

Advances in Interventional Neuroradiology in Stroke : Some Aspects on Vascular Malformations and Occlusive Arterial Disease

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Abstract

Stroke is a serious cause for morbidity and to a lesser extent mortality. Interventional neuroradiological techniques have helped in the effective management of some problems of stroke. Advances in catheter delivery systems, microballoon technology and the use of liquid polymers has proved effective in the treatment of AVMs and aneurysms especially those which were thought inoperable. Endovascular treatment has become the primary modality of treatment in some of these lesions. Similarly, the extension of the technique of arterial dilatation in stenotic lesions is being applied more frequently to the extracranial carotid and vertebral circulations for atherosclerosis and other kinds of arterides with excellent results of long term patency. Intracranial angioplasty is in the process of evolution.

Key words -

**Brain,
Arteriovenous Malformations,
Aneurysms,
Angioplasty,
Intervention**

Stroke is one of the most important causes for morbidity and mortality due to ICH and cerebral ischaemia. Interventional techniques have contributed much to reduce the frequency and severity of the adversities of stroke.

Cerbral vascular malformation

Cerbral vascular malformation account for nearly 7% of patients who have died due to cerebral haemorrhage [1] of the various types of brain AVMs (BAVMs), the ones more pertinent to interventional treatment is arteriovenous malformations (AVM), which have the highest propensity bleed. The natural history of BAVMs has been related to size and topography [2], the age and sex of the patients and the location of the lesions, the presence and type of venous obstruction and associated

arterial aneurysms. The clinical presentation of BAVMs include haemorrhage, seizures and altered cerebral function. While it is clear that BAVMs need to be treated, incidental discovery of a BAVM is not an indication for treatment [1]. Traditional attempts at such studies regarding treatment indications did not consider the value of endovascular techniques and embolization [3].

Currently, the need for treatment is based on the demonstration of evidence of weakness in the angioarchitecture, age of the patient, location of the lesion, associated arterial aneurysm in the feeding pedicle or in the nidus, evidence of venous thrombosis, venous obstructions, hypertension or venous pseudoaneurysms.

The objective of any treatment of a patient with BAVM is to completely and permanently exclude the lesion from circulation. The indications for interventional treatment in BAVMs are determined by comparing the risks of the treatment in BAVMs to the natural history of the individual lesion. Endovascular obliteration of a BAVM if it is to be curative, a permanent nonbiodegradable agent is used to force a cast of the pathological angioarchitecture [4]. Particulate or resorbable agents have no place in the complete cure. Potential recruitment of arterial collaterals may reperfuse the BAVM. In general, when there is complete obliteration of the nidus of the malformation without stasis in the nidus cure is complete. Complete obliteration of BAVMs by embolization has ranged from 10% - 18% [5], [6]. The differences are related to the referral bias of the patient groups. The low cure rate is due to the fact that most patients currently referred for embolization have very large lesions with supply from multiple arterial territories. However, the success rate in single pedicle lesions is close to 95%.

Pre-operative embolization has gained in popularity as a surgical adjunct to AVM excision [7], [8]. The aim is to decrease bleeding, shorten operating time and minimise the risk of normal perfusion pressure break through. Various materials used for preoperation embolization include silicone spheres, polyvinyl alcohol particles, silk or a combination of collagen and alcohol mixtures [8], [9]. Newer liquid polymers like N-Butyl CyanoAcrylate (NBCA) has been used preoperatively in some centres [9]. Embolization of the margins of the malformation first has been found to assist in demarcating the plane of dissection as well as reducing the cortical venous hypertension facilitating dissection.

The Intervention treatment of Brain AVMs started with the use of free, flow directed particulate agents; primarily, silicone spheres, duramater, autologous blood clots and muscle tissue. They were however ineffective in reaching the nidus and more often occluded the major pedicle very proximally [7], [8]. Though Kerber developed the calibrated leak balloon in 1976 [10], it was, unreliable. Acrylics like Isobutyl cyanoacrylate (IBCA) developed in 1980 revolutionised the treatment of BAVMs. The polymerisation time was too short, however. The addition of Iophendylate to lengthen the polymeration time improved the cure rate. Simultaneous development of latex calibrated leak balloon [1] further improved the technical ease and safety. Further, development of variable stiffness microcatheters and steerable micro guidewires was a major breakthrough in technical advancement with the new ability to reach the nidus of an AVM further enhancing the efficacy and safety of the procedure (figure. 1).

Right Carotid Angiogram (A & B) shows AVM in the right temporo parietal region (straight arrow) and draining vein (curved arrow). Plain Radiograph (C) shows NBCA glue in the nidus of the malformation. Post intervention angiogram (D) shows complete obliteration of the malformation.

Initially the complications were rather high, however, increasing experience have improved the safety of these procedures tremendously. While the mortality in large AVMs ranged from 10-20% in the 70's, the current figures for mortality are 1.5 to 5% morbidity are similar to those with surgery. This is better

than the cumulative risk of 1.6% mortality and 1.4% severe morbidity in untreated patients [1]. Currently embolization as the sole form of treatment is done in patients with large malformation, malformation in inaccessible areas and in patients unfit for surgery. Partial embolization is indicated in patients prior to radiosurgery.

