# Case Report

# Retroclival epidural hematoma: A rare location of epidural hematoma, case report, and review of literature

# ABSTRACT

Retroclival epidural hematoma in adults is uncommon. Although most cases are associated with craniocervical trauma, other mechanisms have been reported, such as coagulopathy, vascular lesions, and pituitary apoplexy. We report two adults diagnosed with retroclival epidural hematoma. One patient was an 89-year-old male with leukemia and thrombocytopenia who sustained a fall and developed a traumatic retroclival epidural hematoma with brainstem compression; surgery could not be performed due to his clinical condition and he died 5 days later. The other patient was a 78-year-old female with atrial fibrillation who developed a spontaneous retroclival epidural hematoma as a result of warfarin use; she was treated conservatively with anticoagulant reversal and methylprednisolone and was subsequently discharged without neurological deficit. Retroclival hematomas are primarily treated conservatively due to the difficulty of surgical approach. The bleeding mechanism and dural and venous anatomy of this region tend to limit hematoma expansion.

Keywords: Dural venous anatomy, retroclival hematoma, thrombocytopenia, warfarin

# INTRODUCTION

Epidural hematoma in the posterior fossa was first described in 1941 by Coleman and Thompson.<sup>[1]</sup> and the first clival epidural hematoma was reported in 1986 by Orrison et al.<sup>[2]</sup> Posterior fossa epidural hematomas comprise approximately 4% to 12.9% of all epidural hematomas 3-5 and only 1.2%–12.9% of these are retroclival.<sup>[3-5]</sup> Koshy et al. described the disruption of the tectorial membrane as a possible mechanism of retroclival epidural hematoma development.<sup>[6]</sup> These lesions usually occur in the pediatric population due to predisposing anatomical features and typically develop after hyperextension or hyperflexion neck trauma involving the craniocervical junction. The resulting hemorrhage may compress the brainstem, causing loss of consciousness, paresis, and cranial nerve palsies. The sixth cranial nerve is most commonly affected because of its relatively long intracranial course from the pontomedullary junction to the petrous apex and Dorello's canal.<sup>[6]</sup> In this article, we present our experience with two adult cases of retroclival epidural hematoma.

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# **CASE REPORTS**

#### Case 1

A 78-year-old female presented to the emergency department with a 2-day history of weakness in all limbs. One month previously, she had been diagnosed with atrial fibrillation and began warfarin therapy. She had no known history of trauma and no visible external signs of trauma. On admission to the

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emergency department, she was hemodynamically stable and her neurological examination showed a Glasgow coma scale (GCS) score of 15 and quadriparesis without sensory deficit or cranial nerve palsy. Deep tendon reflexes in all four limbs were normoactive; the plantar reflex was mute. Laboratory investigations showed a platelet count of 202,000, prothrombin time of 26.1 s, and international normalized ratio of 2.23. Brain computed tomography (CT) demonstrated a retroclival hyperdense mass lesion compressing the brainstem [Figure 1]. Sagittal images showed that the lesion extended inferiorly from the dorsum sellae through the foramen magnum to the anterior arch of C1. Magnetic resonance imaging (MRI) and CT angiography revealed no other apparent intracranial abnormalities such as occult arteriovenous malformation, meningeal tumor, aneurysm, traumatic arterial dissection, or visible fracture/dislocation of the skull base, craniocervical region, or spine. MRI of the lumbar spine showed a lesion of low signal intensity on T2-weighted images and high signal intensity on T1-weighted images from L4 to S2 causing mass effect on the thecal sac, compatible with epidural hematoma [Figure 1]. Anticoagulation was reversed in the intensive care unit with fresh frozen plasma and Vitamin K was initiated. Atrial fibrillation prophylaxis was changed to subcutaneous enoxaparin. The patient was managed conservatively with oral methylprednisolone. Daily neurological examinations showed progressive clinical improvement. Craniocervical and lumbar CT 5 days after admission showed no progression of the hematoma and no significant increase in ventricular size. The patient was discharged without neurological deficit. Three weeks after discharge, MRI showed spontaneous



Figure 1: (a) Sagittal cranial computed tomography image at admission demonstrates a hyperdense mass posterior to the clivus. (b) Sagittal craniocervical T1-weighted magnetic resonance image shows a retroclival epidural hematoma extending inferiorly. (c) Sagittal lumbar spinal T2-weighted magnetic resonance image shows an epidural hematoma. (d and e) Follow-up magnetic resonance images 3 weeks after discharge show hematoma regression

resolution of the hematoma and she remained free of neurological deficit [Figure 1].

