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# Deep cerebral venous thrombosis: imaging in eight cases

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**Abstract** Deep cerebral venous thrombosis (DCVT) is a rare, potentially fatal disease. We carried out a retrospective analysis of eight patients presenting with DCVT, using CT and MRI data. Digital subtraction angiography (DSA), MR angiography (MRA) (2D phase-contrast), and angiography were performed in four, four and two patients, respectively. Direct (venous abnormalities) and indirect (parenchymatous involvement) signs of thrombosis were assessed. Follow-up MRI and MRA were performed on two patients. CT was initially normal in two patients, demonstrated a "cord sign" in deep veins in five and deep venous infarcts in three. When performed within a week of admission (seven patients), MRI showed thrombosis as into intense or high signal in deep veins on sagittal T1-weighted spin-echo images in all patients, and on axial T2-weighted spin-echo images in two. Deep venous infarcts were found in five patients. Direct or indirect signs of sagittal or lateral sinus thrombosis were present on CT in two patients and on MRI in five. CT angiography, MRA and DSA were concordant with MRI findings. CT venography showed persistent flow in thrombosed veins. MRI follow-up demonstrated progressive deep venous recanalisation.

**Key words** Venous thrombosis, deep cerebral · Angiography

## Introduction

Although cerebral dural and venous thrombosis is increasingly recognised, thrombosis of the cerebral deep venous system is very rare [1], and poorly documented. Angiography was considered as the definitive test for diagnosis of deep cerebral venous thrombosis (DCVT), but recent reports have demonstrated the usefulness of MRI [2–8]. We present eight cases, with specific reference to CT, digital subtracted angiography (DSA), MRI and MR angiography (MRA).

## Patients and methods

From 1989 to 1998, eight patients (3 men and 5 women) from 17 to 62 years of age (mean 33 years) presenting with DCVT were treated in our hospital.

Patients with neoplastic and septic venous occlusion were excluded. Axial CT before and after contrast medium was performed for all patients, within 2 days of the clinical onset (except patient 1, who had raised intracranial pressure (ICP) for 1 week).

The diagnosis was confirmed by DSA in five cases, on day 2 (case 5), 3 (cases 2 and 6), 4 (case 8) and 10 (case 1) after the clinical onset. In the other three cases, the CT, MRI and MRA findings were sufficiently specific to avoid cerebral angiography. MRI was obtained with a 0.5-T unit within a week of clinical onset (cases 1–4, 6), and 45 days after the onset in case 5. Sagittal T1- and axial T2-weighted spin-echo (SE) images were used.

We also studied cases (1, 3, 4, 6 and 8) with 2D phase-contrast MRA (TR 60, TE 26 ms, flip angle 30°). Sagittal sections were obtained 4 times, coronal and axial sections twice. The average

**Table 1** Deep cerebral venous thrombosis: clinical data and outcome. *RICP* raised intracranial pressure

| Case | Age (years)/sex | Predisposing factors | Presentation | Outcome |
|---|---|---|---|---|
| 1 | 50/F | | RICP | complete recovery |
| 2 | 17/M | Nephrotic syndrome; protein S deficiency | RICP; motor and sensory deficits | sensory deficit |
| 3 | 62/M | | motor and sensory deficits | unknown |
| 4 | 22/F | Oral contraceptive | RICP, fever, neck stiffness | complete recovery |
| 5 | 23/M | | RICP, seizure, deterioration of consciousness, decerebration | vegetative state |
| 6 | 38/F | Oral contraceptive; liposuction | RICP, lethargy, motor deficit | complete recovery |
| 7 | 41/F | Oral contraceptive | RICP, lethargy | complete recovery |
| 8 | 42/F | Oral contraceptive | RICP, lethargy | complete recovery |

velocity-encoding value was 10 cm/s. All examinations were interpreted independently by two neuroradiologists. Parenchymal involvement and direct signs of CVT were analysed separately, as were signs of deep and superficial CVT. CVT was defined as lack of flow void or evidence of thrombus in the dural sinuses and deep cerebral veins on all pulse sequences. The site and extent of thrombus was noted, as were the site, size and appearances of brain lesions. Follow-up was performed with MRI and MRA in cases 1, 4 and 6.

## Results

In five patients there were predisposing factors: nephrotic syndrome and protein S deficiency (case 2), oral contraceptive use (cases 4, 6–8), and liposuction (case 6). Raised ICP was present in five patients, isolated or in association with various, non specific neurological signs. Four patients showed deterioration of conscious level (cases 4, 6–8). Motor and sensory deficits were found in two patients, neck stiffness, seizures or fever in one.

The imaging findings are presented in Table 2. Of the eight patients, two had normal CT the acute phase (the first 2 days after onset). A "cord sign" was found on CT in five patients, as a spontaneous increased density of the straight sinus, isolated (in case 4) or together with increased density of the vein of Galen (cases 5, 7), or of the entire deep venous system (cases 2, 8); case 4 and 5 also showed direct signs of sagittal superior sinus (SSS) thrombosis, consisting of a spontaneous density and an triangle sign after contrast enhancement.

