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Surgical Management of Brain Stem Vascular Malformations

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Summary

Vascular malformations of the brain stem are a histologically heterogenous collection of lesions which most often present with sudden and progressive neurologic deficit related to haemorrhage. Since 1987 the authors have treated eleven cases of brain stem vascular malformation. Seven of the patients were treated with complete surgical extirpation of the haematoma and malformation because of progressive neurologic deficit. Four additional patients made a full neurologic recovery and are being carefully observed for signs suggestive of the need for surgical treatment. Complete surgical excision of brain stem vascular malformations is mandatory for patients with progressive neurologic deficit related to recurrent haemorrhage.

Keywords: Cavernous angioma; brain stem; vascular malformation.

Introduction

Angiography has been considered the diagnostic procedure of choice for elucidating intracranial vascular malformations[49]. In 1956 Crawford and Russell, and subsequently others, documented the character of angiographically occult or "cryptic" vascular malformations[7, 10, 17, 31, 32, 59, 60, 71, 76, 78, 84, 86, 102, 107].

Magnetic resonance imaging makes the designation and description of cryptic vascular malformations obsolete[29, 31, 32, 34, 35]. The malformations which were formerly obscure now have distinct morphologic features which establish their presence and location prior to surgical intervention[35, 65]. In fact, vascular malformations of the brain stem have been considered rare lesions. Prior to 1982, only 26 cases of surgical exploration of intrinsic brain stem haematoma were reported in the literature[1, 2, 3, 18, 36, 43, 44, 54, 56, 57, 63, 66, 69, 72, 74, 75, 78, 79, 84, 87, 94, 97]. Vascular malformation was suspected as the cause of haemorrhage, but was confirmed histologically in only three of those cases[54, 78, 84]. The number of reported cases of surgical treatment of brain stem haematoma and suspected malformation has increased as a result of imaging[4, 13, 15, 20, 26, 27, 33, 34, 40, 41, 43, 45, 46, 47, 49, 53, 55, 58, 61, 64, 67, 68, 70, 73, 82, 83, 85, 88, 89, 94, 95, 98, 100, 103, 104, 105, 106]. This report contributes eleven additional cases of documented brain stem vascular malformation, seven of which were surgically excised.

Material

Eleven patients with brain stem vascular malformations have been evaluated at our institution in 24 months (Table 1). There were five males and six females, ranging in age from twenty-two to forty-three years old. Seven patients presented with progressive neurologic deterioration secondary to recurrent intraparenchymal haemorrhage (cases 1–5, 9, 11). One patient (case 6) has had two episodes of neurologic deficit related to haemorrhage, but has regained complete neurologic function, while another patient (case 7) remains intact after a single haemorrhage. Another patient (case 8) had a cavernous angioma resected from the right temporal lobe in 1982, and a routine magnetic resonance image has documented the presence of a pontine malformation without clinical findings. Another patient has a cavernous malformation of the brain stem which is asymptomatic (case 10). The clinical presentation associated with vascular malformation in the medulla, pons and midbrain are best correlated with the effects of recurrent intraparenchymal haemorrhage. Seven lesions were located in the dorsal aspect of the pons near the floor of the fourth ventricle. One patient (case 9) had a lesion in the lateral tectum; another lesion was buried deep in the pons (case 10). Two patients had lesions in the dorsal mesencephalon (case 5, 11). Presenting symptoms in order of frequency include diplopia, facial numbness, hemisensory abnormalities, incoordination, ataxia and headache. Clinical signs are abducens and occulomotor palsy, internuclear ophthalmoplegia, nuclear facial palsy, nystagmus, dysarthria, facial and hemisensory deficit, hyperreflexia, cerebellar ataxia and hemiparesis.

Angiography was performed in five cases (case 1–5). Only one study documented any angiographic abnormality. Slowly filling veins in the floor of the 4th ventricle suggested a venous angioma (case 1). Angiography was normal in the remaining cases.

