Dural Arteriovenous Fistula following Cerebral Venous Sinus Thrombosis

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A 45 year old previously healthy, non-diabetic, non-hypertensive female presented with history of severe headache and vomiting in August 2011. She was not on any medications. She was conscious, oriented, pupils 3mm, equally reacting to light. Fundus showed bilateral papilloedema. No focal neurological deficits noted. CT brain showed cortical venous thrombosis. She was treated and discharged with oral anticoagulants. In April 2012, she again presented with vertigo and headache with bilateral papilloedema. MR Venogram (coronal view) showed thrombosis of left transverse sinus (Figure 1). MR Venogram (sagittal view) showed thrombosis of straight sinus (Figure 2). Blood investigations including protein C and S, Antithrombin-III, Antiphospholipid antibody and serum Homocysteine were normal. Anticoagulants and antiepileptics were continued. In August 2014, she presented with recurrent episodes of seizures with papilloedema. Digital subtraction angiography (DSA) was done. Right external carotid angiogram (ECA) AP view showed transverse sinus dural AV fistula. The diagnosis of dural arteriovenous fistula Cognard classification Type II (a+b) following cerebral venous thrombosis was made and endovascular surgery planned. But the patient refused due to financial constraints. She developed recurrent seizures which was refractory to treatment and expired.

Dural AV fistula constitutes 10-15% of intracranial arteriovenous malformations. It is more common in females. Symptoms develop during middle to late adulthood. Initiating events include trauma, infection, recent surgery and dural sinus thrombosis. The commonest predisposing factor is venous sinus thrombosis. It is formed by the opening up of microvascular connections within the dura following venous hypertension. Unless intervened, these channels become hypertrophied resulting in direct shunting between arteries and veins. The fistula gets pial blood supply from parenchymal vessels leading to an angiomatous network formation within the partially recanalised sinus. Thus dural sinus receives arterialized bloodflow causing mechanical obstruction of the sinus resulting in retrograde drainage of blood from the sinus to the cortical veins. They can be classified based on the type of venous drainage as Type I (drainage into a dural sinus, with normal antegrade flow), type II (drainage into a dural sinus, with reflux into II a: other sinuses, II b: cortical veins, II a+b: sinuses + cortical veins), Type III (drainage into cortical veins).
veins), type IV (drainage into cortical veins with cortical ectasia) or type V (drainage into spinal perimedullary veins). Risk of bleed is 40% and 65% respectively in type III and IV. Type I and II(a) are benign whereas the others follow an aggressive course if untreated and carries an 8.1% annual risk of hemorrhage. Endovascular therapy like embolization, coiling via transvenous route and stenting are among the first-line treatment for dural AV fistulas.

This case is being presented as it highlights a rare vascular anomaly following cerebral venous thrombosis.

References

