The Rare Cause of Middle Cerebral Artery Infarction Hemorrhagic Shock Due to Multiple Penetran Injury: Case Report

Orta Serebral Arter İnfarktüsü'nün Nadir Bir Sebebi; Multipl Penetran Yaralanmaya Bağlı Hemorajik Şok

Şenay GÖKSU TOMRUK,[a]
Ömer Torun ŞAHİN,[a]
Gülşah KARAÖREN,[a]
Vedat ÇAKIRTEKİN,[a]
Nevin KURT ÇELEBİ,[a]
Nurten BAKAN[a]

[a]Clinic of Anaesthesiology and Reanimation, Ümraniye Training and Research Hospital, İstanbul

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Yazışma Adresi/*Correspondence:*
Şenay GÖKSU TOMRUK
Ümraniye Training and Research Hospital, Clinic of Anesthesiology and Reanimation, İstanbul,
TÜRKİYE/TURKEY
senaytomruk@hotmail.com

ABSTRACT Middle Cerebral Artery (MCA) infarction can be fatal when it is massive. Here, we aimed to present a case with hypovolemia which is a rare cause of MCA infarction. A 47-years old woman with loss of consciousness was admitted due to multiple penetrating injuries at thorax, abdomen and gluteal region. She had hypovolemic shock due to massive hemorrhage and underwent immediate surgery. After surgery, she was transferred to intensive care unit (ICU). On the day 2, neurological examination revealed right hemiplegia and left gaze preference when sedation was withdrawn. Cranial computed tomography showed a severe left MCA infarction. On the day 12, adult respiratory distress syndrome (ARDS) developed and the patient died due to cardiopulmonary arrest on the day 18 of ICU admission. In conclusion, we aimed to emphasize importance of early, detailed neurologic examination for early recognition of MCA infarction, even in case of massive hemorrhage which is an extremely rare cause.

Key Words: Infarction, middle cerebral artery; shock; hemorrhage; hemiplegia

ÖZET Orta Serebral Arter İnfarktüsü (OSAİ) masif olduğu zaman öldürücü olabilmektedir. Bu yazıda nadir görülen sebeplerinden biri olan hipovolemik OSAİ'yı bir vaka ile sunmayı amaçladık. Hipovolemik şokta hastaneye getirilen 47 yaşında şuuru kapalı kadın hastanın, delici-kesici alet ile birden fazla (toraks, gluteal bölge ve batına nafiz) yaralanması mevcuttu. Kanama kontrolü için yapılan acil operasyon sonrası entübe olarak yoğun bakım ünitesine (YBÜ) alındı. YBÜ'de ikinci gününde sedasyonun kesilmesinden sonra yapılan nörolojik muayenede sağ hemipleji ve gözlerin sola deviasyonu tespit edildi. Bilgisayarlı tomografide yaygın OSAİ tespit edildi. YBU'de 12. günde yetişkin respiratuar distres sendromu (ARDS) gelişti, 18. gününde kardiyopulmoner arrest nedeniyle eks oldu. Sonuç olarak nadir sebeblerinden biri olan masif kanama durumunda bile OSAİ'nin acil tanısında erken, detaylı nörolojik muayene çok önemli bir yer tutmaktadır.

Anahtar Kelimeler: İnfarktüs, orta serebral arter; şok; kanama; hemipleji

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Basic findings of middle cerebral artery (MCA) infarction of either side are contralateral hemiplegia, a gaze preference towards lesion side which is probably due to damage of the lateral gaze center (Broadman area 8).* Overall mortality is 15-30%. The mortality is related to infarction within first 30 days, while it is related to pulmonary and cardiac complications on months 2 and 3.[1-3] Our case was a 47-years old woman

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who had multiple penetrating injuries and underwent immediate surgery. After surgery, she was admitted to intensive care unit (ICU) and diagnosed as unexpected MCA infarction. She died due to ARDS and MCA infarction.

CASE REPORT

This case was presented after obtaining informed consent from a family member. Here, we presented a 47-year old woman with multiple penetrating injuries who arrived to emergency department with loss of consciousness and hypovolemic shock. She underwent immediate surgery. Venous access was established as soon as possible and fluid resuscitation was initiated in the emergency department. Penetrating injuries were located at thorax, abdomen and gluteal region; thus, the operation was performed by a general surgeon and a cardiac surgeon together. Standard monitoring including electrocardiography, pulse oximetry, invasive blood pressure, central venous pressure and respiratory gas measurement was employed during surgery. Initial blood pressure was undetectable. After aggressive fluid resuscitation and blood transfusion, blood pressure was measured as 96/67 mmHg (on the 30. min of operation). Preoperative hemoglobin value was 6 g/dL. Overall, 4 packages of red blood cell and 2 packages of fresh frozen plasma were transfused to the patient. In the operation, it was observed that there were cardiac tamponade, bilateral pneumothorax, injury of epigastric artery and vein, laceration on the left lung, left hemothorax and lesion on right gluteal region. Surgery lasted 4 hours and laceration on the left lung was repaired with simple sutures; bilateral chest tubes were inserted; injured epigastric artery and vein were ligated and required intervention was performed against cardiac tamponade. There was no solid organ or bowel injury.