Magnetic resonance imaging was done in all eleven cases. The examination was performed using T_1 weighted (spin-echo TR 500/TE 20 msec) and T_2 weighted (TR 2000/TE 80 msec) technique. All

Table 1. *Clinical Data*

Case	Age	Sex	Symptoms	Preoperative findings	Angiography findings	MRI findings	Histology	Follow-up (Months)	Postoperative status
1	25	M	Diplopia Lt hand numbness Lt facial numness	Rt VI palsy Nystagmus Lt V hypesthesia Lt UE hypalgesia	venous angioma	1.5 cm haematoma acute/subacute haemorrhage abnormal vessels	venous angioma	25	Mild ataxia, Rt VII palsy improved employed as computer operator programmer
2	22	F	Headache Dilplopia Rt face, body numbness Rt hand incoordination	Rt VI palsy Nystagmus Rt V hypesthesia Rt UE, LE hypesthesia Rt dysmetria Appendicular ataxia Hyperreflexia	negativ	2.0 cm haematoma acute/subacute/ chronic haemorrhage abnormal vessels	cavernous angioma	16	Internuclear ophthalmoplegia resolved truncal ataxia employed as school teacher
3[1]	36	M	Headache Diplopia Rt hand incoordination Lt body numbness	Rt VI palsy Rt III palsy Rt UE dysmetria Lt UE, LE hypesthesia	negative	2.0 cm haematoma acute/chronic haemorrhage	cavernous mangioma	he-10	New Lt. VI palsy Rt VI, VII palsy, Rt UE dysmetria all improved
4	39	F	Diplopia Lt face, body numbness Unsteady gait	Rt VI palsy Rt VII palsy Lt VII palsy; partial Lt hemiparesis Ataxia Dysarthria Hyperreflexia	negative	2.5 cm haematoma acute/subacute/ chronic haemorrhage abnormal vessel	cavernous heman-gioma	4	No new deficit ataxia improved
5	43	M	Diplopia Rt facial, palatal numbness Rt hand paresthesias incoordination Unsteady gait	Nystagmus Rt dysmetria, dysdiadochokinesia Ataxia	negative	2.5 cm haematoma acute/subacute chronic haemorrhage abnormal vessel	cavernous haeman-gioma	14	Rt internuclear ophthalmoplegia improved Lt pupil sparing III Palsy improved Appendicular/ truncal ataxia Hydrocephalus treated
6	36	M	Diplopia Rt facial numbness Rt face hypesthesia Unsteady Gait	Nystagmus Ataxia	not done	2.0 cm haematoma acute haemorrhage	—	8	Neurologically intact, not yet operated
7	34	F	Diplopia Lt hand weakness	Rt VI palsy Rt VII palsy Lt hemiparesis Hyperreflexia	not done	2.0 cm haematoma subacute/ chronic haemorrhage	—	12	Neurologically intact, not yet operated

Table 1 (*continued*)

Case	Age	Sex	Symptoms	Preoperative findings	Angiography findings	MRI findings	Histology	Follow-up (Months)	Postoperative status
8	43	F	None	Intact	not done	1.5 cm haematoma subacute/ chronic haemorrhage	—	12	Neurologically intact, not yet operated
9	33	F	Headache Lt analgisea Lt. weakness Diplopia	Rt V, VI, VII, VIII IX, X, palsies Lt hemiplegia Lt analgesia Lt intranuclear ophthalmoplegia	not done	2.0 cm haematoma acute/subacute haemorrhage	cavernous hemangioma	6	Lt internuclear ophthal-moplegia resolved Rt, V, VII, IX, X improving
10	40	F	Headache	Intact	not done	1.5 cm haematoma chronic haemorrhage	—	12	Neurologically intact, not yet operated
11[2]	23	F	Diplopia Ataxia	Rt upward gaze palsy Rt partial III palsy Rt dysmetria Ataxia	negative	2.5 cm haematoma acute/subacute chronic haemorrhage	cavernous hemangioma	7	Lt III, VI palsy improving Lt peripheral VII palsy improv-ing

[1] Patient was previously treated with proton beam therapy, but continued to have neurologic deterioration after treatment.
[2] Patient was eight weeks pregnant and had rapid neurologic deterioration secondary to two haemorrhages in a three day period.

studies documented a consistent morphologic appearance consisting of a central focus of mixed signal intensity representing haemorrhage of various ages. The nidus was surrounded by a low signal ring consistent with hemosidern deposition from remote haemorrhage (Fig. 1). The combined diameter of the malformation and haematoma ranged in size from 1.5 cm to 3.5 cm. Several studies showed a pattern suggesting the presence of multiple vessels containing slow flowing blood (flow void).

