Acta Neurochir (Wien) (1997) 139: 112–119

Acta Neurochirurgica

Duraplasty with Freeze-Dried Cadaveric Dura versus Occipital Pericranium for Chiari Type I Malformation: Comparative Study

V. Vanaclocha[1] and **N. Saiz-Sapena**[2]

[1] Division of Neurosurgery and [2] Department of Anaesthesiology, Clinica Universitaria, University of Navarra, Pamplona, Navarra, Spain

Summary

During the period from October 1, 1989 to October 1, 1995 a total of 26 cases of Chiari type I malformation not associated with syringomyelia were attended in our Hospital. All patients underwent cranio-cervical decompression, with occipital craniectomy and removal of the posterior arch of C_1. In 3/26 (11.5%) cases an additional C_2 laminectomy had to be performed and in 1/26 (3.8%) case the C_3 laminae were also removed. A first group of 13 patients underwent dural repair with freeze-dried cadaveric dura sutured with continuous 4-0 Vicryl® running stitches, reinforced with fibrin sealant (Tissucol®). A second group of 13 patients underwent duraplasty with autogenous occipital pericranium also sutured with continuous 4-0 Vycril® but no fibrin sealant at all was added.

In the first group, in which freeze-dried cadaveric dura plus Tissucol® was used, there were 2/13 (15.3%) cases of CSF leak, requiring some additional skin stitches to stop the leak. In 5/13 (38.4%) cases there were notorious subcutaneous CSF accumulations that required repeated punctures plus compressive bandage. In 6/13 (46.1%) pseudomeningoceles appeared that took a year to clear completely. In the 13 patients who underwent dural repair with autogenous occipital pericranium watertight closure was achieved with sutures only, no fibrin sealant was added at all. Neither CSF leaks through the wound nor subcutaneous CSF accumulations were noted.

We conclude that, in our hands, autologous pericranium taken from the occipital area, gives better results than freeze-dried cadaveric dura mater in duraplasty for surgical repair of Chiari type I malformation.

Keywords: Chiari I malformation; cranio-cervical decompression; freeze-dried cadaveric dura mater; dural repair; dural graft.

Introduction

Chiari type I malformation consists of a caudal displacement of the cerebellar tonsils through the foramen magnum into the cervical spinal canal without descent of the brainstem. Its origin is still under debate. On the one hand it has been shown that the posterior fossa is smaller and shallower in these patients than in normal individuals [42, 62], suggesting that a small posterior fossa caused by an underdeveloped occipital bone is the primary factor [58, 62]. There seems to be a positive correlation between the posterior fossa size and the degree of the cerebellar ectopia, although a small posterior cranial fossa *per se* has no clinical significance as it can be a normal variant, but remains the primary developmental anomaly [58]. On the other hand Marin-Padilla [27] reproduced the malformation in golden hamsters by administering high doses of vitamin A during pregnancy. The basicondrocranium was abnormally short and resulted in small posterior fossae [27–30]. The postnatal growth burst of the cerebellum took place in an underdeveloped posterior fossa, and a plastic deformation of the nervous tissue to adapt to a small posterior fossa would cause the tonsils to displace below the foramen magnum [27–30].

Chiari type I malformation is commonly associated with suboccipital-occipital headache which is aggravated by Valsalva's manoeuvres, straining, cough, sneezing, laughing or postural changes [3, 14, 21, 25, 40, 44, 46–48, 57, 61]. Only the degree of tonsilar herniation significantly correlated with the presence and intensity of this pain [46, 58] suggesting that the compression of the posterior roots of C_1 and C_2 may be the underlying mechanism in the pathogenesis of this headache [2, 15, 41, 44, 57] that is usually relieved by suboccipital decompressive craniectomy [2, 40, 44, 46, 48, 57, 61].

Since the basic problem of the Chiari type I malformation and its associated headache is a small posterior fossa [42, 58, 62], its treatment must be direct-

ed to increase its size. In most cases bone removal is not enough. Dural enlargement by means of a graft is also required [7, 8]. Many materials have been used to graft the dura mater, the most commonly used being freeze-dried cadaveric human dura. We started by using this grafting material to repair cases of Chiari type I malformation. Unfortunately we had some problems with CSF leaks, pseudo-meningoceles and subcutaneous fluid accumulations. Then we decided to change to autologous material to improve the chances of healing and reduce the complication rate, using autologous occipital pericranium. We did the same number of cases as previously done with freeze-dried cadaveric dura mater and then we compared both methods in relation to local healing and CSF problems as well as the incidence and duration of postoperative headaches.

Patients and Methods

Patients

Between October 1989 and October 1995, twenty six patients with Chiari I malformation with no underlying syringomyelia were attended to our service. Age distribution ranged from 19 to 38 years (28.5 ± 9.5 years) (mean ± sd). Male to female ratio was 2/1.8.

Pre-operative duration of symptoms and signs lasted from 13 months to 6 years (3.2 ± 2.8 years) (mean ± sd). The age of onset ranged from 17 to 36 years (26 ± 9.6 years) (mean ± sd). Nuchal headache was the reason for patients seeking medical consultation in 23/26 (88.4%), and to unsteadiness on walking in 3/26 (11.6%).

