Case report

PYOGENIC CEREBRAL ABSCESSE WITH DISCHARGING SINUS COMPLICATING AN EMBOLIZED ARTERIOVENOUS MALFORMATION

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SUMMARY

Brain arteriovenous malformations (AVM) are treated with endovascular embolization either as a definitive treatment or as an adjunct to surgery or stereotactic radiotherapy. Complications of AVM embolization are well known but infection of the embolised AVM nidus is extremely rare. On Pubmed search we found only a single case report of an infected brain AVM after embolization. We report a case of pyogenic cerebral abscess with superior sagittal sinus extension complicating an embolised AVM and discuss the possible etiopathogenesis.

Key words: arteriovenous malformations, cerebral, arteriovenous malformations, therapeutic embolization, sinuses, superior sagittal.

RéSUMÉ

Abcès cérébral à pyogènes étendu au sinus sagittal supérieur compliquant l'embolisation d'une malformation artério-veineuse

L'embolisation est utilisée pour le traitement des malformations artério-veineuses (MAV) soit de façon isolée soit en association avec la chirurgie ou la radiothérapie stéréotaxique. Les complications de l'embolisation sont bien connues mais l'infection du nidus embolisé est extrêmement rare. Une recherche sur Pubmed nous a permis de retrouver une seule observation d'infection cérébrale après embolisation d’une MAV. Nous rapportons un cas d’abcès cérébral à pyogènes avec extension au sinus sagittal supérieur survenu après embolisation d’une MAV cérébrale et discutons la possible étiopathogénie.

Mots-clés: malformation artério-veineuse infectée, embolisation, discharging sinus, angiographie.

Embolization of arteriovenous malformations (AVM) is viewed as a curative procedure or a useful adjunct in the surgical management of cerebral AVM. The reported cure rate with embolization is up to 20% to 40% [12]. Consequently, in the recent years, the emphasis of treatment for cerebral AVM has shifted from surgical resection to endovascular embolization. The risks involved in endovascular embolization are nidus perforation, intracerebral bleeding etc. However, AVM nidus infections are considered a very rare complication. Transient bacteremia and nosocomial sepsis have been reported by various authors [6, 9]. Pyogenic hepatic and splenic abscesses have also been reported after therapeutic angiography [1]. After an extensive review of literature, we came across only one reported case of pyogenic parenchymal infection of the brain in an embolized AVM nidus [7].

We report a case of pyogenic infection of the embolised AVM nidus producing a sinus track to the scalp and discuss its possible etiopathogenesis.

CASE REPORT

A 30-year-old right-handed male was referred to our institution in May 2000 for the management of
NBCA. Gluing of the microcatheter occurred while embolising the last ACA feeder and the catheter got fragmented at the distal supple segment. The proximal portion of the broken catheter subsequently migrated into the right MCA (figure 3a et 3b). Attempts at retrieving the broken catheter fragment failed and the catheter was left in situ. Second embolization was uneventful. During the third sitting of embolization a portion of the glue migrated to the venous side at the torcula (figure 3a), which was successfully removed with a micro snare. Third sitting embolization resulted in more than 80% reduction in the nidus volume. The patient had no neurological deficits following the third sitting of embolization (August 2004), initial angiography revealed total occlusion of the SSS with retrograde flow in the cortical veins (figure 3a-c). The tiny feeders from both right ACA and MCA could not be catheterized. Embolization of dural supply from the right middle meningeal artery was performed with NBCA. The patient’s neurological condition was stable. He was kept on follow up.

