



Posttraumatic Isolated Diffuse SAH Because of Atlas (C1) Fracture Involving Transverse Process without Vertebral Artery Injury: A Case Report

Akshay Patil¹ Sandip Iratwar¹ Ashish Jha¹

¹ Department of Neurosurgery, DMIMS, Jawaharlal Nehru Medical College, Acharya Vinoba Bhave Rural Hospital, Wardha, Maharashtra, India

Indian J Neurosurg

Address for correspondence Akshay S. Patil, MS, MCh, Department of Neurosurgery, DMIMS, Jawaharlal Nehru Medical College, Acharya Vinoba Bhave Rural Hospital, Sawangi, Wardha, Maharashtra, India 442001 (e-mail: dr.akshayarbat@gmail.com).

Abstract

Traumatic subarachnoid hemorrhage (SAH) can be seen in mild, moderate, and severe head injury. Traumatic SAH is usually present in the sulci or superficial subarachnoid space (SAS) of the cortex rather than in the basal SAS. Basal SAH is usually harmful because of the injury to vascular structure resulting into vasospasm and ischemia. Here we are presenting a rare case in which patient is presented with traumatic injury to the neck followed by the isolated diffuse SAH mostly basal without injury to the brain parenchyma. Patient was presented with the raised intracranial pressure features (headache, vomiting) and seizure. On admission, he had a Glasgow Coma Scale of E3V3M5. His computed tomography (CT) of the brain was suggestive of diffuse SAH in all cisterns especially in basal cistern. He was evaluated by angiogram that was suggestive of no major vascular involvement. His CT cervical spine was suggestive of Atlas (C1) fracture involving transverse process with medial and inward displacement of bone, probably causing vascular insult in the form of venous injury leading to the SAH.

Keywords

- ▶ intracranial pressure
- ▶ magnetic resonance imaging
- ▶ subarachnoid hemorrhage
- ▶ traumatic brain injury

Introduction

Traumatic brain injury (TBI) is the most common cause of mortality and morbidity in low- and middle-income countries in adult population.¹ Cases estimated are around 50 million per year. TBI is the most common cause of subarachnoid hemorrhage (SAH). Moderate and severe TBI usually have 33 to 60% of patients with SAH.^{2,3} Road traffic accident, fall from height, and violence (assault) mostly contribute to TBI. Traumatic SAH is adverse prognostic factor leading to progressive neurological deterioration³ because of the vasospasm, electrolyte imbalance, hormonal imbalance (pituitary injury), raised intracranial pressure, and hydrocephalus. It increases the mortality by double in patient with TBI. Here we are presenting a case of traumatic

SAH in various cortical sulci and in all major cisterns after traumatic injury to left side of neck and jaw by hard object. Patient had Atlas (C1) fracture involving left transverse process without injuring major vessel or its tributaries.

A 51-year-old male patient with no known comorbid illness admitted with alleged history of assault by metal rod on the left side of neck followed by vomiting, neck pain, and loss of consciousness. He had one episode of generalized tonic-clonic seizure before admission. Initially he took primary treatment at local hospital and then shifted for further evaluation. On admission patient was E3V3M5. At emergency room, patient underwent computed tomography (CT) brain plain suggestive of diffuse SAH involving bilateral sylvian fissure, cortical sulci, and basal cistern. It was extending till left-side cerebellopontine, medullary fissure,

DOI <https://doi.org/10.1055/s-0043-1778688>.
ISSN 2277-954X.

© 2024. The Author(s).

This is an open access article published by Thieme under the terms of the Creative Commons Attribution License, permitting unrestricted use, distribution, and reproduction so long as the original work is properly cited. (<https://creativecommons.org/licenses/by/4.0/>)

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

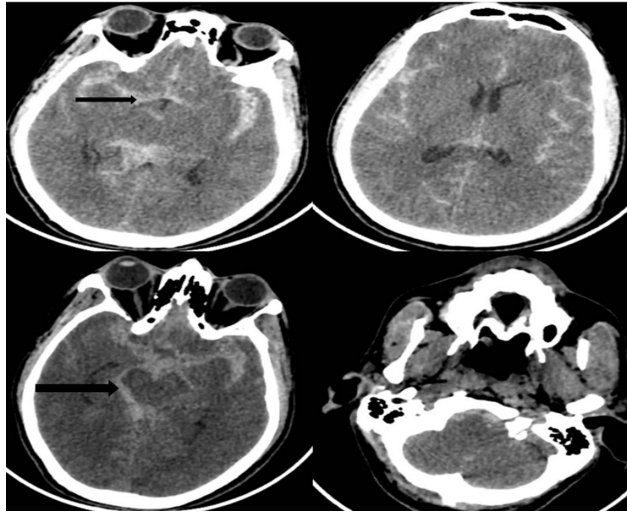


Fig. 1 Computed tomography (CT) brain plain suggestive of subarachnoid hemorrhage (SAH) in bilateral sylvian, suprasellar cistern, interhemispheric space with diffuse basal cistern involvement (black arrow). No intraventricular extension shown. SAH is present in left cerebellopontine angle and left cerebellomedullary cistern. No parenchymal involvement shown.