Nuchal pain was most characteristically pounding, usually posterior and bilateral. Typically it was provoked by coughing, sneezing or any strain, being present in all patients with different severity. Its distribution and intensity was graded by the patients themselves both pre- and post-operatively.

Nystagmus was a common finding, most often horizontal (88.4%), but at times also rotationary (7.6%) or even retropulsating (15.3%). Some patients complained of a feeling that the floor seemed to move up and down on walking (30.7%).

Unsteady gait was very common (73%), with positive Romberg (34%) and positive tandem gait (61.5%). Dysmetria, adiadochokinesis and Stewart-Holmes sign were not seen.

Often there was an absent gag reflex on one (87%) or both sides (30%).

Patients were re-evaluated at 1, 6 and 12 months after surgery. They were asked about the incidence, frequency, localisation and intensity of headaches as well as any other sign that was present before the surgical procedure or that might have appeared afterwards. Clinical examinations, grading and MRI examinations were repeated at every follow-up visit. Only patients with follow-up of at least 6 months were included in this study.

Diagnostic Evaluation

Diagnosis of Chiari type I malformation was always established by magnetic resonance imaging. The level of the cerebellar tonsils was assessed on MRI sagittal T_1-weighted images with the head in neutral position, and the distance of the most inferior part that descended below the foramen magnum was measured in millimetres. Following Barkowich *et al.* [6] Chiari type I malformation was defined as tonsilar herniation below the foramen magnum of at least 3 mm on the sagittal T_1-weighted MR imaging studies.

On every follow-up visit MR imaging studies were performed in order to analyse the position of the cerebellar tonsils, the size of the new cisterna magna and the existence or not of pseudo-meningoceles and/or subcutaneous fluid accumulations.

Operative Intervention

Cranio-cervical decompression was performed in all patients, consisting of sub-occipital craniectomy and removal of the posterior arch of C_1. In 3/26 (11.5%) cases an additional C_2 laminectomy had to be performed, and in 1/26 (3.8%) cases the C_3 laminae were also removed. The craniectomy included the hole posterior rim of the foramen magnum and extended superiorly for about 3–4 cm. It had a trapezoidal shape, in the sense that superiorly was slightly bigger than at the foramen magnum. The idea was to enlarge the size of the posterior fossa and not only to decompress the tonsils. Then the dura mater was opened in a "Y" shape and the tonsils identified. In order to open the vallecula and provide an adequate CSF IV ventricle outlet, the arachnoid was dissected and the tonsils freed from each other and stitched laterally by means of four 6-0 Prolene® stitches. In order to create a new cisterna magna of adequate size, dural repair was undertaken by means of a redundant dural graft.

A first group of 13 patients underwent dural repair with freeze-dried cadaveric dura sutured with continuous 4-0 Vycril® running stitches, reinforced with fibrin sealant (Tissucol®). A second group of 13 patients underwent duraplasty with autogenous occipital pericranium also sutured with continuous 4-0 Vycril® but no fibrin sealant was added at all.

In any case the dura and its grafting material were hitched to the remaining occipital bone and to the muscle layers, keeping it away from any neural tissue, and thus preventing adhesions that would result in obliteration of the newly formed cisterna magna.

Results

The postoperative follow-up period ranged from 6 to 58 months (27 ± 21.7 months) (mean ± sd). There were neither mortalities nor postoperative complications, save those related to the CSF leaks.

In the group in which freeze-dried cadaveric dura plus Tissucol® was used there were 2/13 (15.3%) cases of CSF leak, who required some additional skin stitches to stop the leak. Once the leak stopped, the subcutaneous fluid accumulation required compressive bandage and repeated lumbar punctures. In another 5/13 (38.4%) cases there were notorious subcutaneous CSF accumulations without CSF leak. They also required repeated punctures plus compressive bandages. All this problems delayed postoperative discharge from hospital (12 ± 4.3 days) (mean ± sd). Only 5/13 (38.4%) healed without any problem. In 6/13 (46.1%) pseudomeningoceles (extradural cavity below the muscles lined by meso-

