

Anaesthesia for closed embolisation of cerebral arteriovenous malformations

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Summary

Anaesthetic experience of the first nine patients in Singapore who underwent closed embolisation of cerebral arteriovenous malformations is reported. Six patients had neurolept analgesia and three had general anaesthesia.

Key words: Anaesthesia, embolisation, arteriovenous malformation, neurolept analgesia.

Introduction

Artificial embolisation of Cerebral Arteriovenous Malformations (CAVM) is a therapeutic radiological technique undertaken in the x-ray department. It involves the selective cannulation of cerebral vessels via the femoral artery or through craniotomy of cerebral vessels of the CAVM.^{1,2} Various types of embolic material¹⁻⁹ is then injected into the nidus of the abnormality to cause intra-arterial embolisation. The aim of the procedure is to obliterate the nidus of the CAVM or occlude the feeder vessels so as to reduce its vascularity.

However, passing of the embolic material to vessels other than the CAVM is a major problem. Ideally only the blood supply to the abnormality should be occluded while the arterial supply to the normal tissue is preserved. Thus careful placement of the catheter within the abnormal vessels to maintain control over the occluding agent is very important.

Patients requiring this procedure either have a huge CAVM with multiple feeders, a non-resectable CAVM by nature of its site or size, or have a huge CAVM which is resectable with a high risk of serious neurological deficit.

This form of treatment is not recommended for those with functional brain involvement, those with permanent neurological deficit and those who are symptom-free.

Embolisation can be used as the sole treatment of CAVM¹⁰ or as an adjunct to surgical removal or radiotherapy¹¹.

We report our anaesthetic experience with nine patients who presented for embolisation of CAVM at the National University Hospital (NUH) from 1988 to 1989. This is the first time such procedures were carried out in Singapore and the second such report in the literature¹².

Materials and methods

This is a personal series of anaesthesia for the first nine cases of closed embolisation of CAVM in Singapore. The age range of our nine patients at presentation was 16 to 39 years with six males and three females. Neurolept analgesia was administered to six cases and three had general anaesthesia. Of the three patients who needed general anaesthesia, one had airway problems from morbid obesity and chronic cough and for the other two, the embolisation process through the external carotid artery was expected to be painful.

Embolisation was carried out with bucrylate in five cases (Figs. 1,2,3), detachable silicone balloon in two cases and gelfoam in one case. As we had no previous experience with this type of radiological procedure, we used various anaesthetic techniques in an attempt to find the most suitable method.

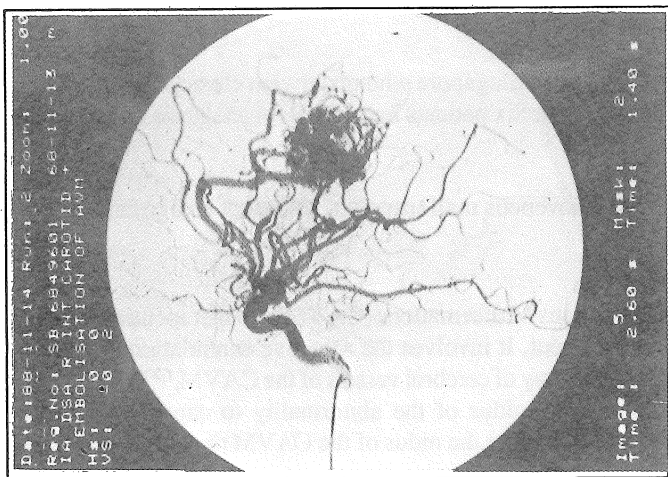


Fig. 1:
Angiogram showing a left parietal arteriovenous malformation

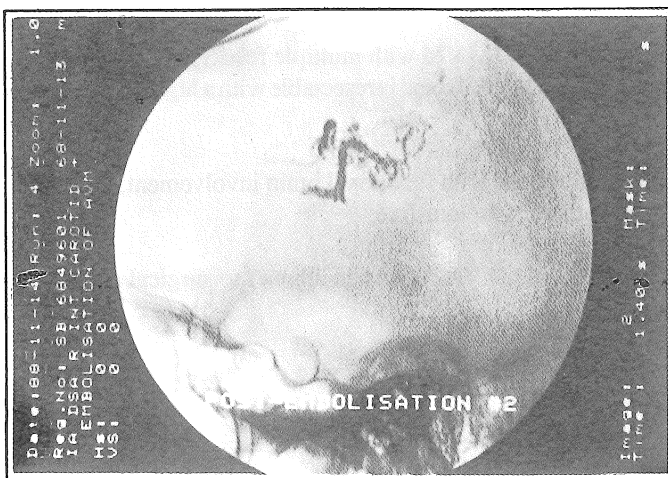


Fig. 2:
Bucrylate cast in feeder vessel of the cerebral arteriovenous malformation

