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Elderly patient with dural sinus thrombosis leading to abnormal medullary veins and cerebral venous infarctions

Alessandro D'Amore, MD; Giorgio Conte, MD; Adriano Viglianesi, MD; Emma Gabriella Muscoso, MD; Rita Chiaramonte, MD; Ignazio Chiaramonte, MD

We present a patient affected by a rapid neurological decline that was connected to subacute partial thrombosis of the straight sinus and associated with dilated superficial and deep medullary veins. An MRI scan confirmed signal alterations compatible with vascular disease and partial thrombosis of the straight sinus associated with dilated superficial and deep medullary veins of the cerebral white matter. Later, another MRI scan showed a replacement of the extensive white matter signal abnormalities, on FSE T2 and FLAIR sequences, by chronic vascular lesions.

Introduction

Cerebral dural sinus thrombosis, a quite uncommon disorder, can cause intracranial hypertension, venous infarction, or cerebral haemorrhage. Progressive thrombosis of dural sinus can obstruct cerebral venous outflow, causing dilation of the medullary veins, which are located within the cerebral and cerebellar white matter. The diameter of these vessels is less than 1 millimeter; they convey blood to the basal veins of Rosenthal, the cerebral internal veins, or cortical veins of the cerebral convexity.

Case report

A 75-year-old man was admitted to our hospital for progressive mental decline associated with headache, neurological bladder, and low sensitivity/paresthesia of the left facial region. Clinical examination showed an inability to walk, with bilateral increased osteotendon reflexes of the lower limbs. Blood analysis showed normal CBC; no signs of infections or autoimmune markers; aPTT, PT, and ET in the normal range; and homocysteine of 35 μmol/L (normal range < 13 μmol/L) and folate of 2.8 ng/ml (normal range 3-14 ng/ml).

The blood assays for these elements were repeated three times and showed no discrepancies. The EEG showed bilateral anomalies in the frontal region. An MRI scan was performed on a 1.5-tesla unit (GE, SIGNA excite HD), including the following sequences:

- Axial SE T1-weighted (TR, 520 ms; TE, 11 ms; 5-mm thickness; gap: 1.5 mm)
- Coronal and axial FSE T2-weighted (TR, 6080 ms; TE,103 ms; 5-mm thickness; gap, 1.5 mm; matrix, 512 x 512);
- Coronal FLAIR (TR, 8200 ms; TE, 108 ms; TI 2000 ms)
- Axial DWI (TR, 8000 ms; TE, 97 ms; 5-mm thickness; gap, 0)

The exam revealed diffuse signal alterations, hyperintense both on FSE T2-weighted (Figs. 1-2) and FLAIR sequences (Fig. 3) in the subcortical and periventricular fronto-parietal white matter, associated with acute vascular lesions (mainly on the left side). We also observed linear signal alterations with an oblique course from subcortical to periventricular areas, mainly identified in SE T1-contrast-

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Drs. D'Amore, Conte, and Viglianesi are in the Department of Radiology, Dr. Muscoso is in the Department of Neuroscience, Dr. Rita Chiramonte is in the Department of Otorhinolaryngology, and Dr. Ignazio Chiaramonte is in the Department of Neuroradiology, all at the University Hospital Catania, Catania Italy. Contact Dr. D'Amore at damorealessandro@virgilio.it.

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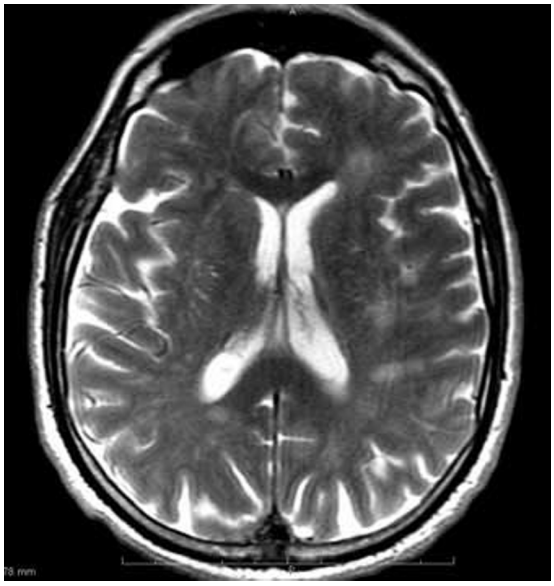