Surgical Technique

Seven patients (cases 1–5, 9, 11) were considered candidates for surgical treatment of their malformations. In these cases, recurrent haemorrhage resulted in progressive neurologic deficit. Preoperatively each patient receives a 10 mg intravenous bolus of dexamethasone as well as 1 gram intravenous administration of a cephalosporin for antimicrobial prophylaxis. The patient is then placed in a lateral oblique position with the thorax elevated 15°. The head is held in slight lateral extension and cervical flexion with the Mayfield three point fixation device. Appropriate padding is placed, including an axillary roll for protection of the brachial plexus. An 18 gauge spinal needle is placed in the lumbar cistern for cerebrospinal fluid drainage and hyperventilation to a PCO_2 25–30 mmHg is maintained.

A midline suboccipital skin incision is made from the inion to C_2 (Fig. 2). A suboccipital craniotomy is than performed using Midas Rex power instrumentation. The dura is opened in a Y-fashion with the incision based on the transverse sinus. The Budde halo system of flexible arm self-retracting blades is used to expose the vermis in the midline. For lesions of the pons or medulla, the lower one third of the vermis is split using the focused CO_2 laser energy at 10 watts which is delivered via a micromanipulator attached to the operating microscope. For lesions of the mesencephalon, the upper vermis is sectioned. The retractors are used to protect the cerebellum and provide visualization of the floor of the fourth ventricle. In cases 1–5, and 11 the vascular lesion or haematoma was easily identified in a subependymal location distorting the floor of the fourth ventricle of dorsal midbrain. The haematoma cavity is opened with the CO_2 laser. The haematoma is carefully resected by using suction for the more liquified component, and Nd : Yag laser energy for the more solid component. The cavity is often subdivided by numerous trabeculations and cavities, suggesting the presence of haemorrhage of various ages. In this series, a vascular malformation was identified in all cases. The nidus of the malformation should be shrunk with Nd : Yag laser, which uses haemogloblin as a heat sink to shrink collagen in walls of vessels[108]. Multiple microscopic arterial feeders, arising from the substance of the brain stem are controlled with impedence monitored bipolar coagulation, and laser treatment. The Nd : Yag laser energy is applied with a hand held fiber, using 1–2 watts of focused power. Some malformations contain large vascular channels which require obliteration with similar techniques. Obliteration of the entire malformation must be achieved. Direct inspection of the cavity is necessary to achieve total excision. Intra-operative angiography is not effective for these vascular anomalies. Postoperative MR is used to confirm total removal of these lesions.