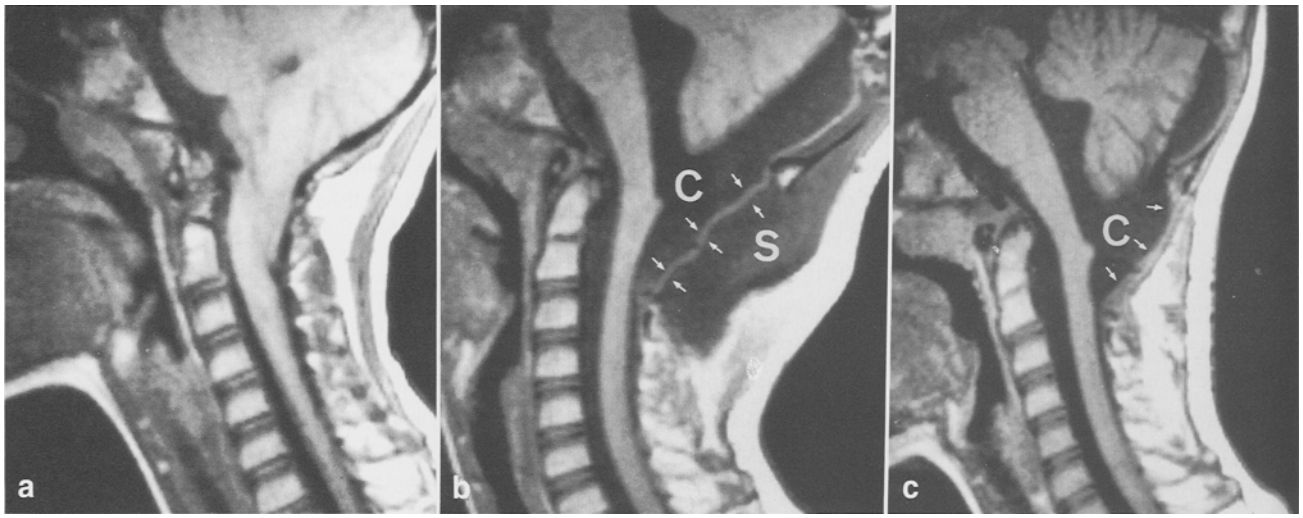

Fig. 1. (a) Pre-operative sagittal T_1 MR imaging study of a patient with Chiari type I malformation. (b) Post-operative sagittal T_1 MR imaging study of the same patient shown in (a). The dural repair was performed with a cadaveric dura mater graft. There is a big subcutaneous fluid accumulation (*S*). New cisterna magna (*C*). The arrows point at the cadaveric dura mater dural graft. (c) One year post-operative sagittal T_1 MR imaging study of the same patient shown in (b). The subcutaneous fluid accumulation has gone. New cisterna magna (*C*). The arrows point at the cadaveric dura mater dural graft

thelium) did finally appear, which took a year to clear completely. Of these 4/6 (66.6%) had undergone CSF leak and/or subcutaneous fluid accumulation, but in 2/6 (33.4%) no problem was detected until 1 month follow-up MRI studies were performed. In summary in 9/13 (69.2%) there were complications. Among the 13 patients who underwent dural repair with autogenous occipital pericranium watertight closure was achieved with sutures only, even though no fibrin sealant at all was added. Neither CSF leaks through the wound nor subcutaneous CSF accumulations were noted. That lead to a shorter postoperative stay (7 ± 2.1 days) (mean ± sd).

Two cases of aseptic meningitis were seen among those receiving freeze-dried cadaveric dural grafts, but none among those in whom autologous pericranium was used.

Nuchal headache disappeared just after the surgical procedure in all previously affected patients. Immediately after cranio-cervical decompression the patients complained of generalised headache on upright posture relieved on decubitus, that lasted 3.3 ± 1.7 days (mean ± sd) for those with pericranium and 17.3 ± 5.4 days (mean ± sd) for those with freeze-dried cadaveric dural graft.

Unsteadiness disappeared completely in 17/18 (94%) of the patients previously affected. One additional patient (1/18, 6%) improved markedly but still complained of some residual problems with balance.

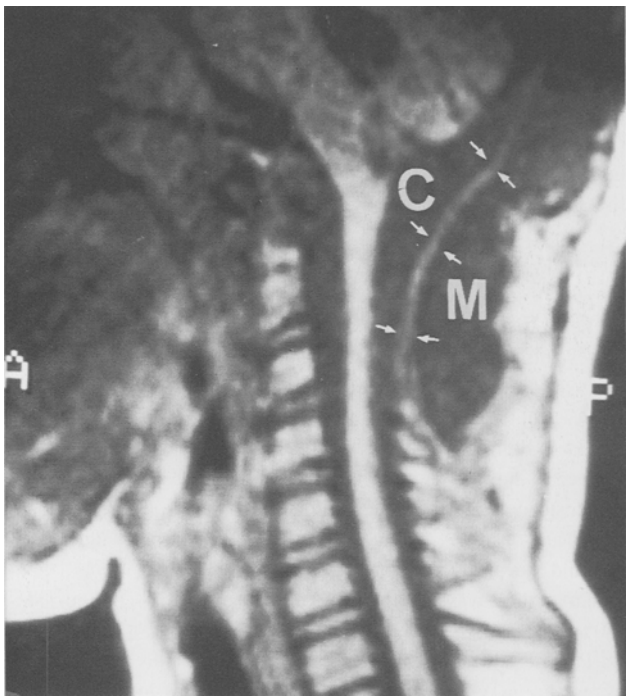

Fig. 2. Pseudomeningocele (*M*) in a patient with freeze-dried dural graft. New cisterna magna (*C*). The arrows point at the cadaveric dural graft

The absent gag reflex reappeared in 75% of those patients in whom it was previously absent.

Clinical improvements happened in the first three months after the surgical procedure, remaining stable ever since.