In seven cases, one or several deep veins and/or dural sinuses appeared isointense (Figs. 1 a, 2 a) or gave high signal (Figs. 3 b, 4) on T1-weighted. This was observed in all patients who underwent MRI in the acute or subacute phase (within 15 days of thrombosis). CT angiography showed persistent flow in thrombosed vein (Fig. 4 d, e). On T2-weighted images, high signal was observed in the deep venous system in only cases 2 and 3, but this was less intense than on T1-weighted images. CT showed deep venous infarcts in three patients and superficial venous infarcts also in three. On MRI, five patients were found to have high signal in the deep cerebral parenchyma on T2-weighting, corresponding to deep venous infarcts (Figs. 2 b, 3 c), bilateral in three. In two patients, one or several superficial venous infarcts were also present. Patients 3 and 5 had only superficial venous infarcts. Patients 3 and 4 had haemorrhagic infarcts, seen as high signal on both T1- and T2-weighted images.

MRA, performed in cases 1, 3, 4, 6 and 8, demonstrated no significant flow in the veins and dural sinuses which were isointense or gave high signal on MRI (Fig. 3 d, e). DSA (cases 1, 2, 5, 6 and 8) was always concordant with MRI and MRA data (Fig. 3 f).

Follow-up showed decrease in high signal on the T1-weighted images in cases 1 and 4 and partial restitution of flow on MRA performed 2 weeks after the onset (case 6, Fig. 3 i).

High signal in the right basal ganglia on T2-weighted images partially disappeared in case 4, studied 5 and 8 months after onset (Fig. 2 d). In case 6, MRI 15 days after clinical onset showed disappearance of high signal in the thalamus with persistence of subtle high signal in the posterior limb of the internal capsule (Fig. 3 h).

## Discussion

The deep cerebral venous system collects blood from cerebral hemisphere white matter, basal ganglia, thalamus and diencephalon. Unlike the superficial system, the topography of deep cerebral veins is constant, and the deep veins are always seen on cerebral angiography [9, 10].

Only a few reports concern DCVT [2–4, 11–14]. About 3–8 % of cases of CVT involve the deep venous system, and only nine cases of DCVT were found in 110 patients with cerebral thrombophlebitis [12]. Many cases have been young children [5, 6, 15]. About 75 % of patients have predisposing factors, including oral contraceptives, pregnancy, puerperium and liposuction [3]. DCVT sometimes occurs as a consequence of a pre-

**Table 2** Imaging findings (*T1, T2WI* T1-, T2-weighted images, *BV* basal vein, *ICV* internal cerebral vein, *LS* lateral sinus, *SS* straight sinus, *SSS* superior sagittal sinus, *VG* vein of Galen)

| Case | CT | | MRI | | MRA | Intra-arterial angiography |
|---|---|---|---|---|---|---|
| | Veins | Brain | Veins | Brain | | |
| 1 | Day 7: normal<br>Day 11: high density posteriorly | Normal | *T1WI*<br>ICV, VG isointense<br>SS high signal<br>*T2WI*<br>High signal SSS, left LS<br>*T1, T2WI* high signal ICV, BV, VG, SS, left LS | Normal | Deep veins, SSS, left LS not seen | Deep veins, SSS, left LS not filled |
| 2 | Day 0: dense deep veins | Infarcts left thalamus, cerebral peduncles, temporal lobe | | Infarcts left thalamus, cerebral peduncles, temporal lobe | Not done | Deep veins, left LS not filled |
| 3 | Normal | Haemorrhagic right parietal infarct | *T1, T2WI:* high signal<br>VG, SS, SSS, right and left LS | Haemorrhagic right parietal infarct | Amputation VG, SS, SSS, right and left LS | Not done |
| 4 | Day 1: dense SS, SSS, triangle sign SSS | Small haemorrhagic cortical infarcts | *T1WI*<br>ICV, VG isointense<br>high signal SS<br>*T2WI*<br>low signal SSS, right and left LS | Infarcts right thalamus, basal ganglia, left parasagittal haemorrhagic cortical infarct | VG, SS, SSS, right and left LS not seen | Not done |
| 5 | Day 1: dense VG, SS, SSS; triangle sign SSS | Normal | *T1WI* (late): persistent high signal right LS | Extensive frontal, parietal and temporal infarcts both sides | | Deep veins, SSS, right LS not filled |
| 6 | Day 0: normal | Normal | *T1WI*<br>ICV, VG,<br>SS isointense | Infarct thalamus (bilateral), posterior limb internal capsule | Left ICV, VG, SS, left LS not seen | Deep veins, left LS not filled |
| 7 | Day 2: dense VG, SS | Bithalamic infarcts, intraventricular haemorrhage | *T1WI*<br>high signal SS, right LS;<br>ICV, VG isointense | Infarcts thalamus (bilateral) right basal ganglia, anterior limb internal capsule | Not done | Not done |
| 8 | Day 1: 1 dense deep veins CT angiography: poor filling deep veins defects in SS, right LS | Bithalamic infarcts | *T1WI*<br>high signal ICV, VG, SS | Infarcts thalamus (bilateral), right basal ganglia, anterior limb internal capsule | Deep veins not seen | Poor filling ICV, VG, defects SS, right LS |

viously unknown superficial dural venous thrombosis [14].