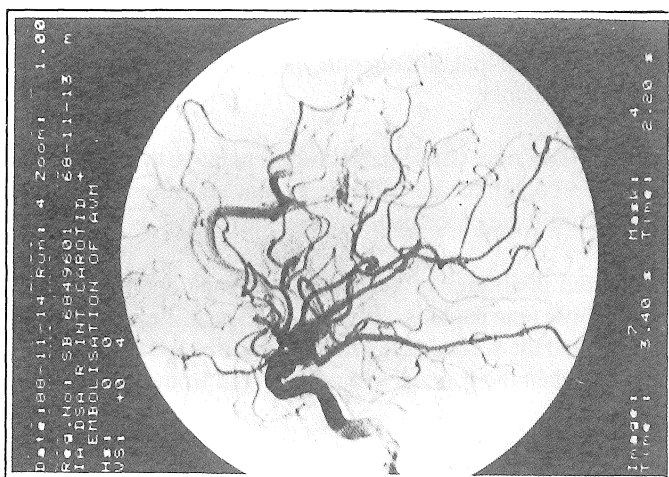


Fig. 3:
Post-embolisation angiogram
showing obliteration of the
cerebral arteriovenous
malformation

All patients were seen by the anaesthetist on the day before the procedure and a thorough assessment was carried out. Premedication consisted of a sedative, ordered for the morning of the procedure.

Once in the X-ray room, intravenous access was secured and for anticipated long procedures, a urinary catheter was inserted. The patient was closely monitored throughout the procedure with ECG, respiratory rate, capnometer, automated BP cuff or intra-arterial line and pulse oximeter. The darkened X-ray room made it difficult to assess the patient's colour. This made close monitoring essential. Neurolept analgesia was induced with combinations of fentanyl, droperidol and midazolam until the patient was sedated and drowsy but rousable. Incremental doses of these drugs were given when necessary to ensure an immobile patient. Supplemental oxygen was administered through the ventimask to maintain adequate oxygen saturation.

For patients who were given general anaesthesia, a balanced technique was carried out using intermittent positive pressure ventilation with nitrous oxide and oxygen, isoflurane and intravenous fentanyl. For muscle relaxation, we used d-tubocurarine in one patient and atracurium infusion for the other two.

The radiologist did not request for controlled hypotension in all the cases. However, four patients required systemic heparinisation and this was achieved with heparin < 2mg/kg, given intravenously. Anticoagulation was reversed with protamine sulphate until the activated clotting time reached pre-operative levels.

Following the embolisation, one patient was sent to the intensive care unit for monitoring because he was morbidly obese and had respiratory decompensation even before the procedure. After a period of observation in the recovery room of the operating theatre, all the other patients were sent to the general ward.

In the post-operative period, steroids was given to all the patients. The regimen was dexamethasone 8 mg three times daily for three days followed by decreasing doses over the next three days. The two patients with balloon embolisation were advised to avoid excessive head movements and coughing as this may cause the embolus to dislodge. Sedatives and cough suppressants were prescribed. Analgesics were ordered for headaches and muscle spasm which were expected in some cases. The groin was also observed for bleeding from the femoral puncture site.

Results

In our series of nine patients (Table I & II), we found that, although the procedure was invasive and uncomfortable, general anaesthesia was not mandatory.

Six patients had neurolept analgesia (Table I). They were comfortable and cooperative throughout the procedure. Neurolept analgesia enabled us to communicate with the patient and allowed regular neurological examinations to be performed during the procedure.

Successful embolisation of the lesion occurred in all nine of the patients attempted. One required further surgical treatment of her CAVM. Of these nine patients, two developed neurological deficits: one had fifth and seventh cranial nerve palsy and the second patient complained of left hemiparesis. These neurological deficits were still present when the patients were discharged from the hospital.

Table I
Summary of cases done under neurolept analgesia

Patient	Sex/Age	Diagnose	Embolic Material
WCHL	M / 20	Parasagittal CAVM	Bucrylate
ORK	M / 23	Bilateral carotico-cavernous fistula	Silicone balloon
EH	M / 16	Right frontal CAVM	Bucrylate
HS	M / 30	Right medial parietal CAVM	Bucrylate
BY	M / 39	Limbic / corpus callosum CAVM	Bucrylate
TOK	F / 50	Parietal CAVM	Bucrylate

Table II
Summary of cases done under general anaesthesia

Patient	Sex/Age	Diagnose	Embolic Material
HBS	M / 39	Dural A-V fistula	Silicone balloon
W	F / 30	Masseter / mandibular AVM	Gelfoam
SK	M / 2	Dural AVM	Bucrylate

Discussion

Interventional radiology with closed embolisation as a therapeutic tool in the treatment of cerebral arterio-venous malformation (CAVM) is now well-established¹³. Patients suitable for such treatment include those with large CAVM with multiple feeders and non resectable CAVM by nature of site or size. Embolisation is used either as the sole treatment or as an adjunct to subsequent surgery or radio therapy.

