency Medicine. 2010 Apr 1;17(4):444-51.
2. Marden FA, Roy SS. Endovascular management of intracerebral and subarachnoid hemorrhage. Current treatment options in cardiovascular medicine. 2005 Jun 1;7(3):197-209.
3. Jiménez-Roldán L, Alén JF, Gómez PA, Lobato RD, Ramos A, Munarriz PM, Lagares A. Volumetric analysis of subarachnoid hemorrhage: assessment of the reliability of two computerized methods and their comparison with other radiographic scales: clinical article. Journal of neurosurgery. 2013 Jan;118(1):84-93.
4. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. Journal of neurosurgery. 1968 Jan;28(1):14-20.
5. Raymond J, Roy D. Safety and efficacy of endovascular treatment of acutely ruptured aneurysms. Neurosurgery. 1997 Dec 1;41(6):1235-46.
6. Byrne JV, Sohn MJ, Molyneux AJ. Five-year experience in using coil embolization for ruptured intracranial aneurysms: outcomes and incidence of late rebleeding. Journal of neurosurgery. 1999 Apr;90(4):656-63.
7. Molyneux A, International Subarachnoid Aneurysm Trial (ISAT) Collaborative Group. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. The Lancet. 2002 Oct 26;360(9342):1267-74.
8. Brinjikji W, Rabinstein AA, Nasr DM, Lanzino G, Kallmes DF, Cloft HJ. Better outcomes with treatment by coiling relative to clipping of unruptured intracranial aneurysms in the United States, 2001–2008. American Journal of Neuroradiology. 2011 Jun 1;32(6):1071-5.

Case Report
DOI: 10.5455/amaj.2016.02.014

A Case of Thrombocytopenia Associated with Valproic Acid Treatment in a Patient with Generalized Myoclonic Seizures

Rima Ibadova, MD

Neurology Department,
Medistyle Hospital,
Baku, Azerbaijan

Correspondence:
Rima Ibadova, MD
Neurology Department,
Medistyle Hospital, H. Aliyev 116,
Baku, Azerbaijan
email: drrimaibadova@gmail.com
Phone: +994506467764

Valproic acid also known as valproate (VPA), divalproex sodium or sodium valproate, is a medication used for treatment of epilepsy and bipolar disorders and to prevent migraine headaches. It is useful for the treatment of seizures in those with generalized seizures, absence seizures, and partial seizures. Valproic acid is commonly used and first line antiepileptic drug, especially in generalized form of seizures.

Commonly reported side effects of this drug include drowsiness, weakness, dizziness, diarrhea, nausea, vomiting, abdominal pain, infection, flu-like symptoms, congenital anomalies, tremor, alopecia, thrombocytopenia, and anorexia. Other different side effects may be nystagmus, tinnitus, pharyngitis, dyspnea, ataxia, amnesia, constipation, depression, weight gain, peripheral edema bronchitis, abnormality in thinking, and fever. In our case we will discuss the patient with severe thrombocytopenia after a month of Valproic acid treatment.

Keywords: Valproic acid, thrombocytopenia, seizures.

Introduction

Valproic acid also known as valproate (VPA), divalproex sodium or sodium valproate, is a medication used for treatment of epilepsy and bipolar disorders and to prevent migraine headaches. It is useful for the treatment of seizures in those with generalized seizures, absence seizures, and partial seizures. Valproic acid is commonly used and first line antiepileptic drug, especially in generalized form of seizures.

Commonly reported side effects of this drug include drowsiness, weakness, dizziness, diarrhea, nausea, vomiting, abdominal pain, infection, flu-like symptoms, congenital anomalies, tremor, alopecia, thrombocytopenia, and anorexia. Other different side effects may be nystagmus, tinnitus, pharyngits, dyspnea, ataxia, amnesia, constipation, depression, weight gain, peripheral edema bronchitis, abnormality in thinking, and fever.