Figure 1. 75-year-old male with dural sinus thrombosis. Axial FSE T2 at the level of lateral ventricles shows diffuse hyperintensity signal of bilateral white matter of the frontal and parietal lobes. Some hyperintese areas near the middle cell and occipital horn of the left lateral ventricles are linked to vascular edema.

enhanced images. These alterations represented dilated medullary veins associated with vascular edema of cerebral white matter. DWI (1000b) confirmed the recent onset of the vascular injuries (Fig. 4).

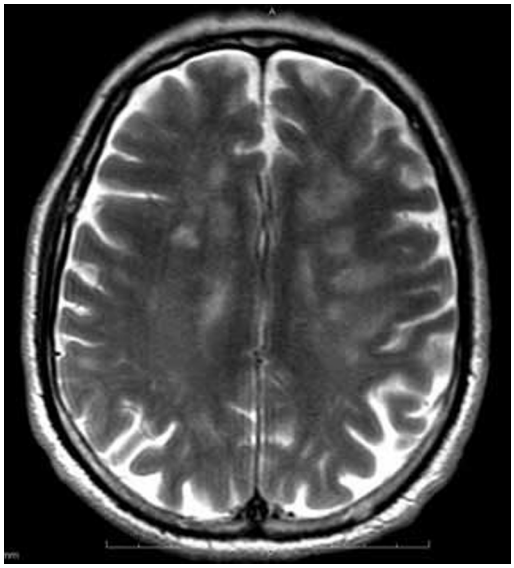

Figure 2. 75-year-old male with dural sinus thrombosis. Axial FSE T2 shows diffuse signal hyperintensity of supraventricolar and subcortical white matter. These are associated with many small and medium hyperintense areas compatible with vascular infarctions.

The SE T1-weighted sequence showed the presence of straight sinus heterogeneous signal alterations. In order to better characterize this radiological picture, we performed 2D-TOF and SE T1-weighted scans after intravenous

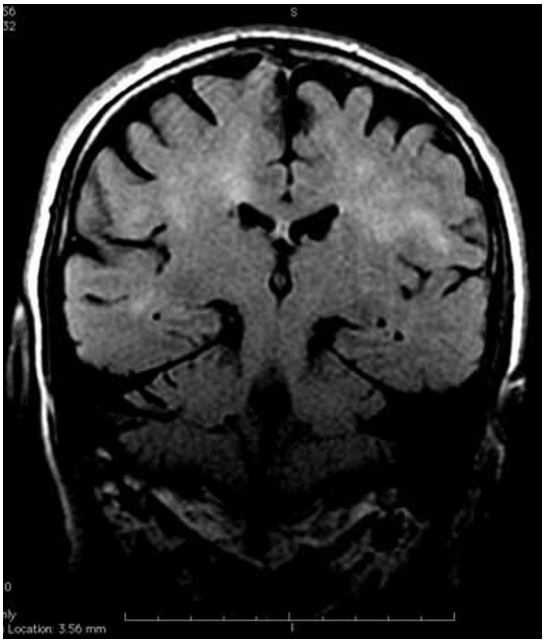

Figure 3. 75-year-old male with dural sinus thrombosis. Coronal FLAIR shows hyperintense signal of periventricular white matter, both in the frontal lobes and the right temporal lobe.