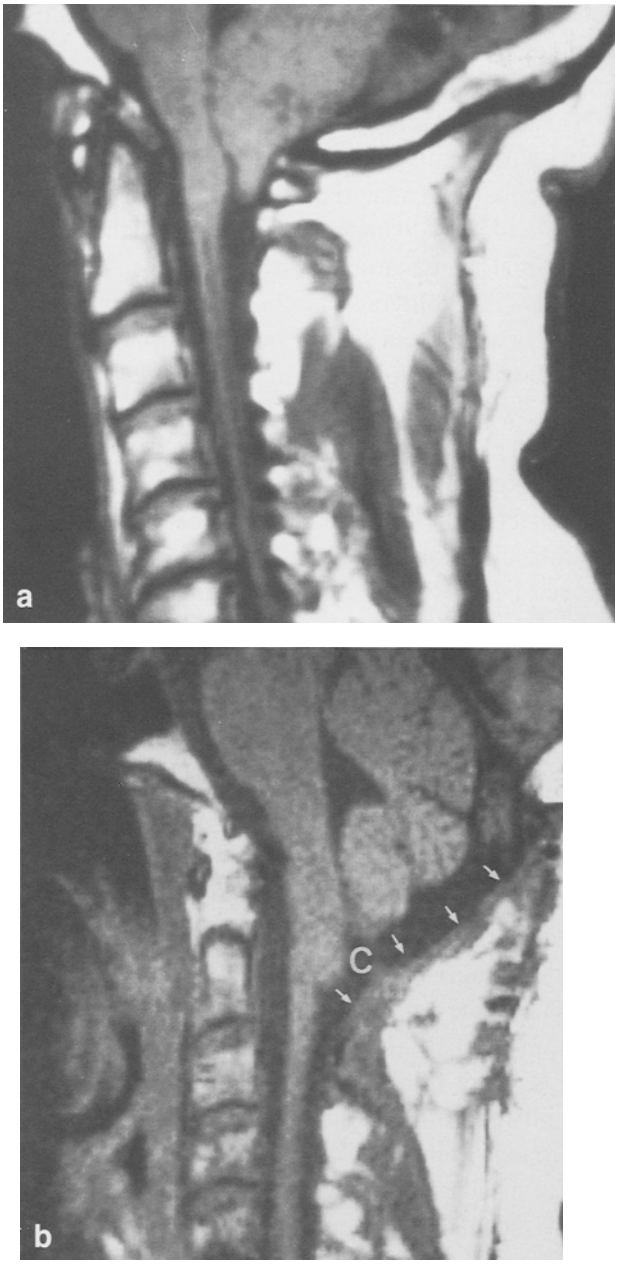

Fig. 3. (a) Pre-operative sagittal T_1 MR imaging study of a patient with Chiari type I malformation. (b) Post-operative sagittal T_1 MR imaging study of the same patient shown in (a). The dural repair was performed with periosteum graft. There are neither subcutaneous fluid accumulations nor pseudomeningoceles. New cisterna magna (*C*). The arrows point at the periosteum dural graft

Postoperative MRI Changes

In all the cases post-operative MR imaging studies showed the formation of an artificial new cisterna magna of big size.

In those receiving freeze-dried cadaveric dura mater, subcutaneous accumulation of fluid was present in all of them at 1 and 6 months follow-up visits, disappearing completely at 1 year (Fig. 1). In 5/13 (38.4%) cases there were notorious subcutaneous CSF accumulations that required repeated punctures plus compressive bandaging. In 6/13 (46.1%) pseudomeningoceles appeared, inducing local swelling and unpleasant feelings for the patient when the area was touched (Fig. 2), which took a year to clear completely.

In those receiving autologous pericranium neither subcutaneous fluid accumulations nor pseudo-meningoceles were seen at all (Fig. 3).

We were also concerned about the possibility that the grafts would stick to the underlying neural tissue, but on repeated follow-up MR imaging studies we were able to reveal that it was not the case in our patients (Figs. 1, 2, and 3).

Discussion

The criteria for an optimal dura mater graft are: no inflammatory reaction against the leptomeninges and the cortex; induce no adhesions to the brain tissue; water and liquor tightness with adequate protection of the cerebrum against external bacteria; availability; durable; flexible; easily prepared; inexpensive; easily sterilised and handled; easily shaped; and known to be chemically inert and nontoxic [9, 19]. Ideally, the material should disappear completely and be replaced by tissues similar to the dura mater [53], providing immediate restitution of a membranous covering for the brain without inducing any adverse reaction in the host or provoking adhesions to the underlying neural tissue. Abbe in 1896 [1] first used rubber as a dural substitute, and ever since a variety of substances, both organic and inorganic, have been used. These include metal foils (gold and silver), aminoplastin, vycril and/or collagen, fibrin film, polyester mesh, silastic membranes, Nylon, Perlon, Teflon, Goretex, and porcine biomembranes, but an entirely satisfactory material has not been found yet [18, 19, 24].