In a long term follow up study of patients with CAVM, Troupp et al¹⁴ found a 10% mortality attributable to the CAVM and 24% of patients were disabled by the lesion. Drake¹⁵ found that parietal, central and infratentorial lesions were associated with a higher mortality compared with frontal, temporal and occipital CAVM. Patient with epilepsy and history of cerebral bleed have a greater chance of subsequent bleed and small CAVM are also more likely to bleed.

The CAVM may be approached by skillfull catheter manipulation and they or their feeding vessels are then embolised with any of a variety of synthetic or natural agents. One of the newest material to be used for this purpose is Bucrylate or Isobutyl 2-Cyanoacrylate^{16,17,18}. This is a liquid adhesive material of low viscosity that polymerises rapidly on contact with blood. Follow-up studies on the effect of this agent on the embolised tissue and its advantages over other embolic substances are still being done^{9,19,20}.

The choice of the embolic material used depend on its availability, the radiologist's familiarity with the substance and the lesions to be embolised.

With increasing use of embolisation for the treatment of CAVM, more patients will present for anaesthetic management during these procedures. Anaesthesia for closed embolisation of CAVM was first reported by O'Mahoney and Bolsin¹² in Perth, Western Australia, in 1988.

The choice of anaesthesia would depend on several factors including age, patient's request, patient's co-operativeness and other medical problems. General anaesthesia with neuromuscular blockade would be preferred in infants and young children, patients with airway problems & chronic backache and those with raised intracranial pressure.

Neurolept analgesia, however, allows neurological assessment to be carried out during the procedure. The ability to diagnose neurological complications immediately, make this the anaesthesia of choice for this procedure.

As many of these patients are very anxious, a thorough explanation of the procedure and the conditions in the X-ray department do much to allay their apprehension. If neurolept analgesia is chosen, the patient must be assured that the anaesthetist will be on hand to provide pain relief should he find the procedure intolerable.

A complete assessment of the cardiovascular system is important as the presence of atherosclerotic and coronary artery disease would be a contra-indication to induced hypotension during embolisation. Any history of epilepsy and anticonvulsant therapy should be noted. Chronic smokers and those with chronic cough and chronic backache may require general anaesthesia to ensure complete immobility during the procedure.

A baseline pre-operative coagulation profile and activated clotting time (ACT) is obtained because systemic heparinisation may be employed during the embolisation.

Pre-medication usually consist of a sedative given orally.

Some radiologists advocate the use of nifedipine²¹ before the procedure to prevent cerebral vasospasm. It has been one of the most widely studied calcium antagonists in this respect. Two oral doses are given. The first one on the night before and the second two hours prior to the embolisation.

The use of deliberate arterial hypotension during the procedure may facilitate the embolisation of the CAVM. The choice of the technique and the ideal arterial pressure for controlled hypotension remains controversial¹².

Controlled hypotension reduces the rates of blood flow through the CAVM. This increases the chance of the embolic material occluding the abnormal feeder vessels and the nidus of the CAVM and decreases the risk of embolisation occurring in the venous drainage system and the normal cortical tissue. Occlusion of the venous part of the CAVM without completely occluding the arterial feeders may result in congestion and bleeding of the CAVM.

Hypotensive agents are also useful in controlling the hypertensive response secondary to vascular spasm often associated with the rapid removal of the intra-cerebral arterial catheters immediately following bucrylate injection.

Heparinisation is necessary to prevent intravascular thrombosis. This is more likely to occur in long procedures and when smaller vessels are cannulated. Heparinisation however, carries a risk of causing bleeding during manipulation of the catheter in the abnormal cerebral vasculature.

Many complications may occur during the close embolisation of CAVM^{7,22,23}. The most dreaded complication is intracranial haemorrhage. This may appear during or in the post-embolisation phase.

Various neurological deficits may occur. These may be temporary and would resolve spontaneously post-operatively. Permanent neurological deficit is due to brain infarct.

Cortical venous thrombosis resulting from venous outflow occlusion without arterial inflow obstruction may lead to delayed bleeding.

Passage of the embolic material to sites other than the intended target and technical faults such as embedding of the catheters in the bucrylate cast have been reported.

Conclusion

Deliberate embolisation is now gaining acceptance as a mode of treatment for CAVM. More patients will present for anaesthetic management for this procedure. Neurolept analgesia is the preferred method. General anaesthesia is recommended for cases with other complicating medical conditions and when immobility cannot be guaranteed.

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