Clinical onset is variable and nonspecific: headache, lethargy, motor or sensory deficits seizures, and sometimes neck stiffness with fever [1]. Disturbances of consciousness are classical, but variable [16, 17]. In our series four patients presented with deterioration of conciousness or lethargy. The illness can be fatal, but outcome seems better for newborns than for older children and adults [5, 15].

For many years, cerebral angiography has been the primary, definitive imaging modality when DCVT was suspected. Usually, all or part of the deep venous system is not opacified (a direct sign of thrombosis). However, diagnosis can be difficult because of anatomical variants; the straight sinus collecting blood from a cerebral hemisphere as well as from a part of the posterior cranial fossa may not be shown. When the diagnosis is uncertain, indirect signs of venous thrombosis, such as collateral venous channels or slow flow must be sought.

Currently primary use of MRI and MRA is recommended, but in most cases CT scan is still the first examination to be performed. It is normal in about 20 %

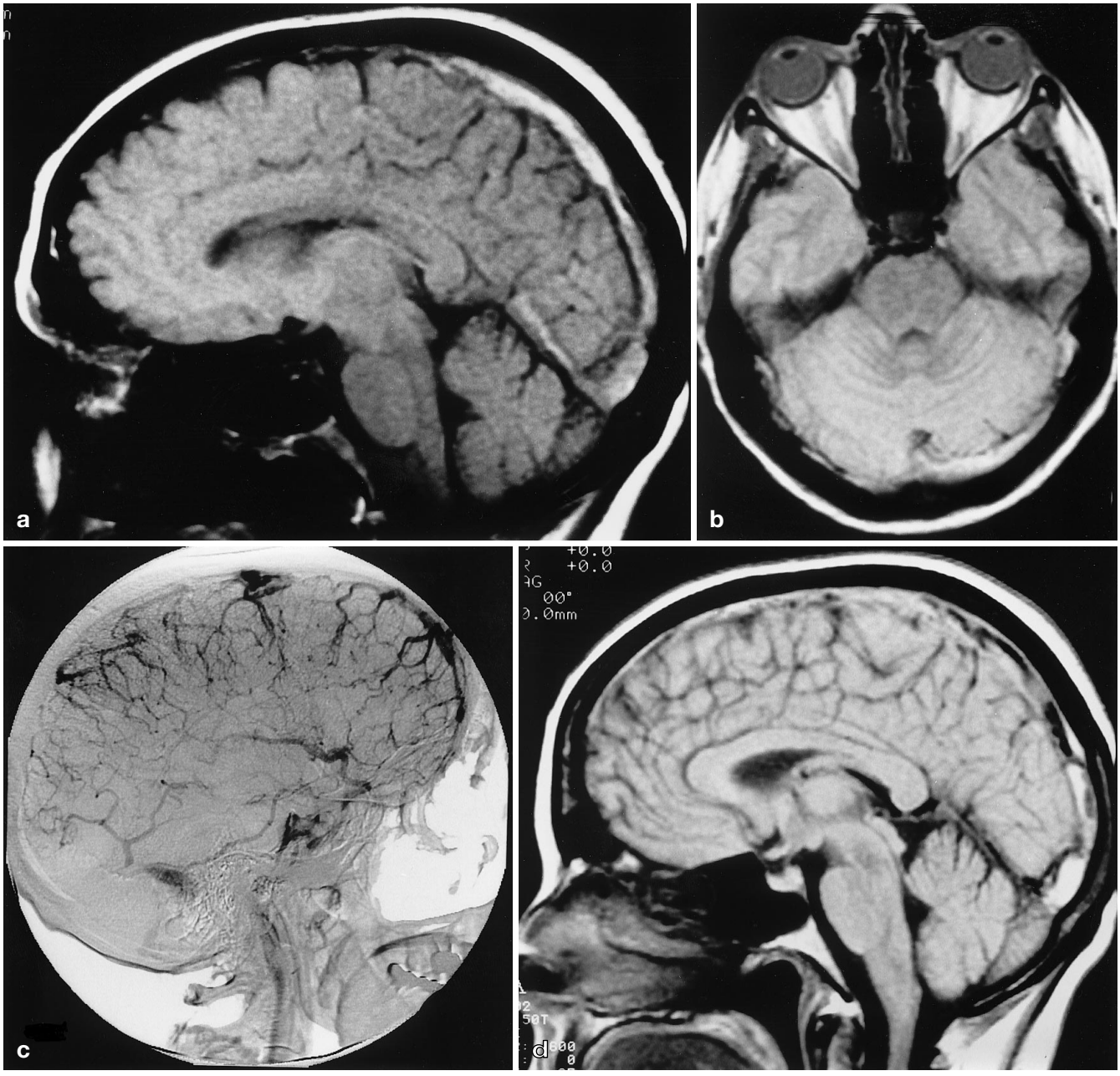


**Fig. 1** Case 1. **a** Sagittal T1-weighted image shows signal isointense with brain parenchyma in the internal cerebral vein, vein of Galen and high signal in the straight sinus and superior sagittal sinus (SSS). **b** Axial proton-density image shows high signal in the left lateral sinus. **c** Left carotid angiogram, lateral projection: internal cerebral vein, vein of Galen, straight sinus, torcular herophili and SSS are not seen. **d** Sagittal T1-weighted image 15 days later shows complete recanalisation (signal void) of the left internal cerebral vein *(arrow),* and partial recanalisation of the straight sinus and SSS

of cases of cerebral venous thrombosis. The "cord sign", spontaneously increased density of cerebral veins, can be seen in all or part of the deep venous system, and is a direct sign of thrombosis. However, a spontaneous density in the area of the straight sinus can be observed in normals [18]. Increased density of thalamostriate veins is described as a specific sign, but was not seen in our series. The triangle sign after contrast medium injection is rare in DCVT, but can be seen in the straight sinus in the subacute phase. Direct signs were found in five of our cases: this sensitivity is higher than previously reported. It is possible that spontaneously high venous