Six months after the last embolization the patient noticed a sinus with pus discharge over the vertex. The patient presented at follow-up only 2 months later. On examination he was afebrile with no additional neurological deficits or features of raised intracranial pressure. A CT head (March 2005) revealed ring-enhancing lesions suggestive of abscess.
CEREBRAL ABSCESS COMPLICATING AN EMBOLIZATION

along with intensely enhancing AVM nidus in right fronto-parietal region with perilesional edema and mass effect (figure 3c). Bone widow setting showed defect in the skull vault at the site of draining sinus (figure 3d). An MRI was performed immediately. Conventional MR sequences, diffusion weighted imaging and proton MR spectroscopy better characterized the infective nature of the lesion involving the AVM nidus and adjacent brain parenchyma. There was abscess formation inside the superior sagittal sinus from which the discharging sinus was extending into the scalp (figure 4a-f). Residual AVM nidus was observed adjacent to the infected part of the nidus. Angiogram performed a few days later demonstrated the persistent small nidus with occluded SSS (figure 5d-f). Cultures taken from the discharging sinus revealed Pseudomonas aeruginosa sensitive to amikacin and cefotaxime. The patient was being

FIG. 3. – a) Lateral view of the skull shows the glue cast along with broken catheters in ACA (solid arrow) and MCA (arrow). Also note glue in the region of torcula (black arrow). b) CT head with bone window setting at the level of suprasellar cistern shows the broken catheter with glue appearing as hyperdense structures in the region of the right MCA (arrow). c) CT head in coronal plane with bone window setting shows the defect in the skull vault from the draining sinus (solid arrow). Note part of the glue cast lying beneath the bony defect (arrow). d) Contrast enhanced CT head shows smooth rim enhancement from the abscess (solid arrow) with associated hypodense edema. Note the presence of glue in the nidus from previous embolization (arrow).


FIG. 4. – a and b) AP view of the arterial and capillary phases respectively of right ICA injection shows the residual nidus from fourth sitting of embolization. c) Lateral view of the venous phase of right ICA injection during the fourth sitting of embolization shows the occluded SSS (arrowhead). The nidus is draining mainly through cortical veins into vein of Labbe and then into the right transverse sinus (arrow).

treated on high dose antibiotics after neurosurgical consultation for 6 weeks as a presurgical measure for abscess evacuation.

**DISCUSSION**

Brain AVMs most commonly present with intracerebral hemorrhage. The next common presentation is seizure. AVMs can also result in focal neurological deficits [2, 8]. Due to the high morbidity and mortality associated with brain AVM from hemorrhage, treatment is generally recommended. Surgical excision is the treatment of choice for the majority of AVMs. Other important modes of treatment include endovascular embolization and stereotactic radiosurgery. Embolization is curative for small AVMs and an adjunct to surgery or stereotactic radiotherapy for large AVMs. Deep-seated AVMs and those located in eloquent areas are often dealt with embolization or stereotactic radiotherapy alone or in combination [8, 10].

Embolization of brain AVMs is mainly performed with polymerizing agent like NBCA injected through flow-guided microcatheters. Alternatively other materials such as poly vinyl alcohol, Onyx, absolute alcohol etc. can be used to embolise AVMs [8]. Complications associated with embolization of AVM
include perforation of vessel, intracerebral hemorrhage, normal perfusion pressure breakthrough, non-target embolization, and migration of the glue to the venous side [11]. Complications specific to the use of polymerizing agent is gluing of microcatheter [3].

Infection of a brain AVM as a complication of embolization is extremely rare. There is only one report of infected brain AVM following embolization in the literature [7]. Mourier et al. reported an infected brain AVM in a 24-year-old patient following embolization. This patient had a right frontal AVM and underwent two sittings of embolization. She presented with features of raised intracranial pressure 4 months after embolization and imaging showed multiple abscesses in the region of the embolised AVM, which was confirmed by stereotactic aspiration biopsy. The offending organism in that case was Staphylococcus aureus and they attributed the source of infection possibly to the handling of the microcatheter or glue. Finally the AVM had to be excised due to inadequate response to antibiotic therapy [7].

As reported by Mourier et al., we also observed delayed onset of infection. In spite of all aseptic precautions through out his treatment we observed infection developing in the AVM eight months after the last embolization.

