SPONTANEOUS INTRACRANIAL HYPOTENSION CAUSING SINUS DURAL ARTERIOVENOUS FISTULA

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DAVF endovascular embolitation showing right trasverse DAVF elimination.

OBJECTIVE to describe the occurrence of sinus dural arteriovenous fistula (DAVF) following spontaneous intracranial hypotension (SIH).

CASE REPORT A 75 years-old man presented a orthostatic headache for 7 days. He was in warfarin therapy for recurrent venous thrombosis of the lower limbs. Hypercoagulative states laboratory tests were negative. Brain MRI angiography showed diffuse pachymeningeal enhancement, brain sagging, typical finding of SIH (Fig. A), bilateral subdural haematomas and a probable right trasverse DAVF (Fig. B), that was confirmed by cerebral angiography (CA) (Fig. C). Spinal MRI and CSF opening pressure were normal. The warfarin therapy was discontinued to avoid the risk of further bleeding resulting in hematoma enlargement. After 12 days he presented a comatose status (GCS=5) probably by venous hypertension and urgent endovascular embolitation of DAVF with Onyx embolic material was performed with awakening and headache disappeared. After 38 days he had nonpostural headache with imbalance and left hand weakness. Brain MRI showed right subdural haematoma enlarged, right cortical fronto-parietal subarachnoid haemorrhage (Fig. D) and disappearance of DAVF. CA showed a parietal cortical venous thrombosis. Lumbar epidural blood patch was performed. Within 3 months he became asymptomatic. Brain MRI was normal after 8 months (Fig. E) and CA showed the total elimination of DAFV after 12 months (Fig. F-G). The patient was asymptomatic 24 months later.

DISCUSSION Our patient developed a typical orthostatic headache by SIH. Subdural hematomas were caused by bridging veins rupture secondary to veins traction by brain downward diplacement from SIH. SIH causes compensatory venous sinus dilatation due to the Monroe-Kellie doctrine. Thus it is theoretically possible that stasis in the venous system may predispose to venous thrombosis. It is known that venous thrombosis may predispose to formation of DAVF. Probably our patient did not have venous sinus thrombosis because he was in warfarin treatment. Therefore, it may be assumed that in our patient venous dilation alone has favored the formation of DAVF by opening embryonic arteriovenous connections. After the effective endovascular treatment of FADV, the patient presented a subaracnoid hemorrhage from cortical venous thrombosis probably caused by persistent SIH. The patient at that time was not taking warfarin. Epidural blood patch effective in the treatment of cortical subaracnoid hemorrhage caused by cortical venous thrombosis.



CONCLUSIONS To our knowledge, the patient described here is unique in the occurrence of subdural haematomas, DAVF, cortical venous thrombosis and cortical subaracnoid hemorrhage following SIH. DAFV expands the neuroradiological sprectum of SIH.

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