In some circumstances a lateral approach is needed to remove

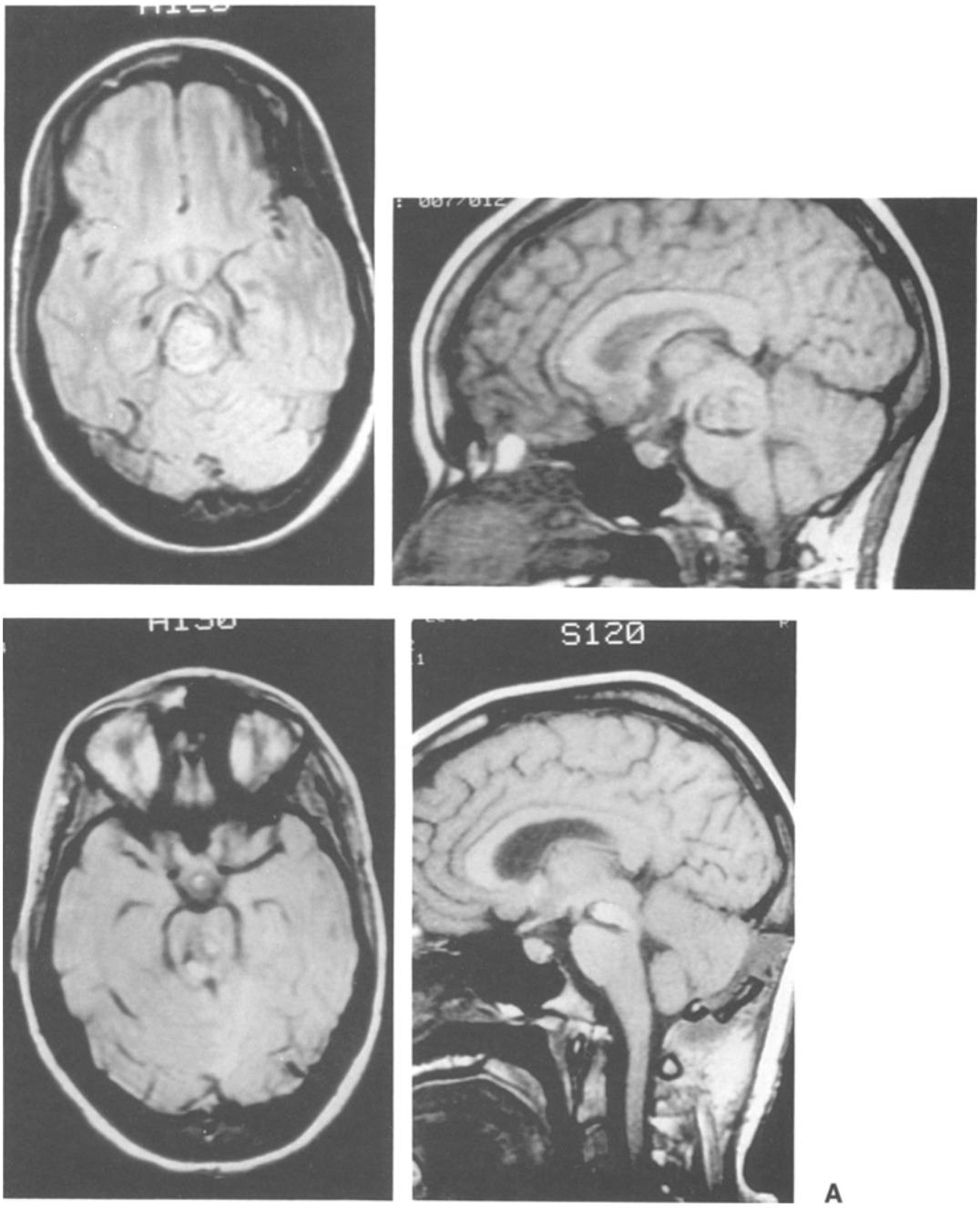

Fig. 1. Magnetic resonance imaging provides definitive diagnostic information regarding cavernous angiomas. T_1 weighted (TR 500/TE 20 msec) technique shows a non-homogenous spherical lesion with mixed signal intensity representative of haemorrhage of various ages. There is a surrounding zone of low signal representing hemosiderin. Preoperative and postoperative images are shown of a mesencephalic (A) and ventral pontine (B) malformation

a haematoma that presents near the exterior surface of the pons or lateral mesencephalon (Fig. 2). In this circumstance the patient is placed in a similar position. A paramedian skin incision, one centimeter medial to the mastiod process will provide access to the cerebellar pontine angle via a suboccipital craniotomy. If the lesion is more rostral or more anterior, a petrosal approach with division of the tentorium will provide excellent access to the lesion.

Ultrasound and steretaxis may be needed in some cases to localize the lesion. Patients should remain intubated until they are alert and cranial nerve function can be assessed. Tracheostomy and gastric diversion is needed for some patients. Sodium nitropusside is used to keep systolic blood pressure below 140 mmHg. Antibiotics are continued for twenty-four hours and dexamethasone for seven days.

