SAGE Open Medical Case Reports
Volume 11: 1–4

DOI: 10.1177/2050313X231178400
journals.sagepub.com/home/sco
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Traumatic retroclival hematoma complicated with hyponatremia and delayed traumatic intracranial hematoma in an adult: A case report

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Abstract

Intracranial hematoma is a common variety of brain insults in trauma. However, posterior fossa hematoma in the retroclival location is quite unusual. There are limited numbers of case reports regarding traumatic retroclival hematoma. Some are managed with surgery in this condition. We present a traumatic retroclival hematoma in a 34-year-old gentleman who sustained brain trauma in a motor vehicle accident. His condition was further complicated by hyponatremia and delayed traumatic intracerebral hematoma in a distant location. The only symptom he had later was severe headache which could be attributed to delayed traumatic intracerebral hematoma and hyponatremia. He was managed conservatively and discharged on the 12th day from the hospital.

Keywords

Retroclival hematoma, intracranial hematoma, hyponatremia, conservative management

Date received: 8 April 2023; accepted: 10 May 2023

Introduction

Retroclival hematoma (RH) is quite a rare entity following trauma in an adult that develops anterior to the brainstem in the retroclival spaces.[1] It is a small subset of posterior fossa extra-axial hematomas, which account for about 0.3% of all acute extra-axial hematomas.[2] It may result from injury of any of the blood vessels in the posterior fossa or at times may be injury to the pre-existing vascular anomalies. Etiology is frequently related to accidental trauma, but other mechanisms have been observed, including coagulopathy, non-accidental trauma, pituitary apoplexy, and ruptured aneurysm. However, sometimes it is spontaneous with no discernible cause.[2] The pathophysiology of the formation of a RH in adults remains controversial.[3,4] It typically develops as a consequence of a high-energy trauma, with the majority of cases occurring in individuals involved in motor vehicle accidents as a pedestrian, cyclist, or passenger.[4] The relation of intracranial hematoma with hyponatremia is well established in the literature.[5,6] We report a case of RH which was further complicated by electrolyte imbalance and delayed traumatic intracerebral hematoma in a distant location.

Case history

A 34-year-old male presented to the emergency department with a history of vehicle accident for 4 h. At initial assessment in ER, he had blood pressure, oxygen saturation, heart rate, and respiratory rate of 110/90 mm of Hg, 97% under room air 94 beats/min and 16/min, respectively. On examination, both pupils were round, regular, and reactive, with normal plantar reflexes. The initial assessment of consciousness

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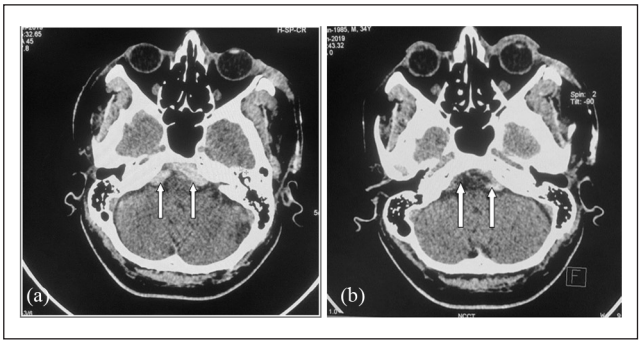

Figure 1. Axial CT scan of the head (a) with retroclival hematoma and (b) with resolution of the hematoma.

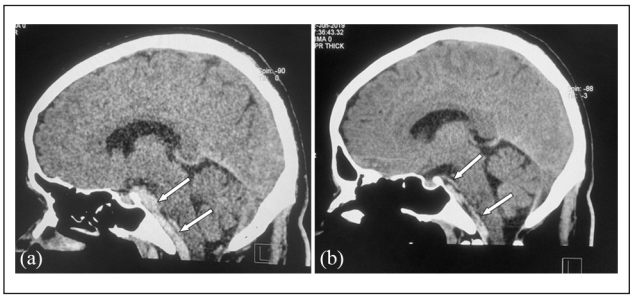

Figure 2. Sagittal CT scan of the head showing (a) retroclival hematoma and (b) resolving hematoma.

was not reliable because of alcohol consumption. His Glasgow coma scale (GCS) the following day was 15/15. The patient had difficulty in swallowing liquid; otherwise, there was no focal neurological deficit. Other systemic examinations were unremarkable.