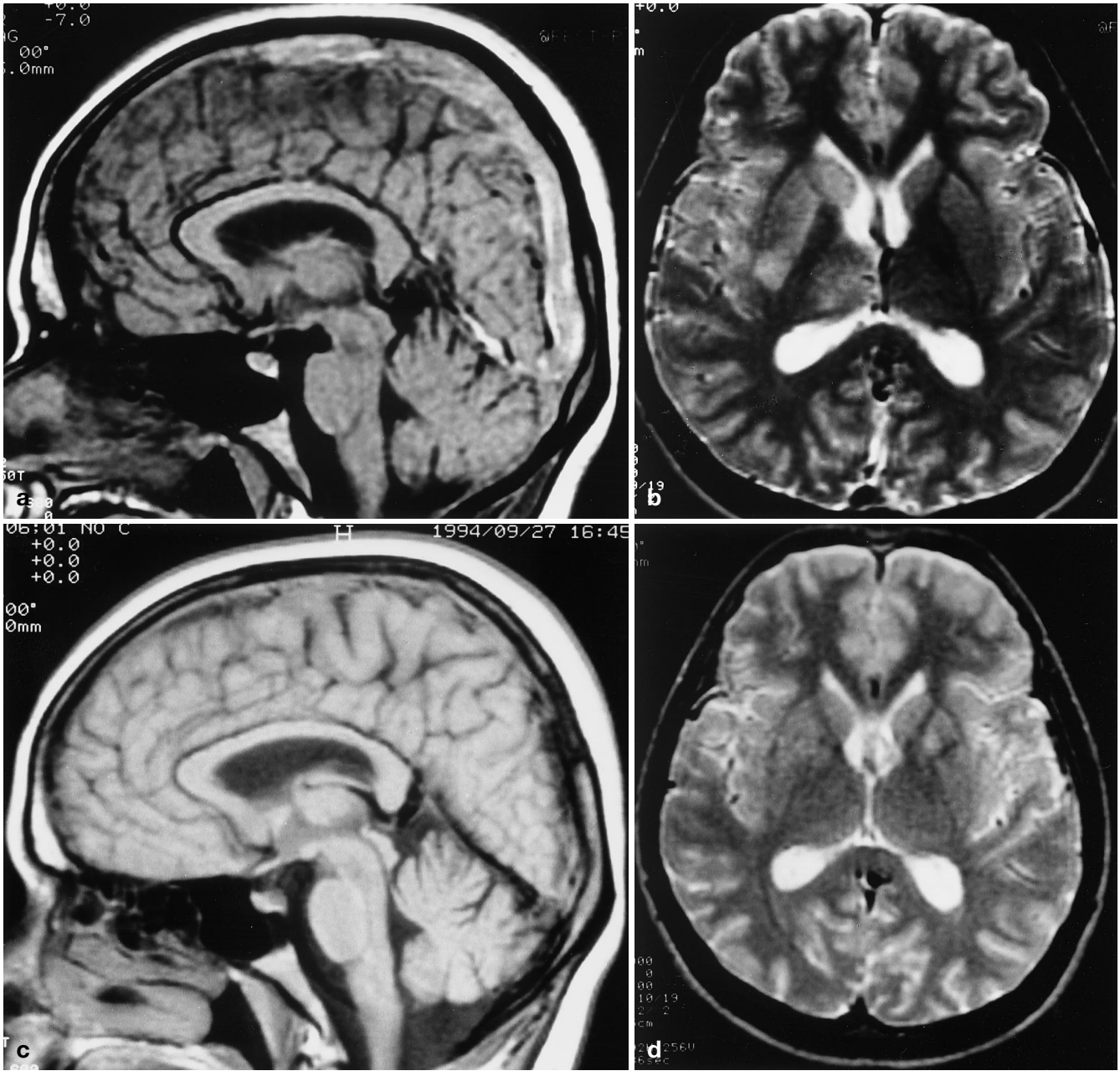


**Fig. 2** Case 4. **a** Sagittal T1-weighted image shows high signal in the SSS, straight sinus, and isointense internal cerebral vein and vein of Galen. **b** Axial T2-weighted image shows high signal in the right thalamus and caudate and lentiform nuclei. **c** Sagittal T1-weighted image 5 months later reveals flow void in the right internal cerebral vein, vein of Galen and straight sinus. The torcular herophili and SSS show irregular areas of isointense signal. **d** Axial T2-weighted image 8 months after onset shows disappearance of parenchymal abnormalities and normal appearance of the internal cerebral veins

density is easier to detect in the deep system because, unlike the SSS or lateral sinus, deep veins lie far from dense bone. CT, initially normal, can show abnormalities several days later (as in our case 1). CT also detects the frequent parenchymal involvement [19] as brain swelling, venous infarcts. CT is also useful in an emergency to eliminate subarachnoid haemorrhage or a brain tumour.

In all our patients, superficial dural thrombosis was also present, and was responsible for similar features, but at different sites: cord and triangle signs in the SSS (cases 4, 5), cortical infarcts (cases 3, 4).