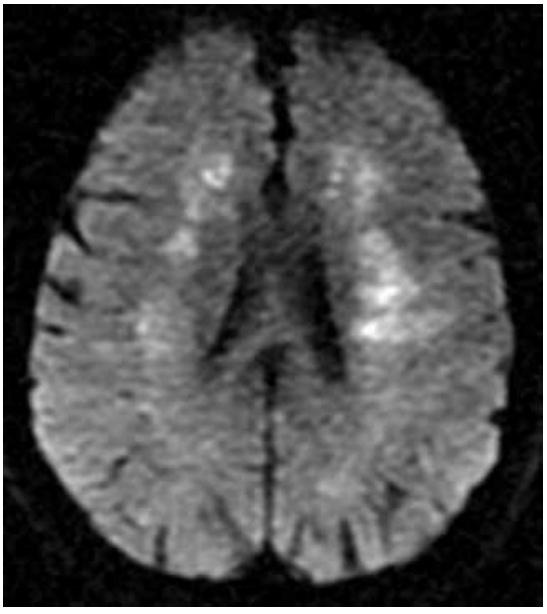

Figure 4. 75-year-old male with dural sinus thrombosis. Axial DWI (1000b) shows hyperintense areas, especially on the left frontal white matter, which are linked to acute vascular lesions.

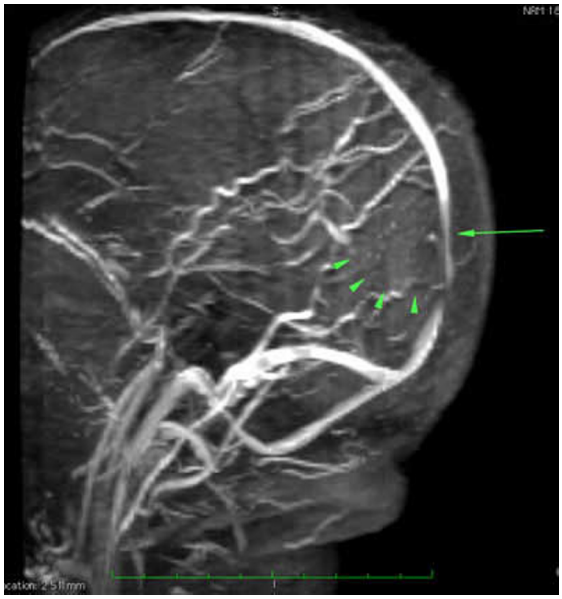

Figure 5. 75-year-old male with dural sinus thrombosis. 3D-MIP (reformatted by 2D-TOF) sequence confirms venous flow reduction of the straight sinus near torcular of Herofilo (arrowheads); a flow reduction is also present at the level of the sagittal superior sinus, near the venous sinus confluence (arrows).

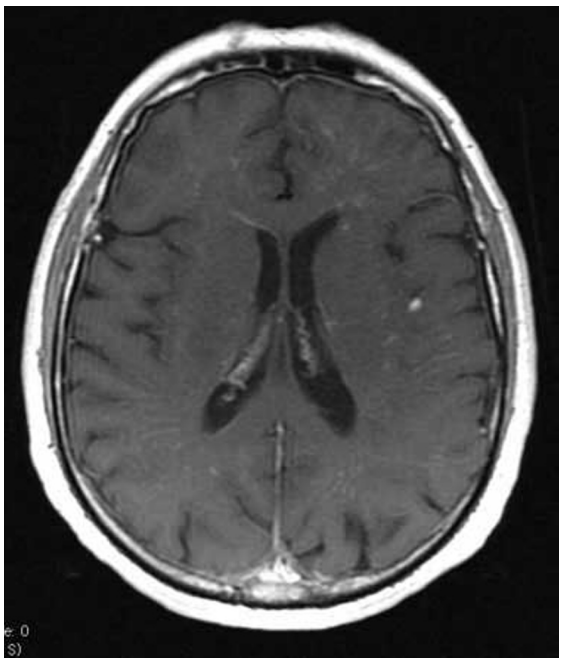

Figure 6. 75-year-old male with dural sinus thrombosis. Axial SE T1 contrast enhancement shows multiple vessels with an oblique course between the wall of lateral ventricles and the subcortical white matter. Note particularly a vessel that surrounds the right frontal horn. A small enhancement area at the level of insular cortex represents minor telangiectasia.

gadolinium injection. The 2D-TOF sequence (TR, 29 ms; TE, 5.1 ms; thickness, 1.6 mm; gap, 0.8 mm) and the following three-dimensional reconstruction showed a reduced venous flow in the straight sinus near the torcular herophilii and in the superior sagittal sinus near the confluence of sinuses (Fig. 5). The flow reduction was compatible with partial cerebral venous thrombosis.

