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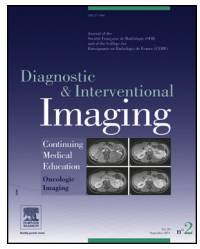

CONTINUING EDUCATION PROGRAM: FOCUS...

Imaging of cerebral venous thrombosis

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Abstract Cerebral venous thrombosis (CVT) is a potentially life-threatening emergency. The wide ranging of clinical symptoms makes the use of imaging in ''slices'' even more important for diagnosis. Both CT and MRI are used to diagnose the occlusion of a venous sinus, but MRI is superior to CT for detecting a clot in the cortical or deep veins. CT can show the hyperintense clot spontaneously and CT angiography the intraluminal defect. MRI also detects this thrombus, whose signal varies over time: in the acute phase, it is hypointense in T2*, whilst T1 and T2 can appear falsely reassuring; in the subacute phase, it is hyperintense on all sequences (T1, T2, FLAIR, T2*, diffusion). MRI easily shows the ischemic damage, even hemorrhagic, in the cerebral parenchyma in cases of CVT. Finally, imaging may reveal pathology at the origin of the CVT, such as a fracture of the skull, infection, tumor, dural fistula, or intracranial hypotension.

Cerebral venous thrombosis (CVT) is a rare and potentially life-threatening pathology. It is therefore an emergency that requires the rapid implementation of anticoagulant treatment, but whose diagnosis can be challenging. The range of clinical symptoms and the variability of MRI or CT findings may explain certain diagnostic errors. The radiologist therefore has three objectives: confirm the diagnosis by showing direct signs of occlusion of the venous structure by a clot, assess any damage to the cerebral parenchyma secondary to this thrombosis by looking for signs of venous cerebral ischemia and finally, attempt to demonstrate an origin or a pathology associated with this thrombosis. The aim of this article is to illustrate the various aspects of CVT on CT and MRI.

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Direct signs of venous occlusion

The thrombus

The occlusive thrombus itself may be detected on CT and MRI. Its appearance is directly linked to the time that has elapsed between the formation of the clot and imaging. Thus, when it is visible on a CT scan without contrast at the start of the disease, the clot appears as a spontaneous hyperintensity in the form of a venous structure, in general tubular in shape; this is the ''cord'' sign. This sign is visible in around 20 to 25% of cases [1] and disappears within 1 to 2 weeks [2]. However, spontaneous hyperintensity of the veins is not specific and may be seen in young patients with a high hematocrit, in cases of polycythemia, or in dehydrated patients [3]. In such conditions, the entire intracranial vascular network — both arterial and venous — will appear hyperintense.

MRI is more sensitive for revealing the clot due to the combination of sequences and high sensitivity to the magnetic susceptibility of blood degradation products, notably on gradient echo T2-weighted images (T2*) [4]. As with CT scan, the intensity of the signal of the clot at MRI depends on its age. Thus, in the first few days, during the deoxyhemoglobin stage, the clot appears isointense on T1-weighted images (WI) and hypointense on T2-WI and FLAIR, thus almost mimicking the normal venous flow signal and creating a potential for diagnostic error (Fig. 1). A venous MRA sequence, but also a T2*, improves diagnostic accuracy, since the thrombus is frankly hypointense, artefactual, on this last sequence. Later, during the 2nd week and the presence of methemoglobin, the clot appears hyperintense on all sequences, i.e. T1-WI, T2-WI, and FLAIR, but also in T2* and on diffusion-WI [5] (Fig. 2). It is important to know that dural venous sinuses with normal circulation have an opposite signal on FLAIR and T2*. In the event of thrombosis, they abnormally show the same signal intensity, irrespective of the timing of the MRI exam. In the chronic phase, the clot signal is very variable and depends on the degree of organization of the clot. It is typically isointense on T1-WI, iso-/hyperintense on T2-WI, and hypointense on T2*.

It is not always easy to see the clot on MRI, venography may therefore be necessary to confirm the diagnosis and visualize the extension of the venous occlusion (Fig. 3).