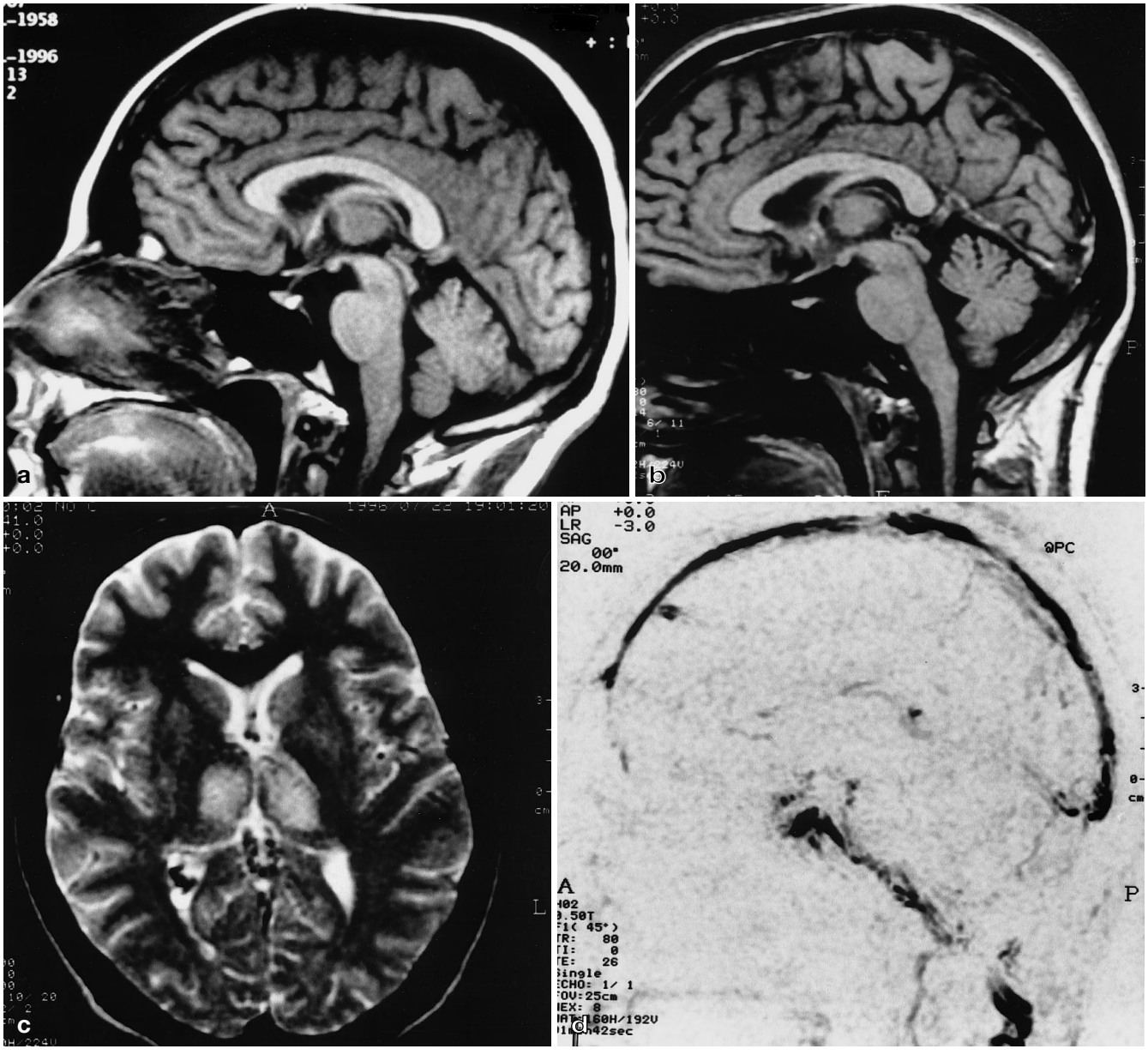


**Fig. 3** Case 6. **a** Sagittal T1-weighted image performed on day of onset shows isointense signal from the internal cerebral vein, vein of Galen, and straight sinus. **b** on day 3 there is high signal in the straight sinus and vein of Galen. **c** Axial T2-weighted image shows high in the thalamus and the posterior limb of the left internal capsule. **d, e** MRA (2D phase-contrast, midline sagittal) sagittal and coronal: the straight sinus is not seen; only the right internal cerebral vein is seen *(arrow)* in either planes. No flow is visible in the left lateral sinus. **f** Left carotid angiogram, lateral projection, shows no filling of the deep cerbral venous system. **g** T1-weighted left parasagittal image 14 days after onset shows partial recanalisation of straight sinus and complete recanalisation of the vein of Galen and internal cerebral vein

Recent reports [20] emphasise the value of CT angiography for assessment of venous thrombosis. CT angiography is performed after conventional CT, using helical acquisition in the axial plane (1–3 mm slices, reconstruction with maximum-intensity projections in coronal and sagittal planes) after contrast medium. The internal cerebral vein and the vein of Galen are always demonstrated on normal CT angiograms. The axial sources images must always be analysed, in addition to the-three dimensional reconstructions. The triangle sign and persistent flow are easily recognised in thrombosed dural sinuses on source images and three-dimensional CT venography. However, in deep venous thrombosis, poor demonstration of thrombosed veins, with col-

