Fatal acute hemorrhagic venous infarction due to the thrombosis of the draining vein of a developmental venous abnormality

Infarctus veineux hémorragique massif par thrombose aiguë de la veine de drainage d’une anomalie veineuse de développement

A 59-year-old woman, complaining of mild headache for two days, suddenly developed complete motor impairment of her right leg and hypoesthesia of her right side.

Figure 1 Axial FLAIR: high signal of the left frontoparietal area; acute thrombosis of the draining collector is seen as iso-signal (large arrow), compared with flow voids within the developmental venous abnormality medullary veins (thin arrow).

Figure 2 DWI: increased ADC values in the involved parenchyma.

MRI showed high signal intensities on T2WIs on the left frontoparieto-rolandic area, with slightly increased ADC, and no haemorrhage. 3D T1WI post-contrast revealed a huge developmental venous abnormality (DVA) with acute thrombosis of a large collector draining in the longitudinal superior sinus. No associated cavernoma was found (Figs. 1—4).

Half an hour after MRI, the patient developed a partial seizure. Two hours after MRI, the patient became comatose and CT revealed a massive parenchymal haemorrhage in the infarcted area (Fig. 5). Despite anticoagulant therapy, neurological deterioration leads to death in a few hours from symptoms presentation.

DVAs are the most frequent cerebral vascular malformations, reported in about 2.5% of autopsies [1], and

Figure 3 Axial T2 GRE-Wis: low signal intensity of the acute thrombosis of the draining collector (A: arrow), without parenchymal haemorrhage (B).
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Figure 4  Axial 3DT1WI with injection (A): the acute clots are seen as iso-signal in the main collector (large arrow). Sagittally reconstructed MIP (B): typical caput medusa of the large developmental venous abnormality.

usually asymptomatic. They are composed of mature normal medullary veins, separated by normal brain parenchyma, draining into a single huge central collector which joins the normal venous circulation. DVAs are considered as a normal anatomic variant admitted to be due to arrest of neuronal migration during the gestation [2]. DVAs are an exclusive venous entity, without arteriovenous shunt. The role of DVAs in draining of normal brain parenchyma is important, and severe complications following their surgical removal have been reported [3].

Complications of DVAs are unfrequent but may be clinically severe. Spontaneous haemorrhage of DVA is rare [4] and should exclude the responsibility of a cavernoma associated to DVAs in about 25% of cases [2,4,5], the spontaneous venous thrombosis of the main collector which leads to acute venous infarcts, and ischemic infarcts is the most usual presentation [2,6]. Severe hemorrhagic infarctions, as in our case, are very rare [6] and, to our knowledge, no case of so dramatic aggravation and fatal issue has been reported yet. The mechanisms leading to the thrombosis of the collector of a DVA remain unclear, stenosis of the collector and chronic venous hypertension have been reported [4,6] but their responsibility in the occurrence of an acute venous thrombosis has not been clearly demonstrated.

3D T1WI post-contrast seems useful to demonstrate thrombosis of DVA.

Conflict of interest statement

None.

References

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