

## Postoperative epidural hematoma contributes to delayed upper cord tethering after decompression of Chiari malformation type I

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Received: 31 March 14 Accepted: 18 June 14 Published: 21 August 14

### This article may be cited as:

Lopez-Gonzalez A, Plaza E, Marquez-Rivas FJ. Postoperative epidural hematoma contributes to delayed upper cord tethering after decompression of Chiari malformation type I. *Surg Neurol Int* 2014;5:S278-81.

Available FREE in open access from: <http://www.surgicalneurologyint.com/text.asp?2014/5/5/278/139384>

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### Abstract

**Background:** Symptomatic arachnoiditis after posterior fossa surgical procedures such as decompression of Chiari malformation is a possible complication. Clinical presentation is generally insidious and delayed by months or years. It causes disturbances in the normal flow of cerebrospinal fluid and enlargement of a syrinx cavity in the upper spinal cord. Surgical de-tethering has favorable results with progressive collapse of the syrinx and relief of the associated symptoms.

**Case Description:** A 30-year-old male with Chiari malformation type I was treated by performing posterior fossa bone decompression, dura opening and closure with a suturable bovine pericardium dural graft. Postoperative period was uneventful until the fifth day in which the patient suffered intense headache and progressive loose of consciousness caused by an acute posterior fossa epidural hematoma. It was quickly removed with complete clinical recovering. One year later, the patient experienced progressive worsened of his symptoms. Upper spinal cord tethering was diagnosed and a new surgery for debridement was required.

**Conclusions:** The epidural hematoma compressing the dural graft against the neural structures contributes to the upper spinal cord tethering and represents a nondescribed cause of postoperative fibrosis, adhesion formation, and subsequent recurrent hindbrain compression.

**Key Words:** Arachnoiditis, arnold-chiary malformation, dural graft, posterior fossa, tethering

### Access this article online

**Website:**

[www.surgicalneurologyint.com](http://www.surgicalneurologyint.com)

**DOI:**

10.4103/2152-7806.139384

**Quick Response Code:**



## INTRODUCTION

Although a variety of surgical procedures exist for the treatment of Chiari malformation, craniocervical decompression followed by duraplasty is probably the procedure of choice. Chiari I surgery failure with cervical spinal cord tethering has been largely described.<sup>[6-8]</sup> It consists of worsening of symptoms with radiological findings that include an impacted cisterna magna without cerebrospinal fluid (CSF) flow in the dorsal

aspect of the cerebellum and upper spinal cord, and the development of a medullar syrinx that tends to be progressively enlarged.

This surgical failure is related with several factors including inadequate bone decompression, type of graft, craniocervical instability, or others like shunt malfunction. Nevertheless, epidural hematoma complicating surgery has not yet been described as a cause of Chiari I surgery failure with cervical spinal cord tethering.

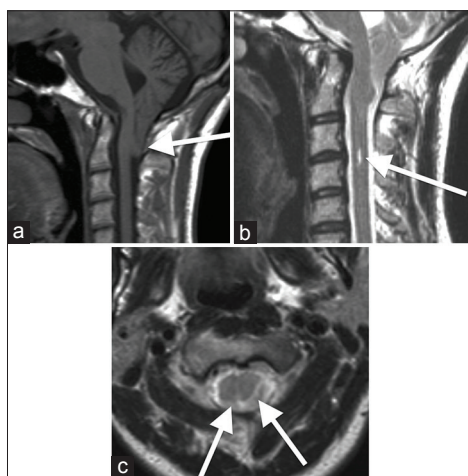
We point to the relevance of postoperative symptomatic or nonsymptomatic epidural collections compressing dural graft against cerebellum and upper spinal cord as an initial cause of surgical failure and tethering.

## CASE REPORT

A 30-year-old male had a history of tensional headache and weakness of the upper limbs for one year prior to the diagnosis of a Chiari malformation type I. A magnetic resonance imaging (MRI) showed mild basilar impression, cerebellar tonsils at C2 level, a small syrinx at C3-C4, and no hydrocephalus [Figure 1a and b].

Accordingly, a standard posterior fossa decompression was performed. It consisted of a suboccipital craniectomy, C1-C2 laminectomy, dura opening, and a lyophilized bovine pericardium duraplasty. The immediate postoperative course was uneventful with a routine computed tomography (CT) scan performed 48 h after surgery demonstrating the absence of complications [Figure 2a]. But, on the fifth day postoperative, the patient suddenly developed severe headache and right hemiparesis. Emergent CT scan revealed a posterior fossa epidural hematoma compressing cerebellum, upper spinal cord, and the fourth ventricle [Figure 2b]. Emergent evacuation of the hematoma with complete clinical recovery was achieved.