The laboratory examination revealed decreased hemoglobin (10.4 g/dL), leukocyte count (7900 cells/mm^3), neutrophil (60%), lymphocyte (32%), platelets (153,000 cells/mm^3), normal prothrombin time (12 s), normal international normalized ratio (INR; 0.9 s), blood urea nitrogen (12 mg/dL), creatinine (0.8 mg/dL), random blood sugar level (112 mg/dL), normal sodium (135 mEq/L), normal potassium (4 mEq/L), erythrocyte sedimentation rate (10 mm/h), and ECG showed normal rhythm.

Contrast tomography (CT) scan head at the presentation showed a hyperdense lesion just posterior to the clivus extending down to the anterior part of the medulla consistent with clotted blood Hounsfield unit (60 HU) and hypodense area in the left occipital region, a non-hemorrhagic contusion (Figures 1 and 2). CT scan of the craniovertebral junction did not show any trauma. However, images showed fracture of left zygomatic arch, anterior and lateral wall of left maxillary sinus with associated hemosinus. X-ray of the cervical spine did not reveal any traumatic lesion.

The patient was admitted to ICU and was managed conservatively with cervical immobilization, intravenous fluid, methylprednisolone, and analgesics. Blood parameters measured on the second day were normal. The renal function test was normal. A repeat CT scan of the head on third day revealed similar status. On Day 5, he developed a severe headache and restlessness. Serum sodium was found to be 126 (135–145) mmol/L. A repeated CT scan of the head showed intracerebral hematoma in the previous non-hemorrhagic contusion site. Hyponatremia was corrected with fluid restriction and 3% NaCl. His headache gradually subsided and his sodium level was restored. His swallowing difficulty subsided gradually. The fracture was undisplaced and treated conservatively with immobilization and a follow-up CT scan. He was discharged from the hospital on the 12th day of admission without any major issues. After a week, 1 month, and 3 months, the patient was doing well with no significant issues.

Discussion

Traumatic RH is a rare entity.[2] According to Agrawal and Cochrane,[7] the most common variant of RH is epidural RH, which accounts for 1.2%–12.9% of all the epidural hematomas. It is relatively asymptomatic, if not associated with other bony or neurological injuries. Most of the studies mentioned about the pediatric age group; however, it is also found in adult. The preponderance of reported pediatric cases relative to adult cases may be attributed to the anatomical differences at the craniocervical junction. Compared to adults, children possess certain features (large head-to-body proportion, small occipital condyles, shallow facet joints, and weak cervical muscles) that increase the mobility of the spine and augment the risk for injury.[2] It is presumed that these hematomas are most likely the result of injury to the dural sinuses at the fracture of the skull base.[8] Many articles considered the stability of the craniovertebral junction and the status of ligaments,[2,8,9] but we did not find any abnormality in our case. Reported cases of traumatic retroclival hematoma in adults are shown in Table 1.

The clinical presentation can vary, with neurological impairment possible as a result of nerve and brain tissue compression or contusion. The abducens, optic, oculomotor, trigeminal, facial, glossopharyngeal, and hypoglossal nerves are the most commonly affected. Patients can also develop hemiparesis or quadriparesis, with more severe cases potentially resulting in brain stem contusion and cardiorespiratory compromise, as well as progressive hydrocephalus.[4,10]

Magnetic resonance (MR) and CT imaging are used to best visualize posterior fossa hemorrhage and intra- or extradural RH, and craniovertebral ligament injuries as well as clot migration, and occipitocervical fractures.[11,12]

Because of the complex anatomy of the craniocervical junction and the difficulties in surgical approach, conservative management, such as cervical immobilization, methylprednisolone, and close monitoring with imaging, is preferred over surgery. However, if the neurological condition worsens or there is craniocervical instability, surgery might be necessary.[13] With conservative management, most of the cases have good outcomes with minimal long-term neurological deficits, with the exception of a few cases[11,14]

Table 1. Cases of traumatic retroclival hematoma in adults.