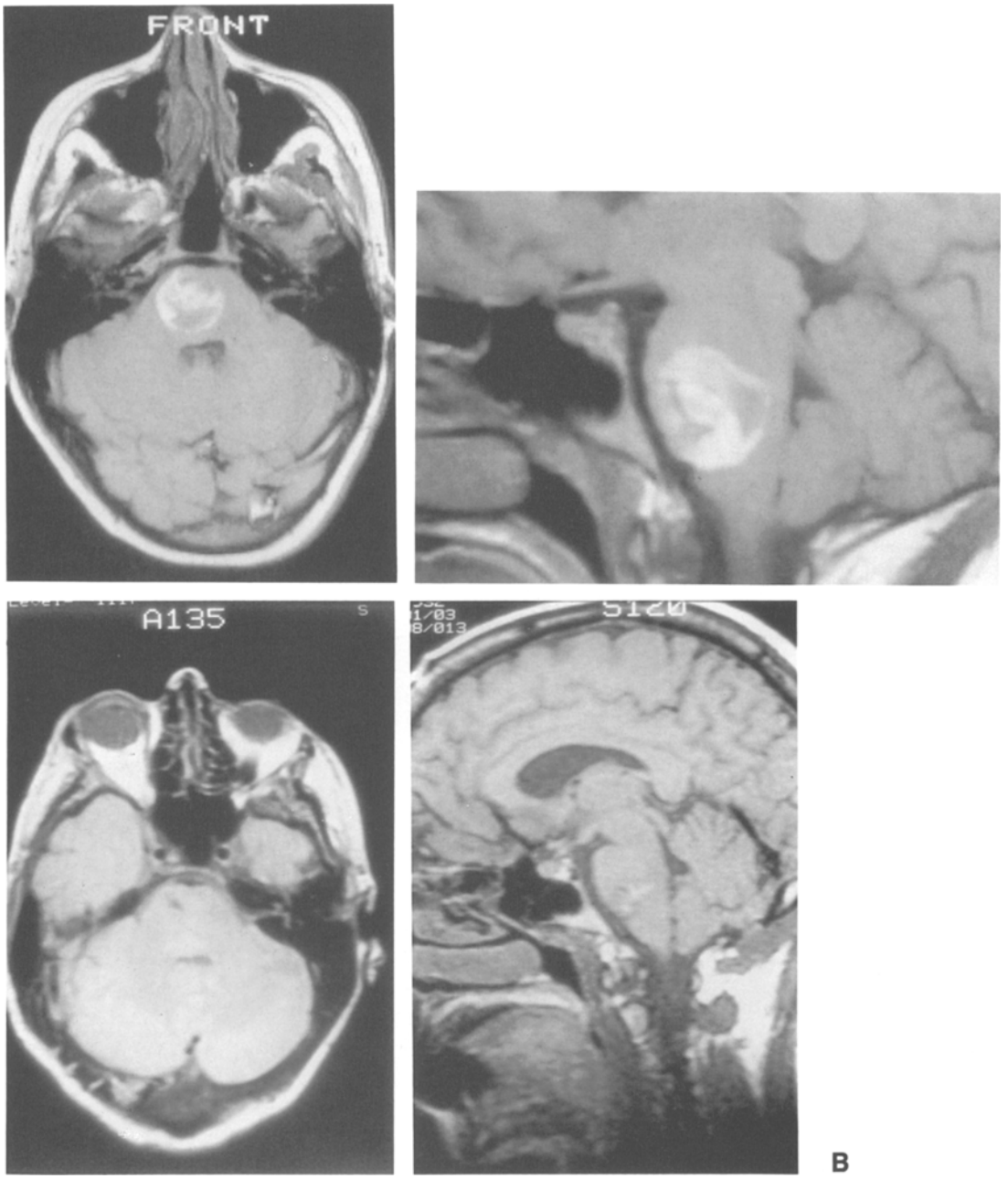

Fig. 1 B

Results

Surgical Results

Histologic examination of the malformations demonstrated a surprisingly heterogenous group of lesions. Case 1 was a venous angioma. Cases 2, 3, 4, 5, 9 and 11 were cavernous angiomas, characterized by thin walled vascular spaces with little or no intervening neural tissue. Cases 3 and 5 also contained minor components of arteriovenous malformations. Several areas demonstrated transitional vessels as well as significant intervening gliotic tissue. Clinical follow-up is now available on all seven surgical patients, ranging from one to twenty-seven months. All patients, except one (case 3) experienced transient neurologic worsening. New neurological deficit consisted of truncal ataxia (3 cases), internuclear ophthalmoplegia (2 cases), abducens palsy (1 case), paralysis of upward gaze (2 cases) and nuclear facial weakness (3 cases). In cases 1–4, 9 and 11 the patient's deficits have resolved to allow them to return to previous employment. Case 5 required ventriculoperitoneal shunting for aqueductal stenosis