One of the most prominent side effects of Valproic acid is thrombocytopenia. In thrombocytopenia, the blood has a lower than the normal platelet count. Normal platelet count may be among 150,000 and 450,000/ ml of blood. [1] It is problem when a platelet count is less than 150.000/ml of blood. But the risk of serious bleeding occurs when the level is below 20,000/ml in blood. [1] Thrombocytopenia can occur in conditions such as certain cancers that affect stem cells, exposure to toxic chemicals, viruses and excessive consumption of alcohol, aplastic anemia etc., when the bone marrow fails to produce platelets [1, 2] Thrombocytopenia can also occur in different conditions when the body destroys its own platelets after the bone marrow makes enough platelets (e.g., in conditions such as idiopathic thrombocytopenic purpura, drug-induced thrombocytopenia and thrombotic thrombocytopenic purpura).Drug-induced thrombocytopenia can be caused by certain drugs such as Valproic acid, vancomycin, phenitoin, quinine, rifampin, and sulfa-containing antibiotics [1, 2, 3].

Case Presentation

Our patient, 12 years old girl presented to our neurological department with the complaints of an intensive and frequent myoclonic jerks without impairment of consciousness about 20-30 attacks per day, especially in the mornings. Myoclonic jerks appeared for the first time at the age of 9 years and started to occur frequently nearly every day in the morning hours since that time and especially symmetrically involved the arms without disturbance of consciousness. Generalised tonic-clonic seizures did not occur. Neurological examination of the patient was normal, only mild tremor was noticed at upper extremities. The girl also had severe psychiatric disturbances. The patient was confused and agitated. She suffered from auditory hallucinations and sleep difficulties. MRI of the brain was normal. Awake EEG recording showed bilateral synchronous fronto-central polyspike-wave discharges with photosensitivity which is similar to EEG features in Juvenile Myoclonic Epilepsy (Fig. 1).

Based on the clinical and EEG findings, the diagnosis was of generalized myoclonic epilepsy. She was started on Valproic acid 20mg/kg per day. After a month of prescription of the drug the patient was absolutely seizure free. We repeated EEG examination and there were no any abnormalities. Blood tests (CBC, ALT, GGT, Valproic acid blood level) were within normal limits, only platelet level was low (77,000/ml). There were no other risk factors for thrombocytopenia or any previous history. The drug was immediately stopped and another CBC was repeated after a week. Her platelet count increased to 274,000 /ml. Valproic acid was changed to levetiracetam for treatment of myoclonic jerks.

Discussion

The common hematopoietic system side effects of this highly used antiepileptic drug include abnormal bleeding time, thrombocytopenia, and partial thromboplastin time with decreased fibrinogen levels and prolonged prothrombin time leading to petechiae, hematoma, bruising and epistaxis. The drug can induce pruritic macular rashes. [3] Reported that, there are may be two possible mechanisms inducing thrombocytopenia. First on is the VPA has a direct toxic effect on bone marrow. Second mechanism is that VPA can stimulate the formation of autoantibody against platelets. It is suggested that contents of erythrocytes and thrombocytes lowered nearly 30 % and 10 %, accordingly in patients on the drug monotherapy. [4]

Our case, similar to some other cases [5], shows that treatment with valproic acid (monotherapy) can be associated with thrombocytopenia. This case shows thrombocytopenia induced by treatment with Valproic acid monotherapy in a young girl after a first the month of treatment. The risk of thrombocytopenia after using of Valproic acid is 5 %, and the risk increases with the level of Valproic acid in the blood and with the age of the patient. [6, 7]

Conclusion

The common blood test (CBC) should be checked periodically in all patients who are treated with Valproic acid. The risks caused by thrombocytopenia can be easily prevented by immediate stopping the drug and checking the patient's CBC every week until platelet count normalized.