and clivus as well (►**Fig. 1**). He underwent angiogram (CT/DSA [digital subtraction angiography]) suggestive of no aneurysm or vascular malformation. Vertebral angiogram was also clear not showing any injury to parent vessel or to Posterior inferior cerebellar artery (PICA) (►**Fig. 2**). His magnetic resonance imaging (MRI) brain and cervical spine was suggestive of blood products in all cisterns with multiple small hemorrhages in corpus callosum. CT cervical spine suggestive of left C1 transverse process fracture with inward and medial dislocation (►**Fig. 3**). Patient was managed conservatively with antiedema, antiepileptic drugs, and cerebral vasodilators. Gradually he improved and subsequent CT brain showed resolution of SAH.

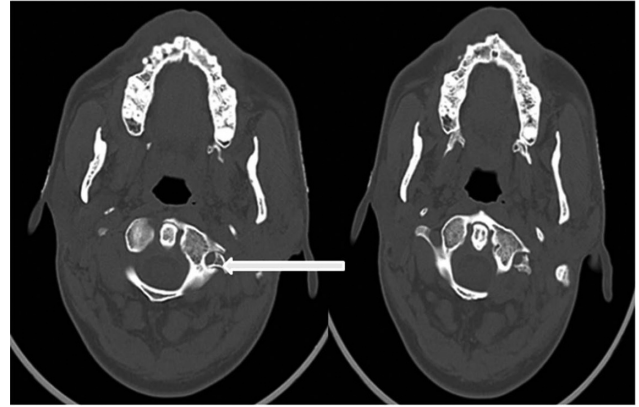


Fig. 3 Computed tomography cervical spine axial images showing fracture of Atlas with involvement of transverse process with inward and medial displacement (white arrow).

Discussion

Traumatic basal SAH is well documented in the younger intoxicated males who have been involved in assault to the head, neck, and face.⁴ Most common form of the vascular injury is from the intracranial portion of vertebral artery.⁵ Possible mechanisms described in the literature for traumatic SAH are (1) direct trauma to vessel, (2) extensive stretching during hyperextension or rotation of the neck, (3) oscillation of brain with shearing forces, (4) increased intravascular pressure from severe blow to cervical internal carotid artery (ICA), (5) tearing of the vein or pial vessels, and (6) dissemination of blood from hemorrhagic contusion into subarachnoid space, and (7) no cause or idiopathic. Basal SAH can be confused with aneurysmal bleed and hence needs to be evaluated. Causes of aneurysmal rupture with SAH after trauma can be because of (1) direct injury due to skull base fracture, (2) overstretching or torsion of ICA or vertebral artery, and (3) possible tearing of prominent bony structure like anterior clinoid process or postclinoid process resulting into

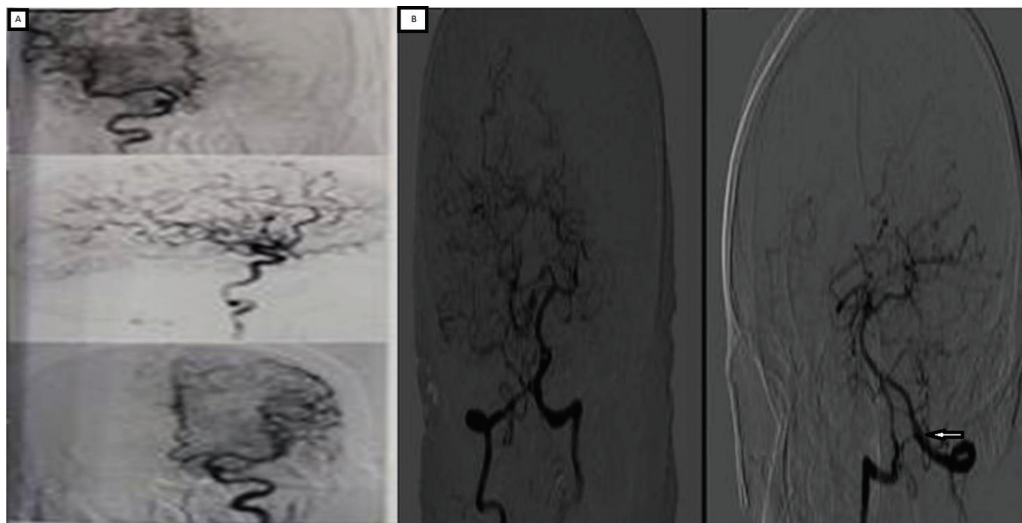


Fig. 2 Digital subtraction angiography4 vessel. (A) Anterior circulation was normal. (B) Posterior circulation also showed vertebral artery on left was normal with normal PICA origin (white arrow).