Brain abscess occurs as a consequence of extension of infection either by contiguous spread or hematogenous spread from an infective focus. The mode of infection often determines the causative pathogen causing brain abscess [5]. Pseudomonas aeruginosa is a gram-negative bacillus and is a well-known cause of central nervous system nosocomial infections in patients with head injury, neurosurgical procedures and long term debilitating diseases [4]. In our patient, this pathogen most likely has been carried to the site of infection through catheters or glue. Colonization of the embolised AVM could have occurred through the arterial route during the procedure or when attempts were made to retrieve the catheter. There after the infection must have spread in the nidus and then to the superior sagittal sinus. The discharging sinus in our case perhaps had developed from the extension of the infection from SSS into an emissary vein to the scalp. Alternatively transient asymptomatic bacteremia could have led to the infection of the nidus. Presence of broken catheter could be a facilitating factor for development of abscess in this case. The initial focus of infection in our case might have been in the thrombosed superior sagittal sinus from which it could have spread to the embolised nidus via the draining veins. The third possible route of infection being the hematogenous route during the embolization of the dural branches in the last sitting. Infection may have occurred in the scalp and then subsequently progressed to the SSS via an emissary vein and then to the AVM nidus.

Development of brain abscess at the site of embolised AVM nidus is an extremely rare event. Extreme care should be taken to ensure proper asepsis during embolization. Broken catheters or other foreign bodies left in situ may act as a nidus for development of abscess.

REFERENCES


Analyses de livres

**NeuroPET**

PET in Neuroscience and Clinical Neurology
K. Herholz, P. Herscovitch, W.D. Heiss
Springer Verlag Berlin Heidelberg 2005
297 pages (1 volume + 1 CD)

La place grandissante des explorations par TEP en pathologie du système nerveux explique la nécessité d’une mise au point sur ce sujet. L’ouvrage de K Herholz, Peter Herscovitch et Wolf-Dieter Heiss répond tout à fait à cette nécessité. Le livre est divisé en trois grandes parties. La première a une orientation clinique et envisage successivement les démences et troubles de la mémoire, les mouvements anormaux, les pathologies tumorale et vasculaire, l’épilepsie et les pathologies psychiatriques. La deuxième partie a une fonctionnelle orientée et traite de la barrière hémosto-encéphalique, de métabolisme cérébral, de transports protéiques, d’imagerie fœtale. La place réservée aux illustrations est majeure et leurs légendes d’une grande clarté. Cet atlas sera très certainement d’une grande utilité à tous les médecins, cliniciens ou spécialistes de l’imagerie, amenés à prendre en charge les affections du système nerveux de l’enfant.

**Vertebroplasty and Kyphoplasty**

D.K. Resnick, S.R. Garfin
Thieme Medical Publishers 2005
(138 pages)

La morbidité rattachée aux fractures vertébrales est importante chez le sujet âgé et le traitement de telles lésions associe traditionnellement immobilisation et antalgiques. Au cours de ces dernières années, le recours aux techniques mini-invasives, vertébroplastie et plus récemment kyphoplastie, s’est considérablement accru. L’effet antalgique quasi-immédiat obtenu, en particulier en cas de tassement ostéoporotique explique en partie l’intérêt porté à ces techniques. De nombreuses questions restent actuellement en suspens et c’est le mérite de Daniel K. Resnick, Steven R Garfin et de leurs collaborateurs de participer avec beaucoup de pertinence à ces débats. Chirurgiens orthopédiques, neurochirurgiens et radiologues interventionnels sont en effet associés dans cet ouvrage pour discuter de sujets aussi importants que la place de la vertébroplastie et de la kyphoplastie dans le traitement des fractures à la phase aiguë et celui des fractures-tassements pathologiques, les avantages et inconvénients respectifs de ces deux techniques, la sélection des patients ou la stabilisation rachidienne. Ce livre manquait ; il sera une référence d’une grande utilité pour tout spécialiste prenant en charge ces fréquentes pathologies.