References

1. Michelson A. Platelets. Amsterdam: Academic Press/Elsevier; 2007.
2. Murray M. Critical care medicine. Philadelphia: Lippincott, Williams & Wilkins; 2002.
3. Wheless J, Willmore J, Brumback R. Advanced therapy in epilepsy. Shelton, CT: People's Medical Pub. House; 2009.
4. Kaipainen P, Westermarck T, Atroshi F, Kaski M, Iivanainen M. Clinical and Hematological Profiles During Valproate Treatment of Epileptic Patients with Intellectual Disability—Case Study and Mini Review. InPharmacology and Nutritional Intervention in the Treatment of Disease 2014. InTech., DOI: 10.5772/57369
5. Nerumalla CS, Shah AA. A case of thrombocytopenia associated with valproic Acid treatment. The primary care companion for CNS disorders. 2012 Dec;15(4).
6. Acharya S, Bussel JB. Hematologic toxicity of sodium valproate. Journal of pediatric hematology/oncology. 2000 Jan 1;22(1):62-5.
7. Conley EL, Coley KC, Pollock BG, DaPos SV, Maxwell R, Branch RA. Prevalence and risk of thrombocytopenia with valproic acid: experience at a psychiatric teaching hospital. Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy. 2001 Nov 1;21(11):1325-30.

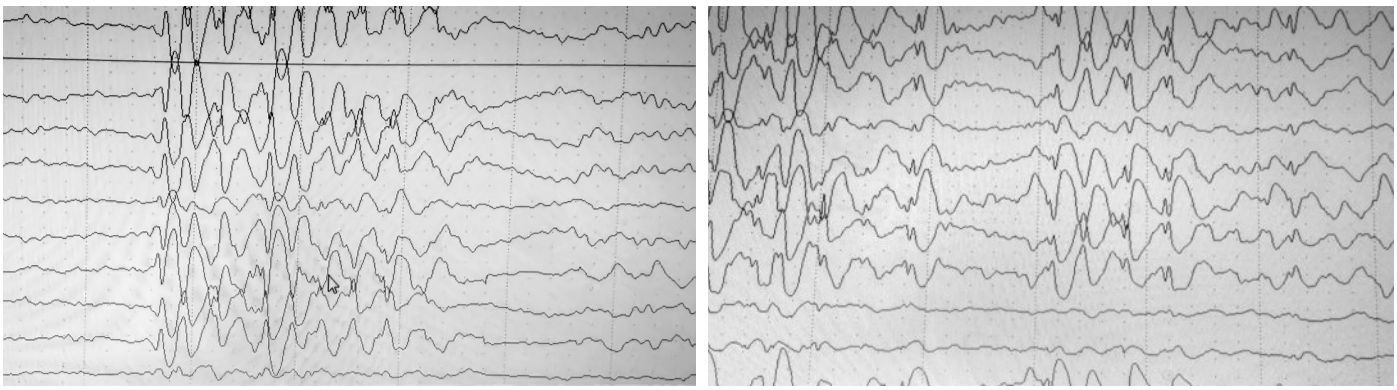


Figure 1.
Bilateral synchronous fronto-central polyspike-wave discharges with photosensitivity in awake EEG which is similar to EEG features in Juvenile Myoclonic Epilepsy.

Case Report
DOI: 10.5455/amaj.2017.01.033

Giant Sialolith: Two cases of successful surgery

Bora Ozden, DDS, PhD[1]
Vugar Gurbanov, DDS[2]
Ezgi Yüceer, DDS[2]
Dilara Kazan, DDS[2]
Levent Acar, DDS[2]

[1] Associate professor at Department of Oral and Maxillofacial Surgery, Ondokuz Mayis University, Samsun, Turkey.

[2] Research assistant at Department of Oral and Maxillofacial Surgery, Ondokuz Mayis University, Samsun, Turkey.