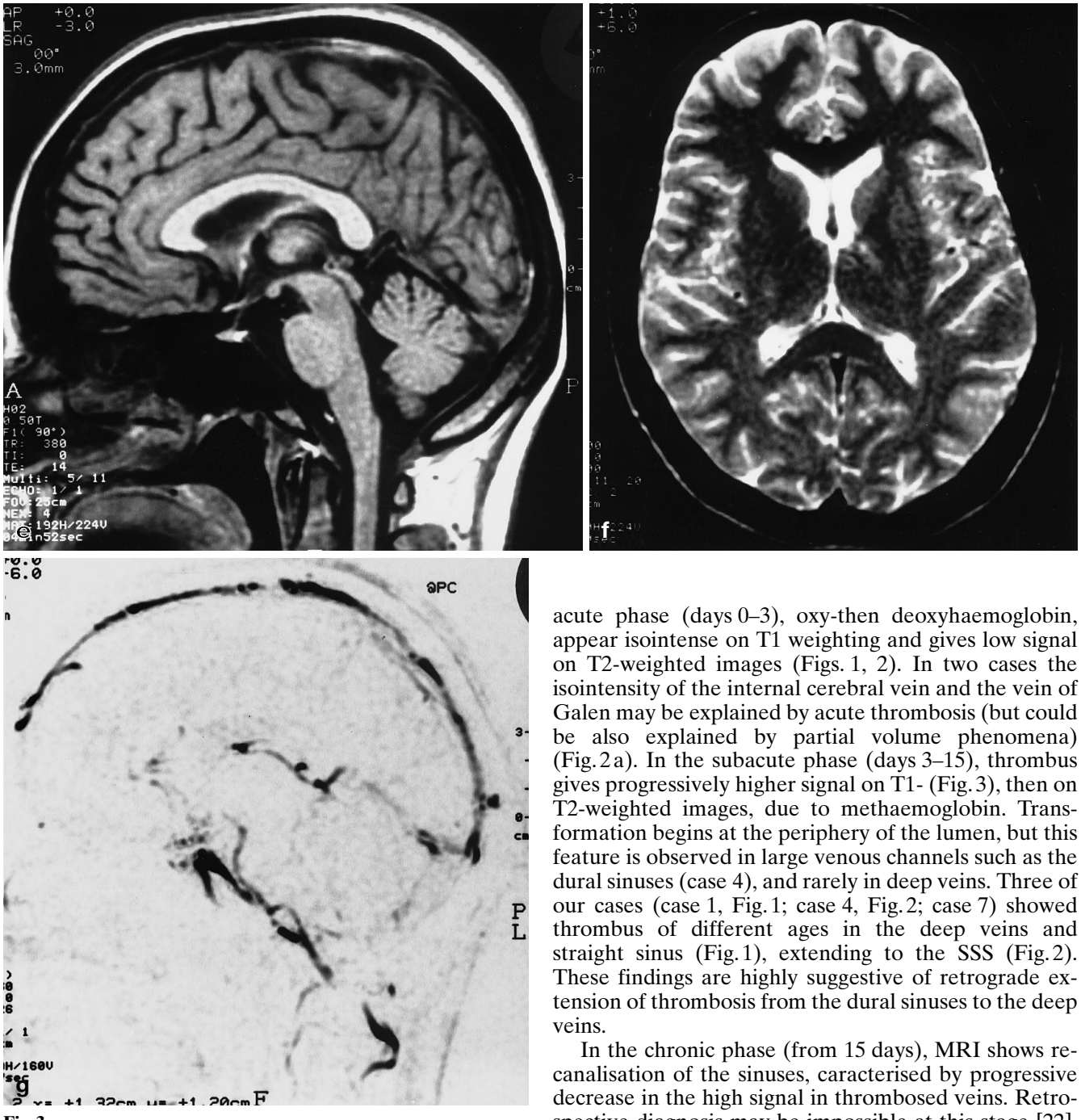


**Fig. 3 e-g**

lateral venous channels, are seen most often [20]. MRI, when performed less than 2 weeks after the clinical onset (all our cases except case 5), shows signal abnormal vein, consistent with thrombosis (Figs. 1–3). Blood catabolism in thrombosed veins produces signal change. Three different phases can be identified [21] using mid- or high-field imagers, and spin-echo sequences: in the acute phase (days 0–3), oxy-then deoxyhaemoglobin, appear isointense on T1 weighting and gives low signal on T2-weighted images (Figs. 1, 2). In two cases the isointensity of the internal cerebral vein and the vein of Galen may be explained by acute thrombosis (but could be also explained by partial volume phenomena) (Fig. 2 a). In the subacute phase (days 3–15), thrombus gives progressively higher signal on T1- (Fig. 3), then on T2-weighted images, due to methaemoglobin. Transformation begins at the periphery of the lumen, but this feature is observed in large venous channels such as the dural sinuses (case 4), and rarely in deep veins. Three of our cases (case 1, Fig. 1; case 4, Fig. 2; case 7) showed thrombus of different ages in the deep veins and straight sinus (Fig. 1), extending to the SSS (Fig. 2). These findings are highly suggestive of retrograde extension of thrombosis from the dural sinuses to the deep veins.

In the chronic phase (from 15 days), MRI shows recanalisation of the sinuses, caracterised by progressive decrease in the high signal in thrombosed veins. Retrospective diagnosis may be impossible at this stage [22]. In the later phase, restitution of flow void indicates partial (Figs. 1 d, 3 g) or complete recanalisation (Fig. 2 c).

MRI must be acquired in different orthogonal planes, to eliminate entry-slice and even-echo rephasing phenomena, which can induce high signal in veins. The sagittal plane seems to be the most informative. In our series, high signal thrombus was always obvious on T1-weighted SE images in this plane, while it was detected in two of eight cases on the axial T2-weighted images. In

