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Case report

Non-surgical treatment of massive traumatic corpus callosum hematoma after blunt head injury: A case report

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ARTICLE INFO

Article history:
Received 10 September 2015
Received in revised form
13 February 2016
Accepted 13 April 2016
Available online 27 April 2016

Keywords:
Corpus callosum hematoma
Blunt head trauma
Conservative treatment

ABSTRACT

Massive hematoma of the corpus callosum caused by blunt head trauma is an extremely rare lesion. Most frequent traumatic lesions involve the corpus callosum are diffuse axonal injuries. They might be associated with small hemorrhagic foci in the hemispheric and brain stem white matter, intraventricular hemorrhages, subarachnoid hemorrhages, traumatic lesions of the septum pellucidum and fornix. Many cases of corpus callosum injury present with permanent disconnection syndrome. We present a case of a 32-year-old female suffered blunt head trauma resulted in massive corpus callosum hematoma which was managed non-surgically. The patient initially had a reduced conscious level and symptoms of disconnection syndrome, and significant recovery was observed at 6 months follow up.

1. Introduction

Focal corpus callosum (CC) injury is an indicator to severe brain injury. This type of injury is likely due to either disruption of axons at the time of the trauma or due to torsion or shearing strains on the CC and it is a measure of severe brain injury. Clinical outcomes include persistent vegetative state or mutism. Disconnection syndrome associated with cognitive deficits as memory loss can occur if the posterior callosal lesion extends as far forward as the fornix. On the other hand, interhemispheric disconnection syndrome after head trauma have been reported rarely in the literature, probably often overlooked [1].

Hemorrhagic lesion of the corpus callosum is a rare feature in subarachnoid hemorrhage (SAH), which may result from ruptured aneurysms of the anterior communicating artery, pericallosal artery or after ruptured arteriovenous malformation (AVM) or arteriovenous fistula [2].

Magnetic resonance imaging (MRI) often demonstrates small hemorrhagic foci in the CC or surrounding structures in diffuse axonal injury [3].

2. Case report

A 32-year-old female was pedestrian hit by a vehicle at high speed. She sustained polytrauma including severe head injury.

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http://dx.doi.org/10.1016/j.pjnns.2016.04.005

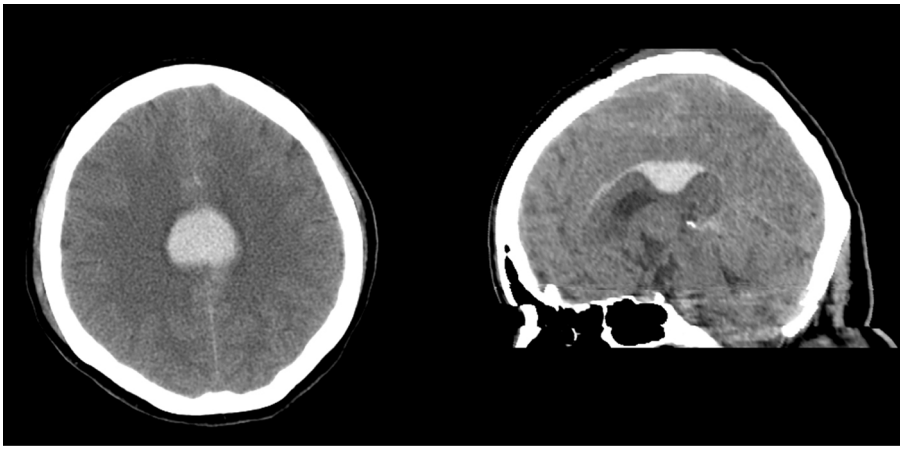

Fig. 1 – CT of the brain on admission.