Correspondence:
Vugar Gurbanov, DDS,
Research assistant at Department of Oral and Maxillofacial Surgery, Ondokuz Mayis University, Samsun, Turkey.
email: surgeryvugar@gmail.com
Phone: +90 506 154 31 65

Sialolithiasis or salivary gland duct calculi are the most common pathologies of the salivary glands. They are the most general cause of acute and chronic infections of the major salivary glands. Salivary stones larger than 15 mm are classified as giant sialoliths. Two case reports describe giant sialoliths of submandibular salivary gland ducts. Also, a new method is used in these cases. It can prevent obstruction of the salivary gland ducts after the surgical removal of the stones.

Keywords: salivary glands, salivary duct calculi, Wharton's duct

Introduction

Sialolithiasis is one of the most common diseases of the salivary glands. Its incidence is aproximately 1.2% in adult population and men are affected more than women (2:1). [1, 2] Submandibular glands are effected 80% of the cases. [2, 3]

Sialoliths are always found in the distal portion of the duct or at the hilum of the gland and more rare in its parenchyma. [1]

Commonly, sialoliths' sizes are between 1 mm and 1 cm. They are rarely seen in a size more than 1.5 cm. Stones larger than 15 mm in any dimension or heavier than 1 gram have been classified as 'giant stones' or 'megaliths'. [4, 5] It could be hypothesized that it takes years to obtain a stone classified as a giant sialolith. The aim of this paper is to present two cases of an unusually sized sialoliths and their treatment methods.

Case Report

First patient was a 51-year-old woman who was referred to our clinic with complaint of a swelling on the left side of the floor of the mouth for more than 2 years. The patient's medical history, drug history, and general physical examination were all non-significant. During the intraoral examination, a large firm swelling was noted with no color changes of the surrounding mucosa. There was no history of pain or aggravated swelling during meals.

Second patient was also a 51-year-old woman with history of epilepsy and chronic depression. During routine intraoral examination, diffuse swelling with normal overlying skin was detected on the left side of the floor of the mouth. The patient was unaware of the swelling. The solid mass was freely movable and there were no signs of pain, discomfort,ulceration, fistula, or infection. During the palpation of the left submandibular gland, the absence of salivary flow from the left Wharton's duct orifice was observed.

For both of the patients, no submandibular swelling was detected during extraoral examination. Occlusal and panoramic radiographies (OPG) and cone beam computed tomography (CBCT) scans were used for diagnosis. Radiographic examination showed a large radiopaque mass, round in shape and approximately 10.6×16.4×15 mm in size for the first case and 15×15.1×9.2 mm in size for the second case, in the left submandibular region (Fig. 1). CBCT scans confirmed similar findings (Fig. 2).

Sialolithectomy was performed with an intraoral approach using local anesthesia. Upward and medial pressure were applied to the submandibular area, and an intraoral incision was made directly over the sialolith to expose it (Fig. 3). A hemostat was used to expose the superior aspect of the stone. After mobilizing the sialolith sufficiently, the