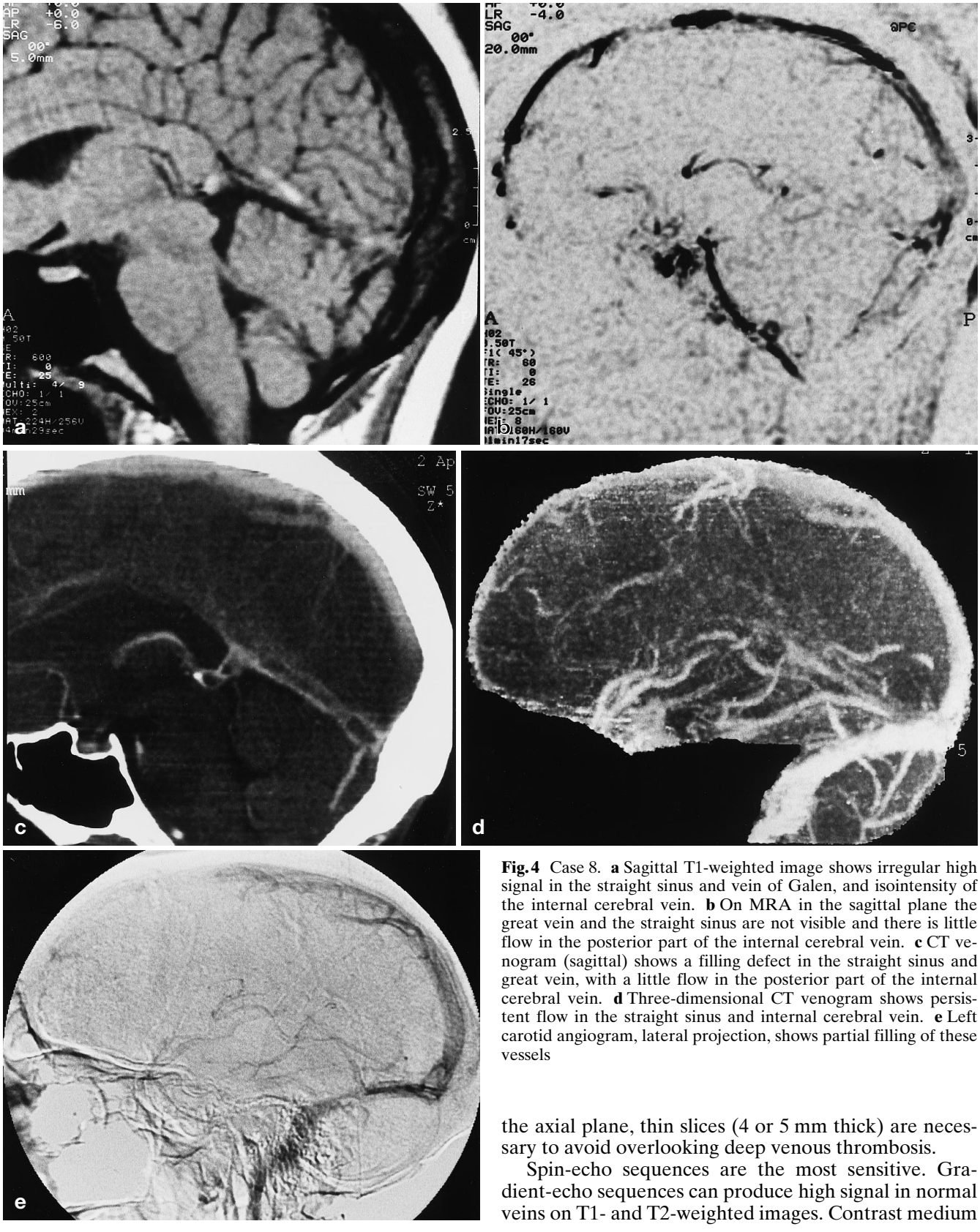


**Fig. 4** Case 8. **a** Sagittal T1-weighted image shows irregular high signal in the straight sinus and vein of Galen, and isointensity of the internal cerebral vein. **b** On MRA in the sagittal plane the great vein and the straight sinus are not visible and there is little flow in the posterior part of the internal cerebral vein. **c** CT venogram (sagittal) shows a filling defect in the straight sinus and great vein, with a little flow in the posterior part of the internal cerebral vein. **d** Three-dimensional CT venogram shows persistent flow in the straight sinus and internal cerebral vein. **e** Left carotid angiogram, lateral projection, shows partial filling of these vessels

the axial plane, thin slices (4 or 5 mm thick) are necessary to avoid overlooking deep venous thrombosis.

Spin-echo sequences are the most sensitive. Gradient-echo sequences can produce high signal in normal veins on T1- and T2-weighted images. Contrast medium

is not routinely used, because it does not prove decisive in thrombosis. Parenchymal involvement is frequent: seven of our eight patients had venous infarcts, with a thrombosed venous channel on MR or DSA.

MRI yields indirect signs of thrombosis; although dilated transmedullary veins were not seen in our series, and may be rare, brain swelling and venous infarcts are frequent. Deep venous infarcts were found in five, in the basal ganglia (caudate and lenticform nuclei), thalamus and the deep white matter of the temporal