After operation, the intubated patient was transferred to the intensive care unit on sedation. The following findings were noted at admission to ICU: bilaterally isochoric and reactive pupils; central venous pressue, 8 mmHg, heart rate, 87 beats per minute, blood pressure, 140/80 mmHg; and hemoglobin, 8,1 g/dL.

On the second day of ICU admission, the patient became hemodynamically stable; then, sedation was withdrawn. Neurological examination revealed right hemiplejia and left gaze preference with limited cooperation. Cranial CT showed a large acute brain infarction at the left middle cerebral artery territory (Figure 1). Consulting neurologist prescribed acetylsalicylic acid (300 mg daily).

On the following day, the patient on mechanical ventilation had somnolence, impaired cooperation and right hemiplegia. Early weaning protocol was failed. Respiratory parameters persisted below weaning values due to either infarct itself or hemifacial neglect. On the day 12, ARDS developed in the patient and she underwent percutaneus tracheostomy on the day 15. The patient died due to cardiopulmonary arrest on the day 18.

DISCUSSION

In case of unilateral hemiparesis, homonymous hemianopsia, hemifacial neglect and sensorial lost or sensorial inattention, the most common cause is contralateral MCA infarction; in addition, aphasia can be also seen if dominant hemisphere is affected.

The primary causes of MCA infarction include ischemia, cardiac embolism, hypercoagulable state, hemorrhage, hypertension, and amyloid or

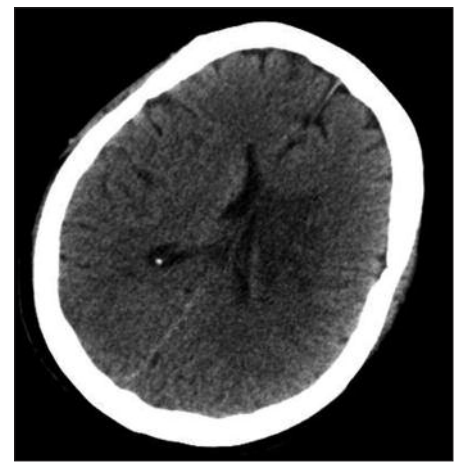

FIGURE 1: Image of head CT scan showing large infarct in the middle cerebral artery territory.

arteriovenous malformation, while secondary causes of infarction include indirect ischemia due to arrhythmia and other causes of hypotension such as hypovolemic shock. Excessive reduction of systemic blood pressure regardless of its cause may lead ''hypotensive stroke''. Large artery strokes may result from hypoperfusion. Such strokes presents more severe baseline neurologic deficit; thus, conventional therapeutic approaches may fail in such patients.[1,2]

All indicated interventions must be immediately performed in patients with ischemia, since "time lost is brain lost''. The goals are to limit area of ischemia/infarct and salvaging the penumbra. The penumbra is a hypoperfused area of focal ischemia that is potentially viable and may be salvaged by timely and appropriate intervention, particularly by maintaining euvolemia. Once stabilization is achieved, the goal of therapy should be directed to the underlying cause. In MCA infarction, medical therapy includes all basic and advanced life support measures. This should be followed by specific therapies such as anticoagulation and thrombolytic therapy in the case of tromboembolic event. After exclusion of hemorrhagic causes, acetylsalicylic acid (ASA) used at doses of 160-300 mg reduces mortality within first week. This is especially true when it is started within first 48 hours in particular. Also, symptomatic treatment should be considered, including antiepileptic medication and agents directing increased intracranial pressure if indicated. Surgical therapy includes surgical decompression based on underlying etiology and severity of disease.[2,4-6]

In our patient, there was an extremely rare cause for left MCA infarction: hypotension due to hemorrhagic shock induced by multiple penetrating injuries. While patient was sedated, there was no diagnosis. Although it was failed to detect MCA infarction in the patient, appropriate goal-directed medical treatment was performed as rapid as possible, including basic and advanced life support; measures directing airway, breathing and circulation parameters and vital signs; immediate correction of hypovolemia; and specific therapy with ASA within the first 48 hours after event.