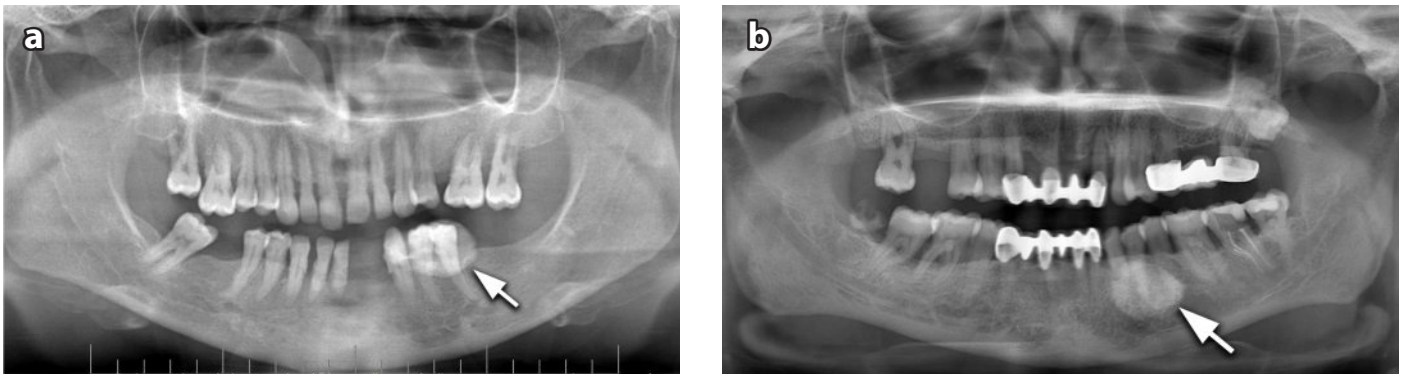


Figure 1.
In panoramic radiographies (OPG) of the first (a) and second (b) case there are shown radiopaque giant sialoliths (arrows).

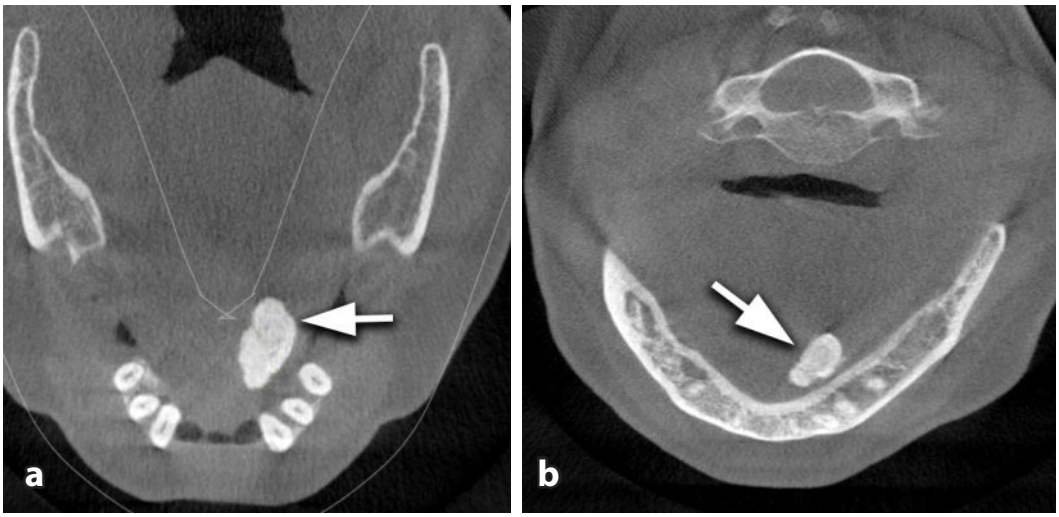


Figure 2.
Cone beam computed tomography (CBCT) scans of the first (a) and second (b) case. Sialoliths are shown by arrows.

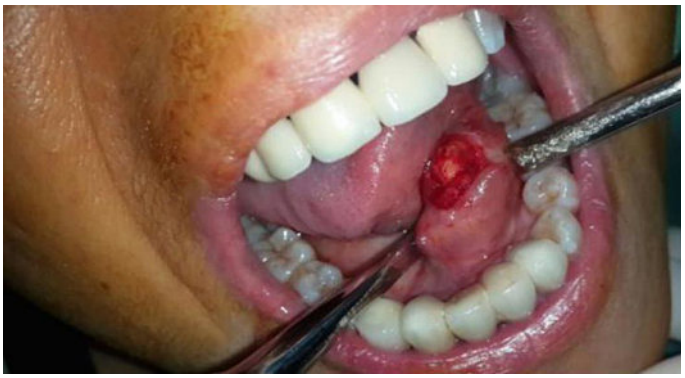


Figure 3.
Intraoral incision over the sialolith.