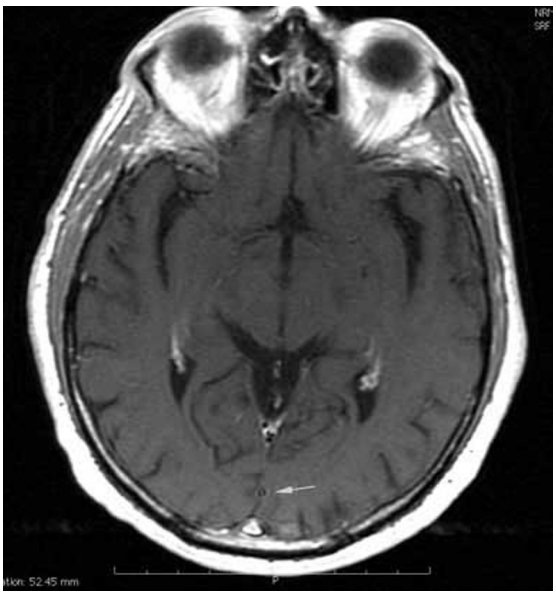

Figure 7. 75-year-old male with dural sinus thrombosis. Axial SE T1 contrast enhancement shows "threadlike" aspect of straight sinus (arrow); this is a direct sign of cerebral sinus thrombosis.

On the SE T1-weighted contrast-enhanced sequence (TR, 520 ms; TE, 11 ms; 5-mm thickness; gap, 1.5 mm), medullary veins were particularly evident and abnormally dilated (Fig. 6); this sequence also confirmed straight sinus partial thrombosis, which appeared "threadlike" (Fig. 7).

Our team proposed cerebral angiography, but the patient refused. Therefore, a MRI control was performed ten days later to evaluate the efficacy of medical therapy.

The patient received enoxaparin (4000 UI x 2) and mannitol (150 cl x 3) for two days. These were stopped and replaced by warfarin (5 mg/day) on the third day. The followup MRI examination showed a minimal reduction of the cerebral white matter signal alterations in FSE T2-weighted and FLAIR sequences, and a reduced medullary vein size, which appeared less visible after contrast administration than in the previous MRI scan. The 2D-TOF sequence did not disclose major changes. These findings were consistent with the improvement of patient's clinical conditions.

Six months later, the patient's clinical condition showed no changes. A new MRI scan was performed: the signal alterations of the cerebral white matter were increased in the FSE T2-weighted sequences (Fig. 8) and decreased in

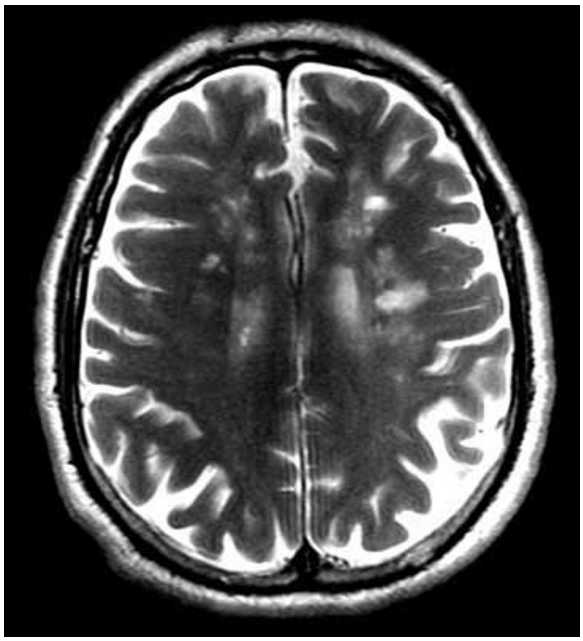

Figure 8. 75-year-old male with dural sinus thrombosis. Axial FSE T2 obtained after 6 months shows a bilateral increase of fronto-parietal white matter signal alterations, with some chronic lacunar infarcts.