In our patient; there was no midline shift on cranial CT; thus, no surgical intervention was indicated. However, there were findings associated with poor outcome such as large lesion on CT scanning, initial unconsciousness, hemianesthesia and hemifacial neglect.[2,7-12]

Unfortunately, the patient became ventilator-depended most probably due to large infarct area; consequently, ventilator associated pneumonia and ARDS developed in the patient. Thus, low volume strategy was employed, and combined antibiotic therapy was initiated based on results of culture tests. However, she died due to cardiopulmonary arrest on the day 18 of ICU admission.

Although we failed to find a MCA infarction case associated with hypotension, 4 case reports were identified on rare causes of MCA infarction in the literature. In these case reports; MCA infarction developed: 1) following disseminated intravascular coagulation; 2) following trauma; it was associated with progressive occlusion of the internal carotid artery after minor head injury; 3) after fall from bicycle, it was due to extra-cranial narrowing of the left internal carotid artery on the neck caused by hematoma; 4. following multiple bee sting. In all reported cases, neurologic findings such as unilateral hemiparesis, unconsciousness, hemiplegia, gaze preference towards lesion side and limited cooperation were present in varying degrees; thus, cranial and neck imaging studies were performed for diagnostic purposes. Neurologic examination isn't only important for neurologist but also for all specialists including anesthesiologist.[13-16]

We suggest that our patient died to ARDS and MCA infarction itself which resulted from hypotension and ischemia. In conclusion, we aimed to emphasize importance of early, detailed neurologic examination for early recognition of MCA infarction. MCA infarction should be kept in mind even in case of massive hemorrhage, an extremely rare cause.

REFERENCES

1. Duncan QM. Neurosurgical critical care. In: Frederick SB, Darryl YS, Janine REV, eds. Current Diagnosis and Treatment: Critical Care. 3rd ed. Philadelphia: McGraw-Hill; 2008. p.673-6.
2. Baldwin K, Orr S, Briand M, Piazza C, Veydt A, McCoy S. Acute ischemic stroke update. Pharmacotherapy 2010;30(5):493-514.
3. White H, Boden-Albala B, Wang C, Elkind MS, Rundek T, Wright CB, et al. Ischemic stroke subtype incidence among whites, blacks, and Hispanics: the Northern Manhattan Study. Circulation 2005;111(10):1327-31.
4. Wang DZ, Nair DS, Talkad AV. Acute Decompressive Hemicraniectomy to Control High Intracranial Pressure in Patients with Malignant MCA Ischemic Strokes. Curr Treat Options Cardiovasc Med 2011;13(3):225-32.
5. Feigin VL, Lawes CM, Bennett DA, Barker-Collo SL, Parag V. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. Lancet Neurol 2009;8(4):355-69.
6. Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke 2007; 38(5):1655-711.
7. Lewandowski C, Barsan W. Treatment of acute ischemic stroke. Ann Emerg Med 2001; 37(2):202-16.
8. Laplane D, Degos JD. Motor neglect. J Neurol Neurosurg Psychiatry1983;46(2):152-8.
9. Grimsen C, Hildebrandt H, Fahle M. Dissociation of egocentric and allocentric coding of space in visual search after right middle cerebral artery stroke. Neuropsychologia 2008; 46(3):902-14.
10. Yaka E. [Pathophysiology of stroke (cerebral blood flow, autoregulation, cerebrovascular reserve, ischemic injury)]. Turkiye Klinikleri J Neurol-Special Topics 2011;4(3):7-11.
11. Tolun R. [Endovascular therapies for acute ischemic stroke: endovascular pharmacologic].Turkiye Klinikleri J Neurol-Special Topics 2011;4(3):66-71.
12. Tolun R. [Endovascular therapies for acute ischemic stroke: endovascular multimodel]. Turkiye Klinikleri J Neurol-Special Topics 2011;4(3):72-83.
13. Viswanathan S, Muthu V, Singh AP, Rajendran R, George R. Middle cerebral artery infarct following multiple bee stings. J Stroke Cerebrovasc Dis 2012;21(2):148-50.
14. Wang CH, Lee MS, Lin LC, Yang RC. Multiple organ infarctions following disseminated intravascular coagulation precipitated by sepsis in a healthy infant: a case report. Kaohsiung J Med Sci 2010;26(12):663-8.
15. Matsumoto H, Kohno K. Posttraumatic cerebral infarction due to progressive occlusion of the internal carotid artery after minor head injury in childhood: a case report. Childs Nerv Syst 2011;27(7):1169-75.
16. Ramanujam P, Lowenstein R. Visual diagnosis in emergency medicine:hyperdense middle cerebral arter sign. J Emerg Med 2007; 33(4):417-8.