Intracranial aneurysm

Surgical exploration and clipping have remained the treatment of choice in the majority of intracranial aneurysms. However, since two decades, endovascular treatment of intracranial aneurysms has progressed much since the first such description of treatment of such aneurysms by Serbinenko from Russia in 1974 [11]. This has been basically due to recent advances in high resolution angiography, microballoon technology and polymers. Debrun et al in 1981 [12] and Berenstein et al in 1984 [13] described carotid occlusion for giant inoperable aneurysms of the detachable balloons by occlusion of the parent artery and demonstrated that proximal occlusion of the parent artery may be performed with acceptable safety (Figures. 2, 3).

Right Carotid Angiogram / Projection (A) shows an aneurysm in the right carotid siphon (arrow head). Plain radiograph (B) shows a balloon inflated with contrast medium in the ICA (arrow). Left Carotid Angiogram (C) and Right Vertebral Angiogram (D) shows collateral filling of the right ICA circulation
Selective Left Internal Carotid Angiogram (A) shows an aneurysm in the cavernous part of the ICA (white arrow). oblique view (B) shows the aneurysm (black arrow). Selective left carotid angiogram (C & D) shows obliteration of the aneurysm with radio-opaque balloon filled with contrast medium and total preservation of the carotid circulation

However, Romodanov and Shcheglov while describing their experience of 119 cases, reported preservation of the parent artery in nearly 80% of their patients and later upto 91% in a much larger series of 614 patients [14]. Hilal in 1988 reported the use of metallic coils for the first time and later Casaco et al [15] reported the use of platinum micro coils detached into aneurysm sac by wires in 71 patients with acute subarachnoid haemorrhage with complete obliteration in 84.5% and more than 90% obliteration in the rest. Of the 8 deaths in their series, 6 patients were in Hunt and Hess grades III and IV [15]. This set to rest, the doubts regarding the role of the coil embolization in the treatment of patients presenting in the acute setting with SAH. Further improvement occurred with the introduction of Guglilemi Detachable (GDC) coils which are detached electrolytically [16].

Available early data with the use of GDC coil embolization is extremely encouraging with further enhancement of the success of endovascular occlusion of intracranial aneurysms [17], [18]. While there are no described complications due to coil embolization, follow up angiograms have shown persistence in some and enlargement in a few of the incompletely treated (> 90%) aneurysms with a small residual neck [19]. The current indications for endovascular therapy include all aneurysms which are primarily surgical unclippable either due to the location of the aneurysms or inability of the patient to undergo surgery. Patients with SAH in poor clinical grade have embolised in the acute setting with follow up surgery at a later date [20]. Saccular aneurysms with a broad neck and fusiform aneurysms are still a problem to treat by the endovascular route, though initial results in the swine are promising [21].

P.T.A

Percutaneous transluminal angioplasty (PTA) is an established method of treatment of coronary, femoral, renal and other arterial stenosis. It has been recently applied to the treatment of stenotic disease of the supraaortic arteries [22]. It has not become a routine therapeutic modality due to the danger of distal embolization of atherosclerotic debris causing inadvertent occlusion of perforating branches originating from the middle cerebral artery or basilar artery when the plaque is compressed. None the less, it is beyond doubt that patients with ischaemia in the territory of the anterior circulation including completed stroke with haemodynamic compromise will benefit from PTA.

The technique of percutaneous interventional transluminal angioplasty started with the introduction of the dilatation balloon by Gruntzig [23]. The initial results of the dilatation procedures on the supraaortic segments of the arterial tree showed success rates of 94% to 97% including the subclavian and the common carotid arteries and 100% of the innominate arteries [22], [24], with no mortality and less than 0.5% morbidity.

Published reports suggest that PTA is the method of choice for treatment of symptomatic treatment of the subclavian artery. Current studies indicate that almost all stenotic lesions of the vertebral artery (> 95%) are smooth, circular and free of ulceration and therefore suitable for PTA [22]. Results of large series on angioplasty of the vertebral arteries for vertebrobasilar insufficiency have shown > 95% successful dilatation with no major and approximately 4% minor complication rates. The long term results of 80-90% cure or improvement suggest PTA as the method of choice for symptomatic vertebral artery stenosis [25]. The application of PTA to the anterior circulation is however still controversial. However, many studies have shown encouraging results, with success rate of 93-95% and a complication rate of major neurological deficits of 1-2% as compared to none in the vertebral artery territory [22]. Most of the advances were due to the introduction of the triple coaxial catheter system by Theron et al designed to protect the anterior cerebral circulation from distal embolization of thrombus during PTA. Further advances in microcatheter systems and microballoon technology carried the PTA procedure to intracranial circulation. The first case of MCA dilatation of an atherosclerotic lesion was by Purdy et al [26]. Initial results of intracranial PTA have shown a success rate of nearly 85% in patients with atherosclerotic stenosis of > 70% of the MCA with a 26% 2 year stroke rate despite medical therapy [27]. Currently, PTA is recommended in symptomatic haemodynamically significant stenosis, morphologically circular and smooth with no evidence of ulceration, heavy calcification or thrombotic deposits, after exclusion of fresh infarction.

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