The substance that is currently most widely used is cadaveric human dura mater, introduced in 1955 by Sewell *et al.* [55], and first reported in humans by Campbell *et al.* in 1958 [11]. This material is deantigenised, de-enzymatised and free of pyrogen, but unfortunately it has been putatively associated with Creutzfeldt-Jakob disease [12, 17, 23, 31–33, 36, 42, 49, 59, 60, 65, 66]. The transmissible agent responsible for Creutzfeldt-Jakob disease is resistant to inactivation by boiling, 10% formaldehyde and ultraviolet or ionising radiation, but it can be inactivated by autoclaving at 134° C for 18 minutes, or by immersion in 1 molar NaOH for one hour. The latter treatment has

been incorporated into the manufacture of commercial cadaveric freeze-dried dura since 1987, but unfortunately some cases have been reported afterwards [23, 31–33, 36, 59]. If one considers the incidence among all the grafted patients, it is really low, but those patients that get infected die irretrievably.

Meningeal reactions after the use of freeze-dried cadaveric dura mater are not uncommon [51, 52]. We have also had two cases among those receiving freeze-dried cadaveric dura mater but none in the pericranium group. In itself it is not a terrible problem but it complicates the postoperative course of the patient prolonging the hospital stay and thus the costs of treatment.

There is a variety of cadaveric dura mater that is not processed by freeze-drying, but rather dehydrated by immersion at 37° C in acetone, hydrogen peroxide, and sodium hydroxide. Afterwards it is thoroughly rinsed with sterilised water, aiming to remove all residue of the chemical agents, and finally sterilised by means of gamma-irradiation. This process is thought to result in less tissue damage than the process of freeze drying, but might lead to a more irritating dural graft [5]. In fact it has been associated in some cases with an immune-type reaction with meningeal signs and cerebrospinal fluid eosinophilia. Although the patients improved temporarily during corticosteroid therapy, surgical removal of the grafts was finally required [5].

Although not common, incompatibility of the implant, resulting in rejection by the body, can also occur [19].

Others have also found a high incidence of pseudomeningoceles when freeze-dried cadaveric dura mater is used to repair Chiari type I malformations [35, 52]. That is also our experience. That might be related to the fact that this material is dead tissue. On reoperation (for other pathologies besides Chiari type I malformation) the grafted dura mater often looks atrophic and fragile.

Collagen products, when used as dural substitutes, tend to promote inflammatory response [22, 24, 39] and do not seem a big advance against freeze-dried cadaveric dura mater except that no cases of Creutzfeldt-Jakob disease have been reported yet. Unfortunately the healing problems are the same as those expected for the cadaveric dura mater that it is no more than an anarchic array of collagen fibres.

The plastic materials (i. e., Nylon, Teflon or Goretex) are neither transformed nor vitalised, but merely ensheathed by connective tissue [19]. The interface between the recipient dura mater and the graft is usually not watertight thus inducing easy CSF leaks, infections and subcutaneous fluid accumulations.

The message arising from all these problems associated with the use of cadaveric dura mater is clear: Autologous graft material should be used whenever possible [45, 64]. Unfortunately some of the autologous dural grafts are not in themselves devoid of problems. Autologous fascia lata [7] entails an unsightly and sore new scar near the hip, and we are not in favour of it, specially in young female patients. Autologous nuchal ligament, that is known to have a very high elasting content [38] has also been used, but that weakens the ligamentous structures of the posterior aspect of the neck, and besides that the amount of grafting material is limited. Splenius capitis muscle flap has been used by some others and seems to be of special value in the management of recurrent CSF leaks complicating suboccipital craniectomy for the treatment of Chiari type I malformation [16]. It can not be recommended as a regular procedure as it entails an extensive local tissue damage as well as dangerous fibrosis of the newly formed cisterna magna with adherences to underlying neural tissues. In our hands the best method by far is the use of a large piece of pericranium obtained from the occipital area, which is exposed anyway during the surgical procedure. It does not need a new scar as fascia lata grafts do, and has the big advantage that with a running suture can be made absolutely watertight, thus avoiding the need for fibrin sealant. This reduces the costs of the procedure as well as the chances of disease transmission. Being watertight the incidence of postoperative headaches on up-right posture is significantly reduced and no pseudo-meningoceles at all are seen. In order to prevent the periosteum patch to adhere to neural tissue and have a good new cisterna magna, the patch is hitched to the remaining occipital bone and to the muscle layers.

Nuchal headache associated with Chiari type I malformation usually disappears after cranio-cervical decompression no matter what method used to repair the dural repair [15, 40, 44, 46, 48, 57, 61]. Nevertheless post-operative headache on upright posture can be a troublesome problem, specially when non-alive material is used to perform the dural patch. In the first group of patients of our series, in whom freeze-dried cadaveric dura was used, that was a nuisance which prolonged unnecessarily the hospital stay and the return to normal daily activities and work. Meanwhile the incidence, intensity and duration of such postop-

erative headaches on up-right posture was significantly reduced in the second group of patients, namely those receiving autologous pericranium as dural graft, with the result of a quicker recovery.