rupture of aneurysm.⁶ CT brain is the primary investigation done to diagnose SAH and its grading. Different score systems are available like (a) Fischer grading, (b) Morris-Marshall Grading, and (c) Green et al grading. Susceptibility-weighted images (SWIs) MRI is very sensitive in detecting small amount of SAH and in identifying intraventricular blood than CT. But SWI poorly identifies blood in basal cistern.⁷

Mostly traumatic basal SAH may associate with vasospasm, electrolyte imbalance, hormonal imbalance, and long-term sequel as hydrocephalus.

In this case, patient presented with traumatic SAH (Fischer grade III) in all cistern (bilateral sylvian, basal and prepontine, left cerebellopontine angle, and left cerebellomedullary cistern). On further investigation, it was found to have fracture of left transverse process of C1 vertebra. MRI cervical spine was not showing any cord injury or hemorrhage. Traumatic rupture of aneurysm with extensive basal SAH is usually associated with skull base fracture. This case is rare and unique probably first case showing diffuse SAH following trauma to neck. Spread of blood from infratentorial cistern to supratentorial cistern may indicate severity. C1 (Atlas) vertebra has unique anatomy. Its transverse foramen harbor V2 segment of Vertebral artery and vertebral venous plexus along with it. C1 nerve root also travels through foramen. Vertebral artery (V2) segment is closest to C1-C2 joint near inferior facet of C1. It takes loop almost 90-degree medially after its exit from foramen transversarium of C1 vertebra and occupies groove over superior surface of the posterior arch the Atlas (C1). Fracture affecting Atlas (C1) transverse process (foramina) with inward and medial impingement of the bone injuring possibly complex venous channels around vertebral artery may lead to diffuse SAH. DSA brain 4 vessel was suggestive of normal vertebral angiogram with normal PICA on that side. Complex venous channels at skull base can have brisk bleeding following trauma and could be possible cause of diffuse SAH in the absence of major vascular involvement. Traumatic injuries to vertebral artery causing dissection, longitudinal tear, laceration, and pseudoaneurysm; posterior communicating artery; anterior choroidal artery; or

ICA are well described in literature. But C1 transverse foramen fracture without involving vertebral artery on angiogram could be first case to represent rare cause of diffuse basal SAH. Hence, CT craniovertebral junction (CVJ) should be done as an adjunct investigation to rule out bony injury whenever possible in traumatic basal SAH cases.

Conclusion

Injury to complex venous channels along skull base around major vessels could be possible mechanism presenting traumatic basal SAH. CT CVJ or cervical spine should be done in basal traumatic SAH to rule out rare causes like bony fracture.

Conflict of Interest

None declared.

References

- 1 Dewan MC, Rattani A, Gupta S, et al. Estimating the global incidence of traumatic brain injury. *J Neurosurg* 2018;130(04): 1080–1097
- 2 Armin SS, Colohan AR, Zhang JH. Traumatic subarachnoid hemorrhage: our current understanding and its evolution over the past half century. *Neurol Res* 2006;28(04):445–452
- 3 Modi NJ, Agrawal M, Sinha VD. Post-traumatic subarachnoid hemorrhage: a review. *Neurol India* 2016;64(Suppl):S8–S13
- 4 Dowling G, Curry B. Traumatic basal SAH. Report of six cases and review of the literature. *Am J Forensic Med Pathol* 1988;9(01): 23–31
- 5 Wong B, Ong BB, Milne N. The source of hemorrhage in traumatic basal SAH. *J Forensic Leg Med* 2015;1(29):18–23
- 6 Ullman JS, Morgan BC, Eisenberg HM. Traumatic subarachnoid hemorrhage. Chapter 14. In: Bederson JB, ed. *Textbook of Subarachnoid Hemorrhage: Pathophysiology and Management*. Meadows, IL, United States: AANS Publication Committee; 1997: 225–37
- 7 Griswold DP, Fernandez L, Rubiano AM. Traumatic subarachnoid hemorrhage: a scoping review. *J Neurotrauma* 2022;39: (1-2):35–48