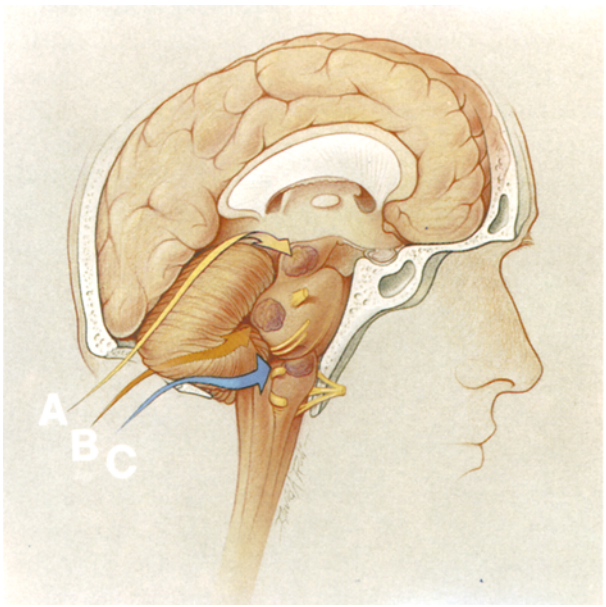

Fig. 2

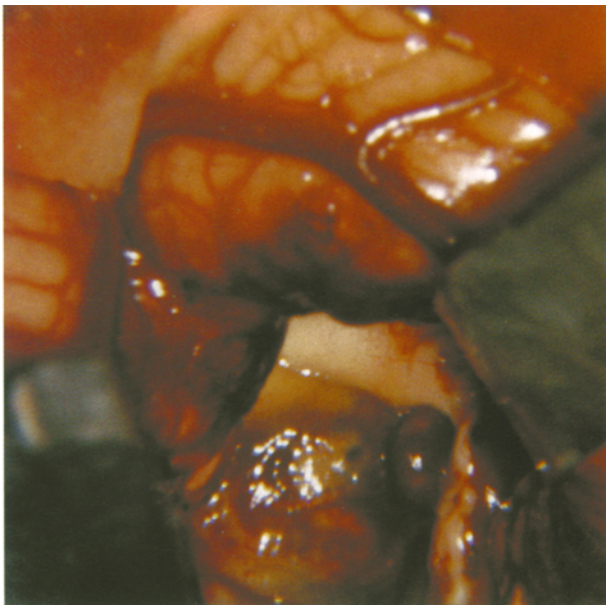

Fig. 3

Fig. 2. This illustration demonstrates the multiple surgical approaches available for brain stem vascular malformations. If the lesion is located in the dorsal mesencephalon, midline suboccipital approach with section of the upper vermis can be done (*A*). A midline suboccipital approach with section of the lower vermis is used for dorsal pontomedullary lesions (*B*). Lesions in the lateral or ventral brain stem can be approached via a lateral suboccipital approach (*C*)

Fig. 3. Cavernous angiomas are well circumscribed lesions which often bulge out of the brain stem. The discoloration and distortion caused by the haematoma can be used to guide resection as in the lesion on the floor of the fourth ventricle

after removal of a midbrain cavernous malformation. Removal of the malformation resulted in aqueductal scarring and subsequent obstructive hydrocephalus. Case 11 was eight weeks pregnant at the time of resection. Postoperative MRI shows complete removal of the haematoma and malformation in all cases.

Results of Non-operative Treatment

Cases 6 and 7 presented with one and two episodes of neurologic deterioration secondary to haemorrhage, respectively. At the present time, both patients are employed and free of neurologic deficit. Case 9 had a cavernous malformation of the brain stem, diagnosed by MRI during workup for headache and she is likewise neurologically intact. These patients are followed on a regular basis for evidence of neurologic deterioration.