One year later, the patient experienced progressive headache and global hyperreflexia with decreased pinprick and light touch sensations on the upper limbs. The MRI revealed adequate bone decompression without ventricular enlargement. However, the absence of CSF signal dorsal to the cerebellar hemispheres, vermis, tonsils, and spinal cord at C1 level was noted.



**Figure 1:** (a) Sagittal T1 MRI shows descended tonsils at C2 level and occupying posterior aspect of the foramen magnum. CSF flow is blocked at this point (arrow). (b) Sagittal T2 MRI shows a small syrinx at C3-C4 level (arrow). (c) Axial T2 MRI shows that the posterior aspect of the foramen magnum is occupied by the descended tonsils (arrows)

A remarkable enlargement of the previous syrinx cavity was also demonstrated at C3-C4 level [Figure 3].

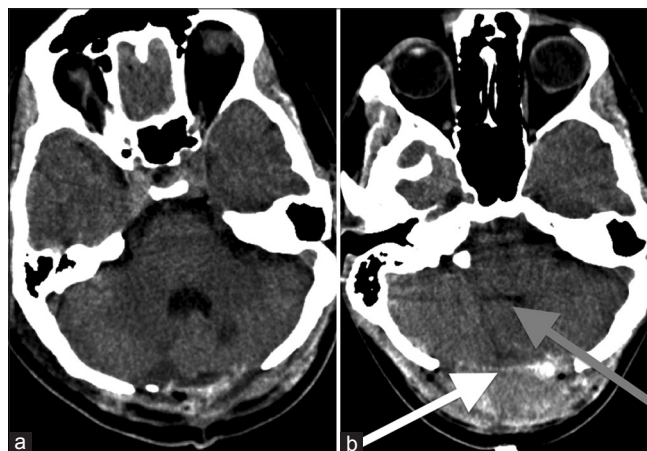
Surgical treatment was decided. Severe arachnoid scars that tethered the cerebellar hemispheres and upper spinal cord to the dura and dural graft were sharply dissected. De-tethering of the spinal cord and aperture of the obex were achieved and the normal CSF flow was re-established. A new duraplasty using lyophilized bovine pericardium dural graft was performed and it was tacked up to the adjacent pericranium in order to keep the graft away from the cerebellar hemispheres and upper spinal cord.

The patient's neurological status recovered only few days after surgery. The postoperative MRI showed decreasing of the syrinx and patency of the subarachnoidal space [Figure 4]. He did not present any complication during the long-term follow-up.

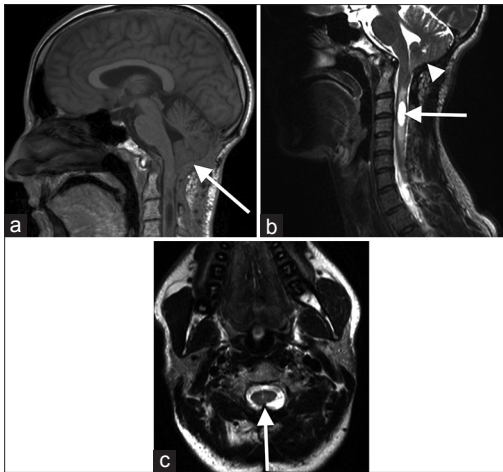
## DISCUSSION

Tethering of spinal cord in lumbar and sacral regions is a more frequent entity than in upper spinal cord. It is associated with congenital anomalies with typically stable symptoms for many years and microsurgical de-tethering is traditionally described as the standard treatment. However, cervical spine cord tethering is infrequent in adults; it usually affects young people and it can be caused by congenital anomalies, fibrous adhesions, and trauma.<sup>[3]</sup> Cervical cord tethering secondary to a previous intradural surgery is also possible.<sup>[7,8]</sup>

There are controversies about the optimal surgical technique for Chiari malformation type I treatment. On the one hand, maintaining dura mater integrity prevents CSF leak and adhesions. On the other, opening of the arachnoid layer can assure the success of decompression, but it facilitates the direct contact between the pia mater



**Figure 2:** (a) Postoperative routine CT scan (48 h postoperative) shows the absence of complications. (b) Urgent cranial CT scan on the fifth postoperative day shows a posterior fossa epidural hematoma (white arrow) causing compression of cerebellar hemispheres, vermis, and fourth ventricle (gray arrow)