#### Case 2

An 89-year-old male with acute myeloid leukemia was referred by the hematology clinic to the emergency department due to a fall. In the emergency department, he was intubated due to a GCS score of 8. On neurological examination, his eyes were closed and he did not open them upon command. Pupils were equal and reactive. He localized pain with both upper extremities and withdrew from pain with his lower extremities. Deep tendon reflexes in all four limbs were hyperactive; plantar reflexes were flexor on the left and extensor on the right. His platelet count was 11,000. Craniocervical CT revealed a right frontoparietal epidural hematoma and a hyperdense epidural lesion that extended from the clivus inferiorly to the odontoid process and compressed the brainstem [Figure 2]. He was immobilized with a cervical brace and administered methylprednisolone. Despite platelet transfusion, a sufficient platelet count could not be achieved. Surgery to remove the retroclival epidural hematoma could not be performed because of the patient's poor clinical condition, comorbidities, electrolyte imbalances, and low platelet count. He died 5 days after evaluation due to neutropenia-related pneumonia.

## **DISCUSSION**

Retroclival epidural hematoma is most commonly caused by trauma and may present with focal neurological deficit, particularly cranial nerve palsy. Most cases are associated with craniocervical fracture/dislocations or ligamentous injuries. Retroclival epidural hematoma is more common in the pediatric age group due to the anatomical features of the pediatric craniocervical junction: the bones have not yet



Figure 2: Sagittal cranial computed tomography shows a hyperdense lesion dorsal to the basion of the clivus

completed development, the occipital condyles are relatively smaller, and the ligaments of the craniocervical junction are more elastic.<sup>[7]</sup> Craniovertebral stability is not yet complete in children, thus they are more susceptible to flexion-extension injury of this region. Although CT is required for diagnosis, MRI is the best imaging modality to determine the relationship between the craniocervical structures and hematoma. The retroclival venous anatomy is comprised of a wide venous network between the two dural leaves overlying the clivus. Veins of this network connect with diploic veins of the clivus by piercing the external dura and with cisternal veins by piercing the internal dura [Figure 3]. The bilayered dura of the dorsal side of the clivus contains the complex basilar venous plexus and clival diploic veins between the meningeal and periosteal leaves.<sup>[8,9]</sup> The basilar venous plexus joins the inferior petrosal sinuses and the internal vertebral venous plexus during its course along the midline of the clivus and may also connect with the superior petrosal sinuses and cavernous sinuses at its caudal end.<sup>[10,11]</sup> Clival diploic veins connect the posterior intercavernous sinus and basilar plexus to anterior condylar veins. The plexus continues as the cavernous sinuses anterolaterally and basilar plexus inferiorly.<sup>[9]</sup> Bridging veins connect the posterior intercavernous sinus to the anterior pontomesencephalic vein.<sup>[12]</sup>

Based on clival venous anatomy and bleeding mechanisms, retroclival hematomas can be classified as epidural, subdural, or interdural.<sup>[13]</sup> A retrospective study by Krishnan et al. reported that most retroclival hematomas are epidural although some cases are interdural.<sup>[14]</sup> In traumatic clival fractures, bleeding may occur from emissary veins that connect the basilar plexus or the inferior petrosal sinuses to the venous channels located in the inferior surface of the clivus adjacent to the pharynx and cause retroclival epidural hematoma.<sup>[9,14]</sup> Retroclival hematomas may also be spontaneous due to coagulopathy and have been associated with intraventricular and subarachnoid hemorrhage.<sup>[15,16]</sup> Almost all subarachnoid hemorrhages isolated to the anterior brainstem cisterns are angiography-negative,<sup>[17,18]</sup> showing that the venous structures of this region have the potential to bleed spontaneously or after trauma. The location of bleeding in the retroclival region is important with respect to hematoma expansion. Epidural hematomas of the lower clivus are restricted from expanding inferiorly due to the insertion of the tectorial membrane. However, subdural hematomas can extend inferiorly from the foramen magnum into the spinal canal. Venous bleeding in the epidural and interdural areas tends to be more limited.<sup>[19-21]</sup> Interdural bleeding is self-limiting because the blood is trapped between the two leaves of the dura, unlike epidural hematomas caused by arterial bleeding. Tectorial membrane stretching at the

craniocervical junction due to trauma causes interdural vein rupture and bleeding. We hypothesize that craniocervical trauma also causes interdural bleeding from the basilar venous plexus, clival diploic veins, or bridging veins due to detachment of the retroclival dura mater.

Including the two patients presented here, 19 cases of adult retroclival epidural hematoma have been reported [Table 1]. One patient underwent pituitary adenomectomy due to pituitary apoplexy, 2 underwent posterior fossa decompression, and 5 underwent posterior fossa decompression and craniocervical stabilization and fusion. One possible cause of retroclival hematoma after pituitary surgery and apoplexy may be related to the interdural venous connections between the cavernous sinuses, petrosal sinuses, and basilar plexus. Among the five patients who underwent craniocervical stabilization, one died after surgery and 3 had good results; information about the postoperative condition of the other patient was not available. Among the two patients who underwent posterior fossa decompression, 1 had good results, the other one had partial recovery. Among the 11 patients who received conservative treatment, 2 died, three patients experienced partial improvement, and 6 had a good result.<sup>[22-32]</sup> The time of retroclival hematoma resorption can vary by several days. There is no clear treatment that hastens resorption. Steroids could be used as treatment because of their membrane-stabilizing effect, as venous and dural tension is important in the bleeding mechanism.

