Management of Intraprocedural Adverse Events during coil embolization of cerebral aneurysms: A Clinical Case Series Review.

A Gordhan

Citation


Abstract

The overall complication rate for endovascular coil embolization of cerebral aneurysms is 5% percent. This is an illustrative case series that describes management aspects and probable precipitants of adverse events during coil embolization and related imaging and clinical outcomes. Major intraprocedural intracranial adverse events during endovascular coil embolization of cerebral aneurysms can be divided into three categories. Thromboembolic, aneurysm or parent vessel perforations/ruptures and embolysate related. Clinical case examples with rescue intervention and outcomes are discussed within each category.

1) Thromboembolic (Case 1 and 2):
This is the most frequent complication and cause of morbidity and mortality. Thromboembolic may occur consequent to failure of sustained and or sufficient systemic anticoagulation. Prevention of localized platelet and clotting cascade activation in the context of thrombogenic device introduction for coil embolization is vital in preventing partial or complete parent, branch or distal vessel occlusion. Embolic propagation of thrombus may arise from native vessel disease and or consequent to mechanical device manipulation during coil delivery and deployment, as well as with the use of adjuncts such as balloon assist.

Frequent monitoring of ACT (Activated Clotting Time) values for systemic anticoagulation with Heparin to confirm twice the baseline value is the primary preventative method. Intraprocedural pharmacologic platelet thrombus dissolution with short and long acting Glycoprotein IIBIIIA inhibitors are effective when administered locally or systemically. Raising cerebral perfusion pressure may augment collateral flow with consequent reduction in the risk of ischemic sequelae.

Intra arterial or peripheral venous administration of GPIIBIIIA inhibitors upon identification of intraluminal thrombus may prevent progression to complete vascular occlusion. Minimal thrombus formation at the coil/parent vessel interface is often observed. Eptifibatide has a shorter half life (2-4 hours) and is advantageous over Abciximab in ruptured aneurysm that potentially require post procedural placement of a ventriculostomy. A fluid bolus followed by appropriate pharmacologic induction of increased blood pressure allows for augmentation of collateral vasculature. Vessel caliber change or limitation of flow may be consequent to vasospasm. This can be addressed by localized intra arterial calcium channel blocker administration.

Figure 1
2) Perforations and ruptures (Case 2 and 3): Intraprocedural aneurysm rupture occurs in approximately 2-3% of cases with an overall preprocedural mortality of 25-50%. They are more likely to occur in small aneurysm and aneurysms that have already ruptured. Aneurysm rupture caused by coil or microcatheter perforation may result in less morbidity and mortality than perforation from a microguide wire. Intracranial vessel injury such as dissection or microguide wire distal branch vessel perforation are also other events included in this category.

Reversal of systemic anticoagulation and expedient continued coil embolization with measures to control raised intracranial pressure that includes preprocedural or timely placement of ventricular drainage catheters are important in managing intraprocedural aneurysm rupture. Judicious monitoring of distal microguide wire tip location is critical in preventing inadvertent distal vessel perforation.

This occurs with greater frequency in ruptured aneurysms. Size and location are additional determinants. A microcatheter tip in the subarachnoid space should not be retrieved. Continued complete coil embolization of the ruptured aneurysm with termination and reversal of systemic anticoagulation are the key initial steps. Measures to control elevated intracranial pressure, including placement of an external ventricular drainage catheter is critical. Intravenous administration of mannitol and patient hyperventilation as well pharmacologic lowering of blood pressure are useful adjuncts. Poor outcomes are related to posterior circulation ruptures and angiographic evidence of elevated intracranial pressure.

3) Embolysate related (Case 5 and 6): Coil unraveling and migration with or without fracture occurs in less than 2% of cases. This often occurs with placement of the finishing series of coils. Early or delayed coil loop herniations into the parent vessel are precipitants of extraaneurysmal thrombogenesis and may be influenced by the type of coil configuration deployed and aneurysm aspect ratios.

Coil loop unraveling and migration is managed with mechanical or surgical coil retrieval. Coil loop herniations can be approached conservatively or treated with loop incarceration by stent implantation.

Coil unrevealing, is defined as the loss of synchronous coil response to the delivery wire. This occurs when an attempt is made at removing a partially implanted coil that has an incarcerated intra-aneurysmal distal end. This can be avoided by finishing with stretch resistant multiple shorter length coils instead of a single long coil. Removal of microcatheter tension before coil retraction prevents an acute orientation of the coil which may precipitate unrevealing. An elongated
unrevealed coil can be tacked to the common femoral artery. Prolapse of a free end pulsating upstream can be fixated with stent.

**Figure 5**

**CASE 5**

**Clinical History:** 49 year old female. Hunt and Hess 1. Fischer grade 2 subarachnoid hemorrhage, 5 mm carotid terminus aneurysm (Fig 1). Intraprocedural Event: Balloon coil support embolization with unevenly detached first coil (Fig 2). 3D omega loop coil herniation into parent vessel after deployment of second coil (Fig 3). Rescue: Repositioning of coil into the aneurysm lumen with balloon support (Fig 4). Non occlusive thrombus at parent vessel aneurysm neck interface (Fig 5). Post intra-arterial Eptifibatide bolus dose for complete clot dissolution. Final catheter angiography (Fig 6).

**Outcome:** No clinical neurologic deficit. Complete aneurysm occlusion at 18 month follow up.

**Figure 6**

**CASE 6**

**Clinical History:** 51 year old female with unruptured left 7mm superior cerebellar artery junction aneurysm \( (\text{Fig 1}) \) : Catheter angiography \( (\text{Fig 2}) \) : 2d catheter angiography.

**Intraprocedural Event:** Finishing coil coil embozation (Fig 3). No intra luminal thrombus (Fig 3).

**Rescue:** Neuroform stent implantation with jailing of herniated looped coil (Fig 4). No intraluminal thrombus of local parent vessel injury (Fig 5).

**Outcome:** No flow limiting in-stent stenosis left posterior cerebral artery (Fig 6): No clinical sequelae.

**Summary:** Each of the adverse events described result in or may progress on to either hemorrhagic or ischemic consequence to the brain with or without resultant clinical neurologic sequelae. Anticipation, early recognition and appropriate management of adverse events during cerebral aneurysm coil embolization are the most crucial determinant of patient morbidity and mortality.

**References**


Author Information

Ajeet Gordhan, MD
Bloomington Radiology, Department of Neurointerventional Radiology. OSF St. Joseph Medical Center, Bloomington Illinois.