One might attempt to reduce the incidence of CSF leaks by reducing the size of the craniectomy. Unfortunately in the Chiari I malformation a posterior fossa of small size seems to be the primary cause for the tonsilar herniation [4, 30, 58]. Thus it seems logical to attempt not only to decompress the herniated tonsils but also to increase the size of the posterior fossa. That demands a bigger craniectomy than just removal of the posterior rim of the foramen magnum. Once bone removal is completed, no real increase in the size of the posterior fossa is achieved unless the dura mater is opened. Then one may open the arachnoid membrane or leave it intact. Some state that it does not need to be violated, as good clinical results can be achieved anyway [20, 26, 50, 51]. That would reduce the incidence of CSF leaks. While that can be true for most cases, in some the neural malformation is significant and needs to be properly addressed [7, 8, 34]. Unfortunately, at present, there is no pre-operative diagnositc method able to predict in which cases the cerebrospinal fluid pathways will not be adequately opened without subarachnoid exploration [7, 8]. Opening the arachnoid layer and spreading the cerebellar tonsils apart provides a better CSF circulation, helping to reduce the incidence of occipital and nuchal pain related to Valsalva manoeuvres [44, 48, 61]. Another additional advantage is that dissecting and hitching the tonsils upwards helps to make it unnecessary to remove more bone than the posterior arch of the atlas. Accordingly we seldom need nowadays to perform a C_2 or C_3 laminectomy.

The most important factor in hindbrain abnormalities is not the caudal displacement of the tonsils in itself but the absence of a cisterna magna [13, 63]. To achieve normal cerebrospinal fluid dynamics at the cranio-cervical junction an artificial cisterna magna must be created [10, 13, 37, 51, 54, 56, 63]. A small cisterna magna does not correct the CSF abnormalities related with the Chiari I malformation. In order to create a cisterna magna of adequate size the dural graft must be large. Not grafting the dura mater exposes the arachnoid to blood and debris, increasing the chances of postoperative arachnoiditis [45].

We conclude that in our hands autologous pericranium, taken from the occipital area, gives better results than freeze-dried cadaveric dura mater in duraplasty for Chiari type I malformation.

References

1. Abbe R (1895) Rubber tissue for meningeal adhesions. Trans Am Surg Assoc 13: 490–491
2. Alarcon J, Dobato JL, Mateo D, Benito C, Gimenez-Roldan S (1992) Malformación de Arnold-Chiari con manifestaciones paroxísticas multiples provocades por la tos. Neurologia 7 (1): 25–29
3. Albers FW, Ingels KJ (1993) Otoneurological manifestations in Chiari I malformation. J Laryngol Otol 107(5): 441–443
4. Alles AJ, Sulik KK (1992) Pathogenesis of retinoid-induced hindbrain malformations in an experimental model. Clin Dysmorphol 1: 187–200
5. Alleyne CH, Barrow DL (1994) Immune response in hosts with cadaveric dural grafts. Report of two cases. J Neurosurg 81(4): 610–613
6. Barkovich AJ, Sherman JL, Citrin CM, Wippold FJ (1987) MR of postoperative syringomyelia. Am J Neuroradiol 8: 319–327
7. Batzdorf U (1988) Chiari I malformation with syringomyelia: evaluation of surgical therapy by magnetic resonance imaging. J Neurosurg 68: 726–730
8. Batzdorf U (1991) Syringomyelia related to abnormalities at the level of the craniovertebral junction. In: Batzdorf U (ed) Syringomyelia: current concepts in diagnosis and treatment. Williams and Wilkins, Baltimore, pp 163–182
9. Bhatia S, Bergethon PR, Blease S, Kemper T, Rosiello A, Zimbardi GP, Franzblau C, Spatz EL (1995) A synthetic dural prosthesis constructed from hydroxyethylmethacrylate hydrogels. J Neurosurg 83(5): 897–902
10. Blagodatsky MD, Larionov SN (1993) Surgical treatment of "Hindbrain related" syringomyelia long-term results. Acta Neurochir (Wien) 123(3–4): 209–210
11. Campbell JB, Bassett CAL, Robertson JW (1958) Clinical use of freeze-dried human dura mater. J Neurosurg 15: 207–214
12. Delasniere-Laupretre N, Alperovich A (1995) Epidemiologie de la maladie de Creutzfeldt-Jakob. Pathol Biol Paris 43(1): 22–24
13. Duddy MJ, Williams B (1991) Hindbrain migration after decompression for hindbrain hernia: a quantitative assessment using MRI. Br J Neurosurg 5(2): 141–152
14. Dysthe GN, Menezes AH, VanGilder JC (1989) Symptomatic Chiari malformations. J Neurosurg 71: 159–168
15. Edmeads J (1988) The cervical spine and headache. Neurology 38: 1874–1878
16. Elsahy NI, Achecar FA (1994) Use of the splenius capitis muscle flap for reconstruction of the posterior neck and skull in complicated Arnold-Chiari malformation. Plast Reconstr Surg 93(5): 1082–1086
17. Esmonde T, Lueck CJ, Symond L, Duchen LW, Will RG (1993) Creutzfeldt-Jakob disease and lyophilised dura mater grafts: report of two cases. J Neurol Neurosurg Psychiatry 56(9): 999–1000
18. Gok A, Zorludemir S, Polat S, Tap O, Kaya M (1995) Experimental evaluation of peritoneum and pericardium as dural substitutes. Res Exp Med Berl 195(1): 31–38
19. Görtler M, Braun M, Becker I, Roggendorf W, Heiss E, Groote E (1991) Animal experiments with a new dura graft (Polytetrafluoroethylene) results. Neurochirurgia 34: 103–106
20. Isu T, Sasaki H, Takamura H, Kobayashi N (1993) Foramen magnum decompression with removal of the outer layer of the