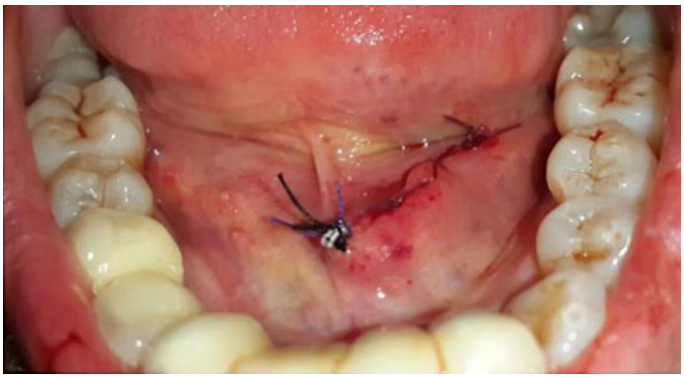


Figure 4.
The suture after removal of the sialolith and catheter placement.

stone was removed with finger pressure. The sialolith was taken out and a catheter was placed that is used for providing vascular access ordinarily, into the Wharton's duct to prevent duct obstruction (Fig. 4). The catheter was fixed with suture for 3 days. At the 1 year follow-up postoperatively, there was no swelling of the submandibular gland and salivary flow was uneventful.

Discussion

Sialoliths can arise in any salivary gland. Submandibular gland is the most affected one (80% to 95%) [3]. 5% to 20% of the cases are found in the parotid gland. The sublingual gland is uncommonly affected (1% to 2%). There are some characterics of the submandibular gland that influence this incidence:

1) Saliva from the submandibular gland is more mucinous and alkaline than the other glands.
2) The Wharton duct is longer, wider and more circuitous than the Stensen duct.
3) Calcium and phosphate quantity in submandibular saliva are higher than the other glands.
4) Gravity acts against the salivary secretion of the submandibular gland. [2]

There are several theories for formation of the calculi and none of them explain exact mechanism. Salivary stones are supposed to occur as a result of deposition of calcium salts around an organic debris that include mucins, bacteria and desquamated epithelial cells. [2, 6] Physical trauma, infection and inflammation of the gland, stagnancy of saliva flow are the other predisposing factors [5]. The use of drugs can be an alternative predisposing factor. Drugs can reduce salivary flow, change electrolyte concentration, decrease glycoprotein synthesis and degrade the cell membranes of the salivary glands [7]. In our second case, it is thought that long-term use of antiepileptic and antidepressant drugs are the ones that caused salivary gland stone.

Giant sialoliths are easily detected on panoramic radiographs as an radioopaque mass nearby the submandibular fossa. Occlusal radiography is a better option to visualize the stone without superposition of the other anatomic structures. To maintain detailed information CBCT is a wise option. In this case, we detected the exact location of the stone using the CBCT. Other imaging techniques, include sialography and ultrasonography also can be used to diagnose sialoliths. Ultrasonography is the best method to differentiate intraglandular and extraglandular masses. Sialography is the recommended method for evaluating abnormalities of the ductal system. This technique is especially useful for the evaluation of inflammatory conditions that are associated with sialoliths. [8]

Salivary stones are classified as ductal or intraglandular. Submandibular sialoliths mostly occur in ducts (75% to 85%) [9]. The location of the stone is very important for management of the treatment. In most of the cases, surgical excision of the stone is usually adequate but for the intraglandular stones complete excision of the affected gland together with the stone(s) must be thought.

Management of sialoliths depends on the size and localization of the stone and duration of the symptoms. Removal of stones with intraoral approach is recommended whenever stones can be palpated intraorally. [10] The most important purpose of the intraoral approach is recovering the secretory function after sialolithectomy. In our cases, we used a stent placed into the orifice of the duct to prevent the obstruction. For the stent placement, the hypospadias silastic stent tubes, pediatric feeding tubes and epidural catheter were used in many cases. [11, 12] Many researchers reported different duration times for stent placement. [11] We used an intravenouos cannula for 3 days. At the 1 year follow-up of the cases, there were no complication and it was seen that the ducts' orifices were open and there were no abnormalities of the secretory functions.