lobe, areas not usually involved in isolated superficial dural thrombosis. The infarcts are often haemorrhagic and bilateral [4, 11, 13]. The cerebellar hemispheres and brain stem may be involved, because the straight sinus collects blood from part of the posterior cranial fossa [10]. However, posterior cranial fossa infarcts are found most frequently in basilar artery thrombosis. In cases of bilateral thalamic infarcts, basilar and deep venous thrombosis should be considered, and MRA or cerebral angiography performed [4]. Thalamic infarcts in five of our cases were associated with cortical infarcts in two.

Like cerebral angiography, MRA demonstrates the deep venous system and dural sinuses, and can be combined with conventional MRI [5]. Phase-contrast (2D) or time-of-flight methods can be used. The latter is based on slice-entry phenomena, with a very short repetition time. However, high signal from moving venous protons can sometimes not be distinguished from thrombus, especially chronic [4].

The PC technique is probably the modality of choice, because only flowing protons contribute to the signal. Two-dimensional [23] or 3-D [7] techniques can be used. Velocity encoding should be 10–20 cm/s to improve detection of small slow-flow veins [4, 7]. Since anatomical variations like hypoplasia or agenesis can mimic venous thrombosis, comparison of MRA and MRI is necessary. MRI and MRA can also be used to follow progressive venous recanalisation.

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