dura as treatment of syringomyelia occurring with Chiari I malformation. Neurosurgery 33(5): 845–850
21. Khurana RK (1991) Headache spectrum in Arnold-Chiari malformation. Headache 31: 151–155
22. Kline DG (1965) Dural replacement with resorbable collagen. Arch Surg 91: 924–929
23. Lang CJ, Schuler P, Engelhardt A, Spring A, Brown P (1995) Probalbe Creutzfeldt-Jakob disease after a cadaveric dural graft. Eur J Epidemiol 11(1): 79–81
24. Laquerriere A, Yun J, Tiollier J, Hemet J, Tadie M (1993) Experimental evaluation of bilayered human collagen as a dural substitute. J Neurosurg 78(3): 487–491
25. Levy WJ, Mason L, Hahn JF (1983) Chiari malformation presenting in adults: a surgical experience with 127 cases. Neurosurgery 12: 377–390
26. Logue V, Edwards MR (1981) Syringomyelia and its surgical treatment – an analysis of 75 patients. J Neurol Neurosurg Psychiatry 44: 273–284
27. Marin-Padilla M (1966) Mesodermal alterations induced by hypervitaminosis A. Am J Embriol Exp Morphol 15: 261–269
28. Marin-Padilla M (1979) Notocordal-basichondrocranium relationships: abnormalities in experimental axial skeletal (dysraphic) disorders. J Embriol Exp Morphol 53: 15–38
29. Marin-Padilla M (1991) Cephalic axial skeletal-neural dysraphic disorders: embriology and pathology. Can J Neurol Sci 18: 153–167
30. Marin-Padilla M, Marin-Padilla TM (1981) Morphogenesis in experimentally induced Arnold-Chiari malformation. J Neurol Sci 50: 29–55
31. Martinez-Lage JF, Poza M, Sola J, Tortosa JG, Brown P, Cervenakova L, Esteban JA, Mendoza A (1994) Accidental transmission of Creutzfeldt-Jakob disease by dural cadaveric grafts. J Neurol Neurosurg Psychiatry 57(9): 1091–1094
32. Martinez-Lage JF, Sola J, Poza M, Esteban JA (1993) Pediatric Creutzfeldt-Jakob disease: probable transmission by a dural graft. Childs Nerv Syst 9(4): 239–242
33. Massulo C, Pocchiari MA, Macchi G, Alema G, Piazza G, Panzera MA (1989) Transmission of Creutzfeldt-Jakob disease by dural cadaveric graft. J Neurosurg 71: 954–955
34. Milhorat TH, Miller JL, Johnson WD, Adler DE, Heger IM (1993) Anatomical basis of syringomyelia occurring with hindbrain lesions. Neurosurgery 32(5): 748–754
35. Meddings N, Scott R, Bullock R, French DA, Hide TA, Gorham SD (1992) Collagen vicryl: a new dural prosthesis. Acta Neurochir (Wien) 117(1–2): 53–58
36. MMWR (1993) Creutzfeldt-Jakob disease in patients who received a cadaveric dura mater graft: Spain, 1985–1992. MMWR Morb Mortal Wkly Rep 42(28): 560–563
37. Morgan D, Williams B (1992) Syringobulbia: a surgical appraisal. J Neurol Neurosurg Psychiatry 55(12): 1132–1141
38. Nakagawa H, Mikawa Y, Watanabe R (1994) Elastin in the human posterior longitudinal ligament and spinal dura. A histologic and biochemical study. Spine 19(19): 2164–2169
39. Narotam PK, Van Dellen JR, Bhoola K, Raidoo D (1993) Experimental evaluation of collagen sponge as a dural graft. Br J Neurosurg 7(6): 635–641
40. Negoro K, Tsuda N, Morimatsu M, Kurokawa Y, Abiko S (1993) A case of cough headache with Chiari malformation (type I). Rinsho Shinkeigaku 33(3): 327–330
41. Nightingale S, Williams B (1987) Hindbrain hernia headache. Lancet 1: 731–734
42. Nisbet TJ, MacDonaldson Y, Bisbara SN (1989) Creutzfeldt-Jakob disease in a second patient who received a cadaveric dura mater graft. JAMA 261: 1118–1121
43. Nyland H, Krogness KG (1978) Size of posterior fossa in Chiari type I malformation in adults. Acta Neurochir (Wien) 40: 233–242
44. Palma V, Sinisi L, Andreone V, Fazio N, Serra LL, Ambrosio G, De Michele G (1993) Hindbrain hernia headache and syncope in type I Arnold-Chiari malformation. Acta Neurol Napoli 15(6): 457–461
45. Parizek J, Sercl M, Michl A, Mericka P, Nemecek S, Nemeckova J, Jakubec J (1994) Posterior fossa duraplasty in children: remarks on surgery and clinical and CT follow-up. Childs Nerv Syst 10(7): 444–449
46. Pascual J, Oterino A, Berciano J (1992) Headache in type I Chiari malformation. Neurology 42(8): 1519–1521
47. Pillay AK, Awad IA, Little JD, Hahn JF (1991) Symptomatic Chiari malformations in adults: a new classification based on magnetic resonance imaging with clinical and prognostic significance. Neurosurgery 28: 639–645
48. Poplawska T, Zalewska H, Jankowicz E, Kozlowski P (1993) Cough headache and vertigo as symptoms of the Arnold-Chiari syndrome. Neurol Neurochir Pol 27(1): 105–109
49. Prichard J, Thadani V, Kalb R, Manuelidis E (1987) Rapidly progressive dementia in a patient who received a cadaveric dura mater graft. MMWR 34: 49–50
50. Raftopulos C, Sanchez A, Matos C, Baleriaux D, Bank WO, Brotchi J (1993) Hydrosyringomyelia-Chiari I complex. Prospective evaluation of a modified foramen magnum decompression procedure: preliminary results. Surg Neurol 39(2): 163–169
51. Sahuquillo J, Rubio E, Poca MA, Rovira A, Rodriguez-Baeza A, Cervera C (1994) Posterior fossa reconstruction: a surgical technique for the treatment of Chiari I malformation and Chiari I/syringomyelia complex. Preliminary results and magnetic resonance imaging quantitative assessment of hindbrain migration. Neurosurgery 35(5): 874–884
52. Sakamoto H, Hakuba A, Fujitani K, Nishimura S (1991) Surgical treatment of the rethered spinal cord after repair of lypomyelomeningocele. J Neurosurg 74: 709–714
53. San-Galli F, Darrouzet V, Rivel J, Baquey C, Ducassou D, Guerin J (1992) Experimental evaluation of a collagen-coated vicryl mesh as a dural substitute. Neurosurgery 30(3): 396–401
54. Samii M, Klekamp A, Sepehrnia A, Bothe HW, El Azm M, Sjuts E, Babapour B (1993) Syringomyelia associate with Arnold-Chiari I malformation and tumours of the posterior fossa. Long-term results of syringo-subarachnoid shunting and decompression of the posterior fossa. Acta Neurochir 123(3–4): 195
55. Sewell WH, Koth DR, Pate JW (1955) The present status of our experiments with freeze-dried grafts. Naval Med Res Ins Rep No MM007-081-10-1413: 291–401
56. Sgouros S, Williams B (1995) A criterical appraisal of drainage in syringomyelia. J Neurosurg 82(1): 1–10
57. Stovner LJ (1993) Headache associated with the Chiari type I malformation. Headache 33(4): 175–178
58. Stovner LJ, Bergan U, Nilsen G, Sjaastad O (1993) Posterior cranial fossa dimensions in the Chiari I malformation: relation