Figure 9. 75-year-old male with dural sinus thrombosis. Axial FLAIR obtained after 6 months demonstrates a reduction of white matter signal alterations, with some chronic lacunar infarcts on the left frontal lobe.

the FLAIR sequence (Fig. 9). Those sequences also demonstrated the presence of many bilateral chronic residual lesions. DWI (1000b) did not show brain parenchyma signal alterations (Fig. 10). Abnormalities of deep medullary veins

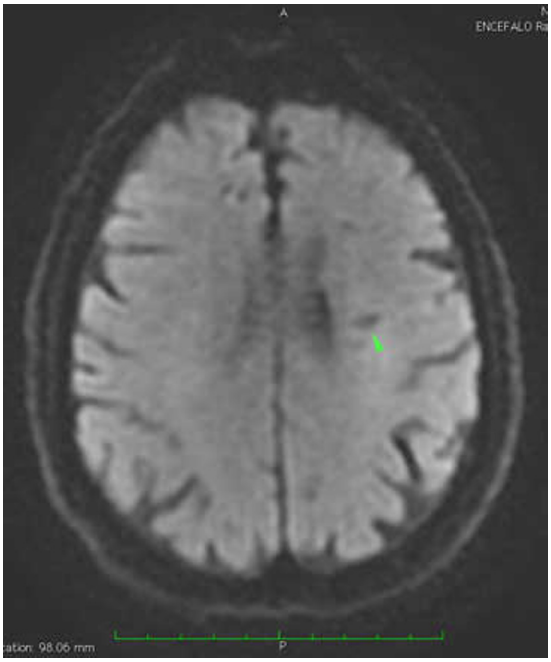

Figure 10. 75-year-old male with dural sinus thrombosis. Axial DWI (1000b) obtained after 6 months shows the sequelae of vascular injuries (arrowhead).

were no longer displayed in SE T1 contrast-enhanced images. In the 2D-TOF sequence, the straight sinus main-

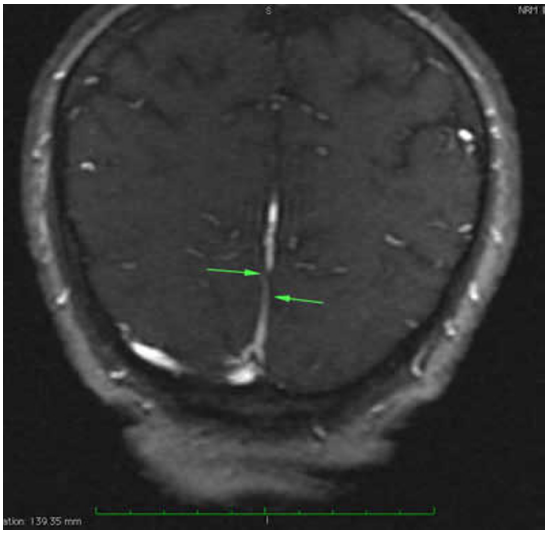

Figure 11. 75-year-old male with dural sinus thrombosis. Coronal 2D-TOF slice obtained after 6 months shows the "threadlike" aspect of straight sinus with minimum residual flow (arrows).