Newer treatment options including sialendoscopy, lithotripsy and laser fragmentation are effective alternatives to conventional surgery. [13]

Conclusion

Giant sialoliths represent a major challenge to oral surgeons in the choice of surgical approach to prevent excision of the gland and possibility of hypoesthesia, dry mouth, or salivary fistulae. The surgical technique for removal of sialoliths should be minimized to prevent gland morbidity. The purpose of this minimized, gland-preserving intraoral approach is restoring normal salivary flow. Salivary obstruction for long periods can cause fibrosis and atrophy of the affected gland. Giant sialoliths should be removed even when asymptomatic to prevent complications. We suggest that a catheter must be used to prevent obstruction of the salivary gland duct after the surgical removal of the stone intraorally. This method doesn't require any extra equipment and it is easy to apply.

References

1. Seifert G, Miehlke A, Haubrich J, Chilla R. Diseases of the salivary glands. Georg. Thieme Inc. New York 1986.
2. Siddiqui SJ. Sialolithiasis: an unusually large submandibular salivary stone. British Dental Journal. 2002 Jul 27;193(2):89-91.
3. Walvekar RR, Bomeli SR, Carrau RL, Schaitkin B. Combined approach technique for the management of large salivary stones. The Laryngoscope. 2009 Jun 1;119(6):1125-9.
4. Raveenthiran V, Rao PH. Giant calculus in the submandibular salivary duct: report of the first prepubertal patient. Pediatric surgery international. 2004 Feb 1;20(2):163-4.
5. Bodner L. Giant salivary gland calculi: diagnostic imaging and surgical management. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 2002 Sep 30;94(3):320-3.
6. Escudier M. Epidemiology and aetiology of salivary calculi. Controversies in the management of salivary gland disease. 2001 Nov 1:249-55.
7. Rauso R, Gherardini G, Biondi P, Tartaro G, Colella G. A case of a giant submandibular gland calculus perforating the floor of the mouth. ENT: Ear, Nose & Throat Journal. 2012 Jun 1;91(6).
8. Burket LW, Greenberg MS, Glick M, Ship JA. Burket's oral medicine. PMPH-USA; 2008. p. 191-223
9. Shetty BN, Sharma P. Unusual case of a projecting intraoral giant sialolith. Indian Journal of Surgery. 2010 Apr 1;72(2):155-7.
10. Rai M, Burman R. Giant submandibular sialolith of remarkable size in the comma area of Wharton's duct: a case report. Journal of Oral and Maxillofacial Surgery. 2009 Jun 30;67(6):1329-32.
11. Su CH, Lee KS, Tseng TM, Hung SH. Post-sialendoscopy ductoplasty by salivary duct stent placements. European Archives of Oto-Rhino-Laryngology. 2016 Jan 1;273(1):189-95.
12. Hiremath V, Patil AG, Aparna S. Surgical management of Stenson's duct injury using epidural catheter: A novel technique. Nigerian journal of clinical practice. 2013;16(2):266-8.
13. Sun YT, Lee KS, Hung SH, Su CH. Sialendoscopy with holmium: YAG laser treatment for multiple large sialolithiases of the Wharton duct: A case report and literature review. Journal of Oral and Maxillofacial Surgery. 2014 Dec 31;72(12):2491-6.

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.

• • •

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

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.

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

Azerbaijan Medical Association

P.O. Box - 16, AZ 1000, Baku, Azerbaijan

Tel: +99412 492 80 92, +99450 328 18 88

info@azmed.az, www.azmed.az

Reach your Global Audience

For more advertising opportunities:

www.amaj.az,

advertising@amaj.az

+99412 492 8092,

+99470 328 1888

Azerbaijan Medical Association

azmed.az