# CONCLUSION

Retroclival epidural hematomas are quite rare in adults. Investigation for a causative lesion such as arteriovenous malformation, aneurysm, tumor, or arterial dissection should be initiated after diagnosis. After trauma, craniocervical



Figure 3: Illustration showing dural venous structures and connections between the two layers of dura at the clivus

Patient number	Literature	Age	Sex	Mechanism	Association with cranioverebral injury	Clinical presentation	Treatment	Outcome
1	Datar et al.[18]	75	Μ	Trauma	Type II fracture of odontoid	Headache, neck pain, stiffnes	Surgical stabilization of odontoid	Died
2	Perez et al.[19]	68	Μ	Trauma	Odontoid base fracture	Cardiorespiratory arrest	Conservative	Died
3	Calli <i>et al</i> . <sup>[20]</sup>	42	Μ	Postoperative posterior fossa decompression	No	Not available	Posterior fossa decompression	Good recovery
4	Goodman <sup>[21]</sup>	62	Μ	Pituitary apoplexy	No	Nausea, vomiting, headache	Transsphenoidal resection of a hemorrhagic pituitary tumor	Good recovery
5	Cho et al.[22]	36	Μ	Spontaneous	No	Neckpain, headache	Conservative	Good recovery
6	Oliviero <i>et al.</i> <sup>[23]</sup>	66	F	Trauma	Whiplash injury	Severe tetraparesis, respiratory muscles weakness, dysarthria, dysphagia	Conservative	Partial recovery, in the context of pontomedullary infarction
7	Ratilal <i>et al</i> . <sup>[4]</sup>	26	F	Trauma	Cervical hiperextension	Bilateral abducens palsy, bilateral trigeminal numbness, left hypoglossal palsy	Conservative	Recovery with mild cranial nerve palsies
8	Khan <i>et al</i> . <sup>[24]</sup>	19	Μ	Trauma	Transverse clivus fracture	Multiple cranial nerve palsies	Conservative	Recovery with cranial nerve palsies
9	Fuentes et al. <sup>[25]</sup>	47	F	Trauma	Bilateral condyle fracture	Teatraparesis, left abducens palsy	Posterior fossa decompression	Mild hemiparesis, able to walk
10	Piccirilli <i>et al</i> . <sup>[26]</sup>	76	Μ	Spontaneous	Clival fracture	Loss of consciousness, headache, nausea, vomiting	Conservative	Good recovery
11	Tomaras et al.[27]	36	Μ	Trauma	No	Neckpain and headache	Conservative	Good recovery
12	Garton <i>et al</i> . <sup>[28]</sup>	20	Μ	Trauma	Atlanto-occipital dislocation with ligamentous injuries	Bilateral 6 <sup>th</sup> CN deficit, incomplete quadriplegia	Occiput-to-C3 fusion	Good recovery
13		38	F	Trauma	Displaced anterior occipital fracture	Bilateral 6 <sup>th</sup> CN deficit	Occiput-to-C2 fusion	Good recovery
14		44	F	Trauma	Occipital condyle– C1 distraction and subluxation, rotary subluxation of C1-C2, C5– C6 fracture and subluxation	Loss of abduction of the right eye, mild right hemiparesis	Occiput-to-C2 instrumented fusion	Neurological improvement
15		67	F	Not available	Sublaxation of C1-C2 with ligamentous injuries	Bilateral 6 <sup>th</sup> CN deficit, right 3 <sup>rd</sup> CN deficit	Fusion of C1 to C2 with C1–C2 transarticular screw fixation	Not available
16	lzumida <i>et al</i> . <sup>[29]</sup>	64	Μ	Trauma	No	No neurological deficit	Conservative	Good recovery
17	Pineda <i>et al.</i> <sup>[30]</sup>	35	Μ	Trauma	Displaced right frontotemporal fracture and fractures of the right orbital floor	GCS 13	Conservative	Good recovery
18	Case 1	78	F	Spontaneous	No	Quadriparesis	Conservative	Good recovery
19	Case 2	89	Μ	Trauma	No	GCS 8	Conservative	Died

#### Table 1: Review of the available literature

fracture/dislocations and ligamentous injuries should not be overlooked. Conservative treatment consisting of cervical immobilization, methylprednisolone, and close follow-up imaging is a more suitable management option than surgery because of the anatomical complexity of the craniocervical junction and the difficulty of surgical approach. However, surgery may be required in the presence of progressive neurological deterioration or craniocervical instability.

#### **Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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#### **Conflicts of interest**

There are no conflicts of interest.

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