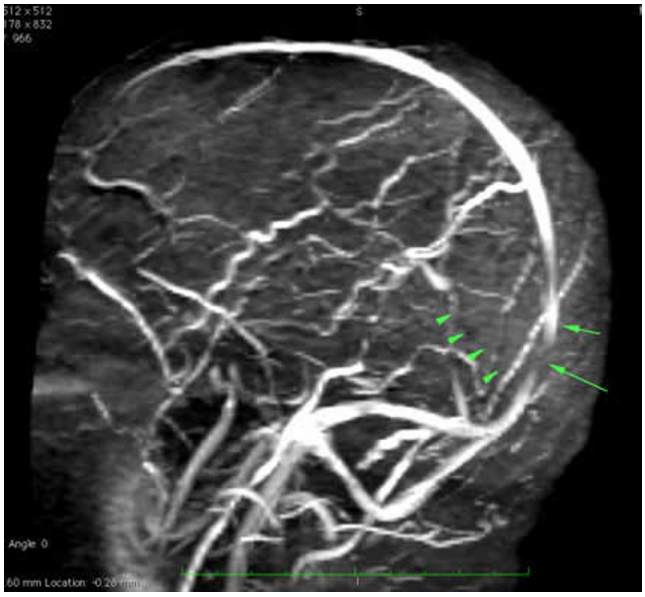

Figure 12. 75-year-old male with dural sinus thrombosis. 3D-MIP (reformatted by 2D-TOF) sequence obtained after 6 months does not show changes at the level of straight sinus (arrowheads); the superior sagittal sinus is now slightly more viewable (short arrow,) but there is still absence of flow near the confluence of the sinuses (long arrow).

tained its "threadlike" aspect (Fig. 11); in the 3D-MIP reconstruction of this sequence, the superior sagittal sinus was minimally more visible than in the previous examination (Fig. 12). A followup was scheduled for 1 year, assuming no clinical changes.

Discussion

In our case, there were many dilated superficial and deep medullary veins, associated with extensive edema of the subcortical and periventricular white matter. There also was a partial thrombosis of the straight sinus near the torcular herophilii, related to the abnormalities of medullary veins.

Medullary veins are divided into supratentorial and infratentorial systems. The supratentorial system provides two groups of veins. The superficial ones are short venous channels in the white matter, from 1 to 2 centimeters below the gray matter, and drain to the cortical surface; deep medullary veins, which are longer venous channels in the white matter just below the superficial group, drain toward the lateral ventricles (1). A third group, the transcerebral veins, also exists.

In the supratentorial group, superficial medullary veins join cortical or hemispheric veins, eventually draining into the superior sagittal sinus, while the supratentorial deep medullary veins converge to the anterolateral corner of the ventricular frontal horns, the head and body of the caudate nucleus or mid-body of the lateral ventricles, the temporal horns, the trigones, and the occipital horns. Developmental venous anomalies (DVAs) (1, 2) can be detected in these points of flow convergence. The medullary veins flow into the corresponding subependymal veins that drain into the internal cerebral vein and then into the vein of Galen.

The anatomy of the infratentorial medullary veins also includes superficial and deep draining groups: the superficial group is composed of anterior veins (draining into cerebellar hemispheric veins and petrosal sinuses) and posterior veins (draining into the vermian veins and then into the transverse sinus or torcular herophili).

The infratentorial deep medullary veins converge into the subependymal veins of the fourth ventricle, which opens into the veins of the lateral recess of the fourth ventricle and then drains into the petrosal sinus. Others drain into the anterior and lateral transpontine veins, connecting through the pons-mesencephalic veins into the Galen's system.

Under normal conditions, the presence of superficial and deep medullary veins in the brain is unusual. Abnormalities of deep medullary veins (3, 4, 5) can be seen in malignant gliomas and neurological diseases like Sturge-Weber Syndrome, vascular malformations (AVMs), DVAs, and venous thrombosis.

The complete or partial thrombosis of the dural venous sinus has an incidence of 5-7 cases per million in children and 3-4 cases per million in adults, accounting for 0.5 % of all strokes. No racial predilection has been observed, but this condition is more common in women than in men, and mortality has been reported to range from 13.8% to 48% in acute cases (8). Causes can be acquired or genetic diseases such as antiphospholipid syndrome, protein C and S deficiency, lupus coagulant, and Leiden factor V mutation, and different types of cancers. Hyperhomocysteinemia is also a strong independent risk factor.

Other risk factors for cerebral sinus thrombosis are as follows:

- Hematological diseases (paroxysmal nocturnal hemoglobinuria, thrombotic thrombocitopenic purple and polycitemia)
- Collagen-vascular diseases (Wegner granulomatosis, Beçhet syndrome, and systemic lupus erythematosus)
- Infections (meningitis and sinusitis) spreading into the paranasal sinuses
- Minor traumas

Iatrogenic causes are neurosurgery procedures, lumbar punctures, and medications (contraceptives, corticosteroids, epsilon-aminocaproic acid, thalidomide, tamoxifene, and EPO). Cerebral dural sinus thrombosis is rarely associated with spontaneous intracranial hypotension, hypothyroidism (9), or iron-deficiency anaemia (10).

