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

Akshay Patil, Sandip Iratwar, Ashish Jha

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

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).

Introduction

Traumatic brain injury (TBI) is the most common cause of mortality and morbidity in low- and middle-income countries in adult population. 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. Road traffic accident, fall from height, and violence (assault) mostly contribute to TBI. Traumatic SAH is adverse prognostic factor leading to progressive neurological deterioration 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 the left side of neck and jaw by hard object.

Patient had 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.

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

ISSN 2277-954X.
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.

Discussion

Traumatic basal SAH is well documented in the younger intoxicated males who have been involved in assault to the head, neck, and face. Most common form of the vascular injury is from the intracranial portion of vertebral artery. 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
rupture of aneurysm.\textsuperscript{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.\textsuperscript{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**