Admission Glasgow Coma Scale (GCS) was (E4, V1, M5), aphasic, with severe right lower limb weakness (MRC Grade 1/5) with an up-going right plantar response. A computer tomography (CT) scan of the brain demonstrated a large hematoma in the CC involving mainly the splenium (Fig. 1). MRI and magnetic resonance angiography (MRA) did not reveal any vascular cause (Fig. 2). Associated injuries included: fractures of the C6, C7 spinous processes, an undisplaced fracture of the left articular facet of C7, fracture of the antero-superior aspect of T3 and T4 vertebral bodies, left 6th ribfracture, fracture of pelvis, fracture of shaft of left tibia (treated by open reduction and internal fixation).

The patient was treated with intensive care, anti-seizure medication, nasogastric feeding and bed rest for the pelvic injury. Her conscious level was improved at 14 days post-injury, and she started to show improvement of the right lower extremity motor power after three weeks post-trauma under influence of condensed physiotherapy sessions. At six month follow up the patient was fully conscious, oriented, could verbally communicate on her mother tongue fluently and also started walking with little assistance (the gait difficulties were due to the pelvic and lower extremity injuries not the consequences of the head injury), could climb the stairs and she was independent in taking care of herself.

The neuro-psychological and cognitive function assessment of the patient could be performed only partially for several reasons: the patient's mother tongue was unique in this country, she could not speak the local language and she was not able to communicate fluently in English as well. Initially she was obtunded due to the head trauma, later on due to the pain medication for the other injuries. She had also a major emotional trauma for she lost her two years old daughter during the accident. However, obviously the patient was aphasic initially for two weeks and later on she started to communicate verbally. Initially we could find left sided neglect syndrome, impaired visual recognition, alexia and she seemed to be akinetic. At the time of the six months follow up, she mentioned she had some difficulties with understanding what she reads; she had no problems of memory and of communicating on her native language. She demonstrated left sided apraxia.

Regular MRI brain follow ups were done on the 5th day, 1, 3, 6 month after injury, which revealed gradual reduction in size of the hematoma (Figs. 3 and 4).

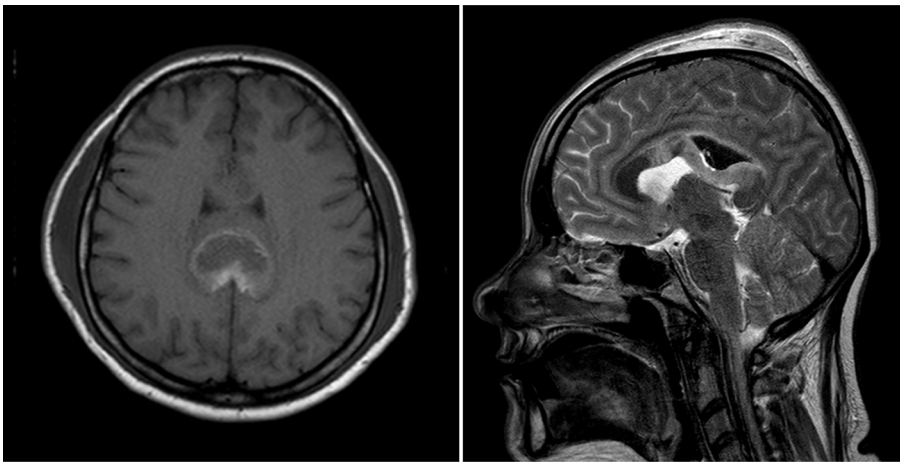

Fig. 2 – MRI of the brain done one month after the impact.