Venous occlusion

Between CT and MRI, there are numerous ways of performing a non-invasive venography [2,3]. To avoid flow problems, enhanced images are now recommended. CT angiography (Figs. 3 and 4) is a remarkable method for demonstrating the occlusion on raw slices (empty delta sign), on multiplanar reconstructions in thick slices (MIP), and even in 3D (volume rendering), notably for cortical veins [2]. There is no single acquisition protocol for venous CT angiography, but the latter should include an inframillimetric helical acquisition, from the vertex to the foramen magnum, around

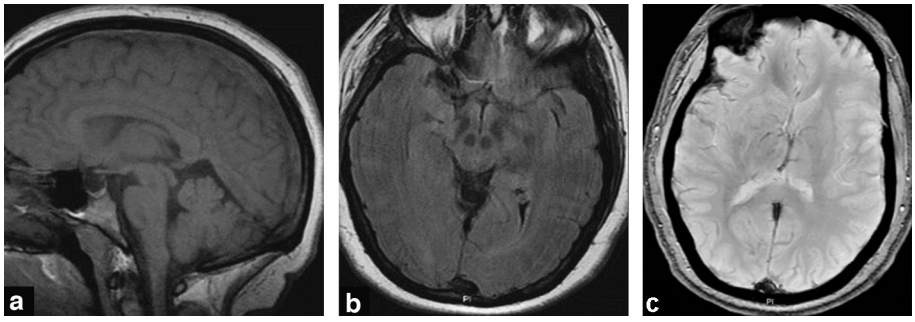

Figure 1. Acute venous thrombosis. (a) sagittal T1; (b) Axial FLAIR; (c) Axial T2*. Acute thrombosis may go unnoticed in T1 and FLAIR, as the sinus has a near-normal signal on these sequences. The axial T2 slice in gradient echo helps to narrow-down the diagnosis by showing an unusual frank hypointensity in the occluded venous structures, and notably the superior sagittal sinus. This hypointensity is artefactual, linked at this stage to the presence of deoxyhaemoglobin, and appears larger than the actual size of the sinus itself.

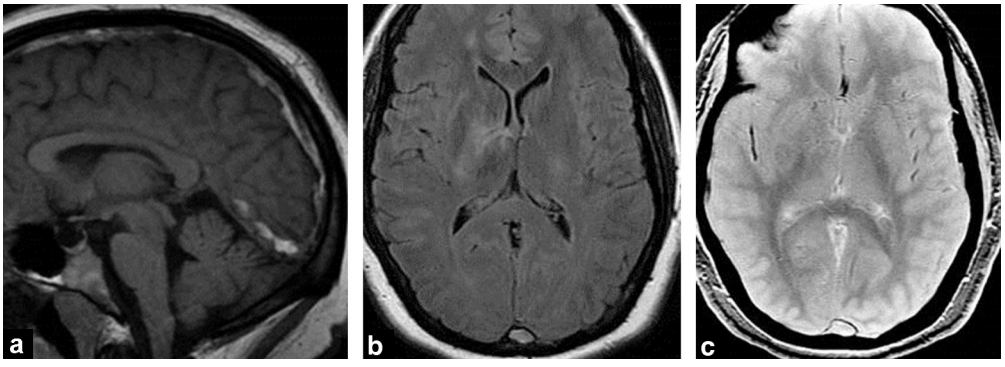

Figure 2. Evolution of the signal of the venous thrombosis at day 7 in the same patient as Fig. 1. (a) sagittal T1; (b) axial FLAIR; (c) Axial T2*. Due to the transformation in extracellular methaemoglobin, the clot is now clearly hyperintense on all of the sequences, including T2*. Thus, in the subacute stage, if the diagnosis does not pose any problems on T1 and FLAIR, T2* can be misleading as the signal of the clot is close to the normally circulating sinus. Note the appearance of capsulo-lenticular parenchymal suffering in FLAIR.