Dear Editor,

Mania secondary to brain lesion is common, but is mostly associated with tumor, stroke and head injury (Cummings, 1997). Vascular malformations, however, are rarely associated with secondary mania and when reported, they are usually related to the occurrence of mania post-resection (Benjamin et al., 2000). Only two cases of mania occurring with a coexisting vascular malformation have been reported; one case of pontine cavernous malformation and another case of frontal arteriovenous malformation (AVM) (Gross & Herridge, 1988; Yetimalar et al., 2007). We report a case that presented in a manic state, and was discovered to have a massive AVM. To our knowledge, this is only the second report describing secondary mania to an AVM.

A 54-year-old man presented with a 2-month history of irritability, reduced sleep, talkativeness, grandiosity, over-familiarity and spending money indiscriminately. Over the same period, he had intermittent generalized headaches. There was no significant past psychiatric history or substance use disorder. Neither was there any known family history of psychiatric illness. He was fully orientated on neurological examination with no motor or sensory deficits, except for equivocal plantar reflexes. The rest of the physical examination was unremarkable.

Computed tomography of the brain showed an extensive right parietal-temporal-occipital AVM with midline shift. Cerebral angiogram revealed a large AVM in the right temporoparietal region with multiple feeding vessels (Figure 1). A diagnosis of organic mood disorder (right cerebral AVM with secondary mania) was made and the patient was started on sodium valproate 400 mg bid and haloperidol 15 mg tds. The attending neurosurgeon added carbamazepine 400 mg bid as prophylaxis against possible seizures. The patient gradually improved over 2 weeks. For the AVM, only conservative management was planned due to the extensive involvement of brain tissue. After 3 months, the antipsychotics were gradually tapered off and the patient was maintained on sodium valproate 400 mg bid and haloperidol 15 mg tds. The patient has remained symptom-free for 16 months. We plan to maintain him on his current medication for the long-term, in view that he may have a high risk for further episodes as the AVM may grow in size.

An organic cause was suspected in our patient due to the late onset of symptoms, history of headaches, absence of any significant past psychiatric history or family history, and no substance misuse. The right-sided location of the lesion is in keeping with reports that support the role of right hemisphere involvement in secondary mania (Cummings, 1997). Though AVM are usually congenital, this patient only became symptomatic at a relatively late age. This could be explained by the fact that some AVM enlarge over time, and may need to reach a certain size before clinically manifesting themselves.

Brain lesions should be suspected when a patient presents with late onset mania, especially if signs, however subtle, suggest an organic cause.

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References