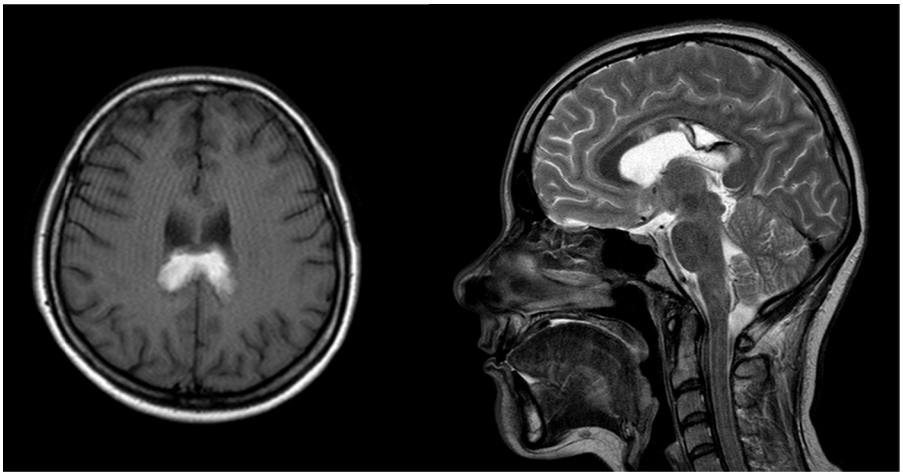

Fig. 3 – MRI brain done after three month from the impact.

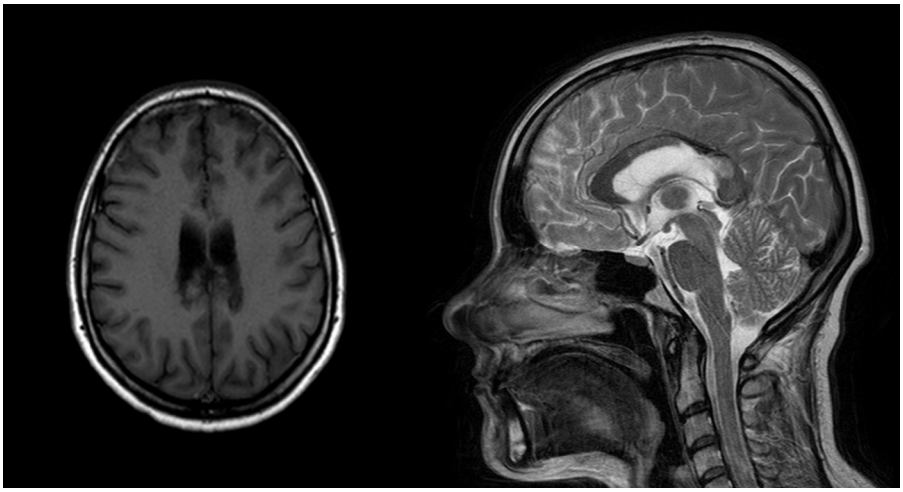

Fig. 4 – MRI brain done six month after the impact.

3. Discussion

Corpus callosum hematoma can be caused by vascular abnormalities (ruptured pericallosal aneurysm or vascular malformation), hypertension, amyloid angiopathy, intra-tumoral or encephalitic hemorrhage. It is rarely caused by traumatic brain injuries [4].

Severe head injuries are often associated with rotational forces resulting in shear injuries in brain parenchyma causing diffuse axonal injuries and commonly involve the CC. This has been described by Gennarelli et al. [5] and it has been reported in other studies that brain displacement during head impact against sharp edge of the falx cerebri can cause CC hematoma [6].

On reviewing the literature, we found that the common anatomical location of traumatic CC hematoma is the posterior portion of the CC (body and splenium) as its thick fibers is more affected by rapid displacement of the brain during the impact [7,8]. On the contrary, spontaneous aneurysmal SAH associated with hematomas in the CC usually involves the ventral aspect of the anterior CC [9]. It has been also reported that traumatic CC hematoma may be accompanied by interhemispheric fissure, septum pellicidum or intraventricular hemorrhage [10,11]. Similarly, Shigemori et al. [12] reported five cases of massive hematoma of the CC caused by blunt head trauma with concomitant intraventricular hemorrhages and SAH [13].

In this case the CC hematoma is likely to have been originated from small focal contusions causing mainly a compressive effect and peri-focal edema of the CC bundles rather than direct extensive axonal injury. This pathology can explain the satisfactory clinical outcome during and after the spontaneous absorption of the hematoma resulting in near full recovery at the follow up after sixth months.

Our patient's excellent prognosis was favored by her young age, good coma score on admission, focal CC involvement (splenium only), and no signs of diffuse axonal injury, traumatic aneurysmal rupture or vascular injury. Other studies reported worse prognosis with old age, low admission GCS and diffuse CC damage [14].