Clinical presentations include headaches (88%), focal neurological deficits (36%), papilledema (28%), mental status disorders (22%) and cranial nerve syndromes (18%) (6). Differential diagnoses include acute strokes, intracranial epidural abscesses, staphylococcal meningitis, status epilepticus, and subdural empyemas. The symptoms are related to the blockage site (7, 8), and for this reason the knowledge

of cerebral anatomy is essential. The cerebral infarction may occur with thrombosis of cortical veins or sagittal sinus secondary to flow obstruction; lateral sinus thrombosis may be associated with headache and pseudotumor cerebri-like syndrome; the extension into the jugular bulb may cause jugular foramen syndrome; or cranial nerve palsies may be seen in the thrombosis of the cavernous sinus as a compressive phenomenon.

CT is usually the first diagnostic approach for suspected dural sinus thrombosis. The diagnosis is based on the confirmation of thrombus within the venous sinuses, thrombosed cortical veins, collateral venous channels, and the "delta" or "cord" sign (direct signs). In addition to this, edema, stroke, or hemorrhagic lesions (indirect signs) can be present.

The "cord" sign (11) refers to a homogeneous, hyperattenuating, cordlike appearance of the brain on an unenhanced CT scan: it appears as the result of increased attenuation either in the dural sinuses or in a vein filled with thrombus. The intraluminal venous thrombosis behaves more like a parenchymal haemorrhage and is therefore hyperattenuating for the first week or more after formation.

Cerebral CT-venography is a quick and useful way to confirm or exclude the diagnosis of sinus thrombosis, when an MRI study gives equivocal results.

MRI examination, using the morphological SE T1- and FSE T2-weighted sequences, can show consequences like edema, infarction, or hemorrhage. Cytotoxic or vasogenic edema may be visible, like diffuse hyperintensity, using diffusion-weighted images (DWI). This finding may be associated with many pathologies, but the study of apparent diffusion coefficient (ADC) can differentiate it from the signal originating from arterial infarcts. In the last few years, the introduction of noninvasive and highly sensitive diagnostic techniques, such as Magnetic Resonance Venography (MRV), has changed our knowledge about cerebral sinus thrombosis.

A partial or complete sinus thrombosis causes a stopping of venous outflow: in the cases of acute thrombosis, loss of flow void on T1-weighted images along with hypointensity on T2-weighted images makes the assessment of sinus occlusion difficult. In the subacute stage, the conversion of hemoglobin to methemoglobin can result in loss of the normal void on T1-weighted images and in hyperintensity of the blood clot. Using T1-weighted contrast-enhanced images, the real presence of filling defects on the dural sinus can be recognized. The DWI sequence can show increased signal corresponding to intravascular. The corresponding low values of the ADC suggest restriction of water molecule movement at the site of occlusion (12, 13). An intracranial venous outflow study can also be implemented through flow-sensitive sequences like 2D-TOF or phase-contrast MRV (6, 7, 8). The 2D-TOF pulse sequence is sensitive to slow flow, especially when it uses coronal acquisition, and when it uses the saturation band to eliminate arterial signal. This sequence presented some limitations because venous anatomy variants are common in the population, and a hypoplastic sinus or prominent arachnoid granulations can simulate sinus thrombosis; furthermore, in this sequence the thrombus (both in the intracellular and extracellular methemoglobin stage) showed increased signal and falsely simulated blood flow. Other technical problems are motion-sensitivity artifacts and insensitivity to slow flow, easily avoided through the phase-contrast MRV.

The gold standard for cerebral sinus thrombosis remains conventional digital subtraction angiography. This exam shows a real configuration of arterial and venous flow that minimizes the possibility of wrong diagnosis or the nonrecognition of vascular lesions associated with thrombosis as the presence of arteriovenous fistulae. Unfortunately, our patient refused angiography and possible endovascular treatment.

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