Case 8 is a 43 year old mother of three children all of whom have multiple cavernous angiomas by magnetic resonance and histologic confirmation. She had a cavernous angioma removed from the right temporal lobe after a haemorrhage in 1982. She has a pontine malformation which was found on a screening MRI, and remains asymptomatic.

Discussion

Dandy first reported surgical removal of a brain stem haematoma in 1932[32]. He suggested that the lesion was secondary to a cavernous angioma, but no histologic confirmation could be obtained. In the majority of subsequent reports, the cause of haemorrhage was not identified. Pathologically documented cases include cavernous angiomas, thrombosed arteriovenous malformations and venous angiomas.

Two surgical approaches have been used to achieve access to vascular malformations of the upper brain stem. Haematomas that extend towards the lateral aspect of the brain stem can be removed through subtemporal transtentorial or a lateral suboccipital approach[39, 81, 101]. The midline approach to the floor of the fourth ventricle through the vermis provides broad access to central lesions[14, 34, 62, 66, 75]. Direct access to rostral lesion can be obtained via a transoral/transclivial approach.

Magnetic resonance imaging is important in determining the surgical approach[80]. Localization of the haematoma is accurately determined with MR[29]. Magnetic resonance also demonstrated the relative age of

the haemorrhages[31, 32]. The aging of haemoglobin is documented with a differential signal intensity[29, 31, 34]. Characteristically, brain stem vascular malformations have a nodular, heterogenous signal surrounded by a low signal ring indicating the presence of hemosiderin. Flow void documenting the site of a vascular malformation is inconsistently present. This finding is related to the dynamically slow flow nature of blood in these lesions[35].

Surgical treatment of brain stem vascular malformations has not been uniformally accepted. Some writers have argued the lesions are associated with limited morbidity and no mortality[77]. Risk of brain stem exploration is considered prohibitive. Recent experience, however documents that progressive recurrent haemorrhage results in consequential disability[19, 21, 30, 48, 51, 90]. Moreover, some authors have reported mortality associated with brain stem vascular malformation[8, 14, 23, 28, 37, 50, 96]. Extirpation of the malformation should arrest the progressive deficit if surgical removal is appropriately planned and executed. Surgical morbidity can definitely be minimized. The midline approach to the fourth ventricle is associated with transient truncal ataxia due to splitting of vermis. New cranial nerve deficits related to actual removal of the lesion from the dorsal brain stem has been transient and are most likely secondary to postoperative oedema.

Some reports conclude that cavernous angiomas are benign lesions[10, 29, 42]. Two of our cases, with the characteristic MR appearance of a cavernous angioma, however, also had a histologic portion of an active arteriovenous component. This finding cannot be elucidated by MR. Only histologic evidence documents the more ominous nature of this surgical lesion.

Two reports in the literature recommend stereotaxic drainage of brain stem haematoma[5, 9]. This technique can cause temporary improvement in neurologic deficit by decreasing mass effect of the haematoma, however, the risk of recurrent haemorrhage from the malformation still persists.

Radiation therapy has been used to treat brain stem vascular malformations with inconclusive results[92]. Gamma radiosurgery is no longer recommended for treatment of brain stem vascular malformations. In a series of 17 cases, eight have continued to have progressive neurologic deterioration due to recurrent haemorrhage or radiation injury. Radiosurgical treatment of cavernous angioma is not recommended until further evaluation of these treatment failures has been completed[93]. Direct surgical approach is imperative when disabling neurologic deficit occurs and fails to reverse rapidly. Minimally affected patients may be suitable for surgery provided the lesion is located in an accessible area or remote from strategic structures. Intact patients should be followed until evidence of neurological worsening occurs; surgery is then the treatment of choice. Failure to regain rapid recovery is another indication for surgical removal. Rapid recurrent haemorrhage led to irreversible deterioration in several patients that we have evaluated. Unfortunately there are no diagnostic criteria to determine which lesions are more likely to rebleed in a more devastating manner.

Conclusion

Vascular malformations of the brain stem can produce significant neurologic disability secondary to recurrent haemorrhage. MR imaging provides the definitive diagnostic tool to characterize these lesions. Total surgical resection of the malformation and haematoma is necessary to prevent progressive and irreversible neurologic deficit in selected cases.

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