Significant clinical improvement of our patient inspite of slow radiological resolution of the hematoma supports the decision of not attempting to evacuate this lesion leads to a favorable outcome. Other researchers reported that surgical

treatment of CC hematoma can contribute to high morbidity (deficits of memory, dysexecutive cognitive and behavioral syndrome, and disturbances in interhemispheric transfer of learning) and mortality due to the damage of a large number of callosal fibers and the adjacent brain tissue during interhemispheric transcallosal approach and hematoma evacuation [15,16].

4. Conclusion

We report a rare case of isolated corpus callosum hematoma after closed head injury. The patient had clinical features of disconnection syndrome and made a satisfactory recovery with non-surgical management. We recommend to treat such cases conservatively if patient is neurologically stable and clinically improving.

Conflict of interest

None declared.

Financial disclosure

None declared.

Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

REFERENCES

[1] Vuilleumier P, Assal G. Lesions of the corpus callosum and syndromes of interhemispheric disconnection of traumatic origin. Neurochirurgie 1995;41(2):98–107.
[2] Takatoshi S, Naoki Y, Osamu S. Hematoma in the splenium of the corpus callosum in the subacute stage of subarachnoid hemorrhage. Neurol Med Chir (Tokyo) 2010;50(3):209–12.
[3] Kasahara T, Toyokura M, Shimoda N. Cerebral hemorrhage restricted to the corpus callosum. Am J Phys Med Rehabil 2005;84(5):386–90.
[4] Kanpolat Y, Mertol T. Haematoma of cavum septi pellucidi due to hypertension. Acta Neurochir (Wien) 1987;89(3–4): 135–6.
[5] Gennarelli T, Thibault L, Graham D. Diffuse axonal injury: an important form of traumatic brain damage. Neuroscientist 1998;4:202–15.
[6] Du Y, Han Z, Zheng S. Delayed massive traumatic hematoma in the corpus callosum: two case reports with literature review. NMC Case Rep J 2014;1:37–41.
[7] Jain SK, Sundar IV, Sharma V. Bilateral large traumatic basal ganglia hemorrage in a conscious adult: a rare case report. Brain Inj 2013;27:500–3.
[8] Mendelsohn DB, Levin HS, Harward H. Corpus callosum lesions after closed head injury in children: MRI, clinical features and outcome. Neuroradiology 1992;34:384–8.
[9] Erbaş G, Oner AY, Akpek S. Corpus callosum hematoma secondary to isolated inferior sagittal sinus thrombosis. Acta Radiol 2006;47(10):1085–8.
[10] Matsukawa H, Shinoda M, Fujii M. Genu of corpus callosum as a prognostic factor in diffuse axonal injury. J Neurosurg 2011;115:1019–24.
[11] Risse GL, Gates J, Lund G. Interhemispheric transfer in patients with incomplete section of corpus callosum. Arch Neurol 1989;46:437–43.
[12] Shigemori M, Kojyo N, Yuge T. Massive traumatic haematoma of the corpus callosum. Acta Neurochir (Wien) 1986;81(1–2):36–9.
[13] Mochizuki K, Ochi H, Ogura Y. A case of diffuse axonal injury in violent death. Leg Med (Tokyo) 2009;1:518–9.
[14] Zheng WB, Liu GR, Kong KM. Coma duration prediction in diffuse axonal injury: analysis of apparent diffusion coefficient and clinical prognostic factors. Conf Proc IEEE Eng Med Biol Soc 2006;1:1052–5.
[15] Atzema C, Mower WR, Hoffman JR. National emergency X-radiography utilization study (Nexus) II group: prevalence and prognosis of traumaticintraventricular hemorrhage in patients with blunt head trauma. J Trauma Neurol 2006;60:1010–7.
[16] Peltier J, Roussel M, Gerard Y. Functional consequences of a section of the anterior part of the body of the corpus callosum: evidence from an interhemispheric transcallosal approach. J Neurol 2012;259(9):1860–7.