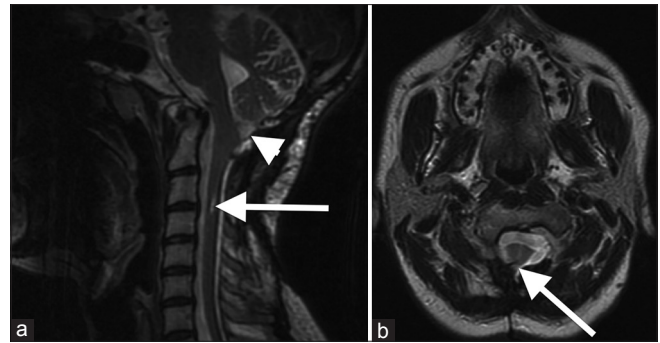
**Figure 3: (a)** One year after initial surgical procedure, sagittal T1 MRI shows the patent bone decompression of the posterior fossa and fibrous tissue (arrow). **(b)** Sagittal T2 MRI: shows a great syrinx cavity at C3-C4 levels (white arrow) and the absence of CSF signal around the cerebellar hemispheres (arrowhead). **(c)** Axial T2 MRI at the level coincident with obex: it shows the intense adherence of arachnoid tissue (arrow), which produces a blockage of CSF flow

with dura mater and dural graft. This and specially any blood spilled intradurally are facts that may promote the scars. A better knowledge of foramen magnum CSF dynamics and the use of intraoperative sonography after posterior fossa bone decompression can aid in the decision whether to open or not to open the duramater.<sup>[4]</sup>

When a duraplasty is considered, the type of dural graft used can also play a role in the outcome. The ideal graft should be nonimmunogenic, allow a watertight closure, and avoid cortical adhesions. The most usual materials for dural grafts are: Autologous pericranium, bovine pericardium, lyophilized cadaveric dura, synthetic products, ligamentum nuchae, and fascia lata. Pericranium and fascia offer a nonimmunogenic and inexpensive dural substitute.<sup>[1]</sup> When available for harvest, autologous pericranium is associated with better rates of aseptic meningitis, wound infections, and pseudomeningocele formation compared with allografts. Aseptic meningitis, wound infections, and pseudomeningoceles are less frequent using autologous grafts, although the rates of incisional CSF leakage and reoperation are higher.<sup>[1]</sup> For authors like Hopkins,<sup>[2]</sup> synthetic grafts seem to maintain hindbrain space, accelerate syringomyelia improvement, and decrease treatment failure.

There are many causes responsible for the recurrence of the Chiari symptoms after surgical treatment: Instability at the craniocervical junction, recurrent syringomyelia, shunt malfunction, or regeneration of the foramen magnum. But, according to the literature reviewed, postoperative epidural hematoma has not been yet published as a contributor to recurrent Chiari symptoms.

The recurrence of Chiari symptoms is usually developed long time after the procedure and they are related to



**Figure 4: (a)** The syrinx cavity has become smaller (white arrow) and CSF signal appears in the posterior aspect of cerebellum-medulla (arrowhead). **(b)** The dural graft has been detached from the parenchymal surface (arrow)

cervical myelopathy: Neck and upper extremities pain, Lhermitte's sign, weakness and numbness in extremities. In cases of upper spinal cord tethering, delayed syrinx cavity may appear on various levels.<sup>[7]</sup> The pathophysiological mechanisms proposed are complex. Blockage at obex level causing progressive accumulation of CSF in the ependymal canal is one possibility. Dorsal displacement of spinal cord with disturbance of the normal more straight flow of the CSF is another.<sup>[5]</sup> Further studies using new flow techniques in magnetic resonance could help to elucidate it. Finally, neck movements with a relatively fixed spinal cord could result in mechanical distortion and intermittent or progressive spinal cord ischemia.<sup>[7]</sup>

Nonautologous grafts, like the one we used, have been associated with postoperative fibrosis, adhesion formation, and subsequent recurrent hindbrain compression initiated by the graft itself.<sup>[6]</sup> Nevertheless, in our clinical case, the epidural hematoma pulled the dural graft up against the upper spinal cord and cerebellar pia mater and this contributed to initiate the massive arachnoid scar reaction observed at revision surgery. It is remarkable that, despite the dural graft used in the revision surgery for debridement was of the same kind that the one used in the initial decompressive surgery, no late upper spinal cord tethering was achieved in postoperative radiological studies.

Asymptomatic or symptomatic postsurgical epidural collections after posterior fossa decompression for Chiari malformation could play an important role in the development of late complications such as recurrence of Chiari symptoms, tethering, and syringomyelia. When the diagnosis of postoperative tethering of the upper spinal cord is established, surgical debridement and de-tethering has good clinical and radiological results arresting the neurological deficits in all the cases published.<sup>[7,8]</sup>

## CONCLUSIONS

While epidural hematomas have not been so far reported as a cause of Chiari I surgery failure with cervical spinal cord

tethering, similar anatomical effects can be found whenever a large pseudomeningocele creates a sizeable mass effect on the dura, thus displacing the graft against the cerebellum and the cervico-medullary junction. This phenomenon may promote arachnoid scars between the dural graft and the upper spinal cord and may derivate in later upper spinal cord tethering, hindbrain compression, and Chiari I surgery failure. Avoiding postoperative epidural collections can help to prevent later recurrence of the symptoms.

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