Article	Age (years)/ gender	Traumatic cause	Findings	Treatment	Outcome
Oliviero et al.[14]	66/female	Whiplash injury	Severe tetraparesis, respiratory muscle weakness, dysarthria, dysphagia	Conservative	Partial recovery, in the context of pontomedullary infarction
Casey et al.[12]	18/male	Found collapsed in street (seemingly trivial head injury)	GCS 13; headaches and nausea, minimal displacement of the odontoid peg in relation to the atlas	Conservative	Recover with no neurological deficits
Sridhar et al.[24]	18/male	Fall from a two-wheeler	GCS 15; loss of consciousness	Operative (right-sided far lateral approach)	Recovered
Sridhar et al.[24]	19/male	Fall from a moving bus	GCS 15; headache and neck pain	Conservative	Recovered
Datar et al.[11]	75/male	Fall from standing height, also on warfarin	GCS n/a; headache, neck pain, and stiffness; type II odontoid fracture	Operative	Death due to airway obstruction on 14th day
Pérez-Bovet et al.[1]	68/male	Car accident	GCS 15; occipital and chest (seat belt bruise); odontoid base fracture.		Death due to cardiorespiratory arrest
Izumida and Ogura[25]	64/male	Hit the back of his head against the ground in a syncopal event	GCS n/a; history of syncope	Conservative	Recovered
Solorio-Pineda et al.[26]	35/male	Fall from 5-m height	GCS 13; retroclival subdural hematoma that extended to the C2 level; nondisplaced right frontotemporal fracture and fractures of the right orbital floor, lateral wall/ roof, and nasal bones; left mandibular ramus fracture, plus a distal metaphyseal fracture of the left wrist	Operative	Recovered
Piccirilli et al.[4]	76/male	Unclear	GCS 13; nausea and vomiting, clival fracture	Conservative	Recovered
Caglar et al.[13]	89/female	Fall	Quadriparesis, GCS 15, plantar reflex mute, no visible fracture/dislocation of skull base	Conservative	Recover with no neurological deficits

that result in death.[4] The duration of RH resorption can vary and there is no established treatment to accelerate the process. However, steroids may be administered due to their membrane-stabilizing effect, as venous and dural tension play a role in the bleeding mechanism.[13]

Electrolyte imbalance is not an uncommon entity in head injury patients. Hyponatremia is a condition that may result from either syndrome of inappropriate antidiuretic hormone (SIADH) or cerebral salt wasting (CSW).[5] Most of the time in our clinical setup, a clear distinction between these two causes of hyponatremia is not made and is not treated symptomatically. Electrolyte imbalance is a self-limiting condition most of the time. However, hyponatremia is considered as an independent predictor of poor neurological outcome in patients with traumatic brain injury.[15,16] Rajagopal et al.[17] suggest that early treatment with fludrocortisone in cases of hyponatremia with natriuresis can lead to reduced hospitalization. This protocol may be safer in tropical areas where fluid restriction can be dangerous, and it also eliminates the need to distinguish between SIADH and CSW. Proper hyponatremia management is essential for preventing complications and adverse outcomes. Hemorrhagic transformation of brain contusion is not an uncommon condition, described in several studies.[18–21] It may contribute to the subsequent clinical deterioration and need for surgical intervention. It was believed to have been caused due to the continued bleeding of microvessels fractured at the time of injury, exacerbated by overt or latent coagulopathy.[22] The concept of traumatic penumbra, an area of reduced metabolism and more susceptible to secondary insults, has recently been emphasized.[23]

Conclusion

Though a rare condition, RH may be life-threatening, at times, due to compression on the posteriorly lying vital centers in the pons or medulla. Another essential condition to look on is the status of the craniovertebral junction. Our case emphasizes the importance of continuous monitoring of patients with traumatic brain injury, even if their initial evaluation indicates a minor injury. The development of an intracerebral hematoma

and decreased serum sodium levels in this patient emphasizes the importance of close monitoring and appropriate management of potential complications.

Acknowledgements

The authors do not have any acknowledgment to report for this manuscript.

Author contributions

C.P.Y., S.D., and H.B.B. wrote the original manuscript, reviewed, and edited the original manuscript. M.B., S.L., I.S., and P.S. reviewed and edited the original manuscript.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Informed consent

Written informed consent was obtained from the patient for the anonymized information to be published in this article.

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