to pathogenesis and clinical presentation. Neuroradiology 35(2): 113–118
59. Takayama S, Hatsuda N, Matsumura K, Nakasu S, Handa J (1993) Creutzfeldt-Jakob disease transmitted by cadaveric dural graft: a case report. No Shinkei Geka 21(2): 167–170
60. Thadani V, Penar PL, Partington J, Kalb R, Janssen R, Schonberger LB, Rabkin CS, Prichard JW (1988) Creutzfeldt-Jakob disease probably acquired from a cadaveric dura mater graft. Case report. J Neurosurg 69: 766–769
61. Van den Bergh R (1992) Headache caused by craniospinal pressure dissociation in the Arnold-Chiari-syringomyelia syndrome. J Neurol 239(5): 263–266
62. Vega A, Quintana F, Berciano J (1990) Basicondrocranium anomalies in adult Chiari type I malformation: a morphometric study. J Neurol Sci 99: 137–145
63. Williams B (1991) Pathogenesis of syringomyelia. In: Batzdorf U (ed) Syringomyelia: current concepts in diagnosis and treatment. Williams and Wilkins, Baltimore, pp 59–90
64. Williamson MR, Boyd CM (1989) Prosthetic vascular graft infections: diagnosis and treatment. Crit Rev Diagn Imaging 29(2): 181–213
65. Willison J (1989) Up-date: Creutzfeldt-Jakob disease in a second patient who received a cadaveric dura mater graft. MMWR Morb Mortal Wkly Rep 38(3): 37–38
66. Yamada S, Aiba T, Endo Y, Hara M, Kitamoto T, Tateishi J (1994) Creutzfeldt-Jakob disease transmitted by a cadaveric dura mater graft. Neurosurgery 34(4): 740–743

Comments

This is a nicely written paper on an important surgical detail in surgery for Chiari malformation: the type of dura repair.

The authors mention that a large graft is needed. I do not agree with this statement. If the decompression is merely undertaken to enlarge the foramen magnum and to create a cisterna magna, a graft of about 3 × 3 cm is all that is required.

A number of recent publications recommend one to open only the dura or even just the outer dural layer. They claim just as good results avoiding problems of csf-leakage completely in this manner. I personally am in favour of arachnoid dissection suggested by the authors.

M. Samii

Correspondence: Dr. V. Vanaclocha, Division of Neurosurgery, Clinica Universitaria de Navarra, Avda. Pio XII 36, Apdo Correos 4209, E-31008 Pamplona, Navarra, Spain.