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Venous Sinus Occlusive Disease: MR Findings

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PURPOSE: To study MR patterns of venous sinus occlusive disease and to relate them to the underlying pathophysiology by comparing the appearance and pathophysiologic features of venous sinus occlusive disease with those of arterial ischemic disease. **METHODS:** The clinical data and MR examinations of 26 patients with venous sinus occlusive disease were retrospectively reviewed with special attention to mass effect, hemorrhage, and T2-weighted image abnormalities as well as to abnormal parenchymal, venous, or arterial enhancement after intravenous gadopentetate dimeglumine administration. Follow-up studies when available were evaluated for atrophy, infarction, chronic mass effect, and hemorrhage. **RESULTS:** Mass effect was present in 25 of 26 patients. Eleven of the 26 had mass effect without abnormal signal on T2-weighted images. Fifteen patients had abnormal signal on T2-weighted images, but this was much less extensive than the degree of brain swelling in all cases. No patient showed abnormal parenchymal or arterial enhancement. Abnormal venous enhancement was seen in 10 of 13 patients who had contrast-enhanced studies. Intraparenchymal hemorrhage was seen in nine patients with high signal on T2-weighted images predominantly peripheral to the hematoma in eight. Three overall MR patterns were observed in acute sinus thrombosis: 1) mass effect without associated abnormal signal on T2-weighted images, 2) mass effect with associated abnormal signal on T2-weighted images and/or ventricular dilatation that may be reversible, and 3) intraparenchymal hematoma with surrounding edema. **CONCLUSION:** MR findings of venous sinus occlusive disease are different from those of arterial ischemia and may reflect different underlying pathophysiology. In venous sinus occlusive disease, the breakdown of the blood-brain barrier (vasogenic edema and abnormal parenchymal enhancement) does not always occur, and brain swelling can persist up to 2 years with or without abnormal signal on T2-weighted images. Abnormal signal on T2-weighted images may be reversible and does not always indicate infarction.

Index terms: Venous sinuses; Brain, magnetic resonance; Brain, diseases; Brain infarction

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Venous sinus occlusive disease (VSOD) is a serious, potentially lethal (20% to 78%) problem (1) that may present a confusing, nonspecific

clinical picture. Stroke-like symptoms with a slow onset, especially in a clinical setting with known predisposition to venous thrombosis, suggest the diagnosis. Predisposing factors include ear and mastoid infection (2), pregnancy and puerperium (3), oral contraceptives (4), hypercoagulable states, disseminated intravascular coagulation (5), polycythemia, acquired and congenital heart disease (6), sickle cell anemia (7), severe dehydration (8), trauma (9, 10), Behcet syndrome (10), and neoplasms (11). Often, however, the true diagnosis is unsuspected before radiologic evaluation.

The radiologic diagnosis of venous sinus thrombosis, including the “delta” sign on contrast-enhanced computed tomography (CT) (12) and the signal characteristics of thrombus and lack of flow in the sinus on magnetic resonance (MR) imaging (13), is well known. Although venous

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infarction and hemorrhage are known complications, the patterns of parenchymal and vascular changes on MR have not been well documented.

The present investigation was designed to study the MR appearance of VSOD and compare it with that of arterial cerebral ischemia. An attempt was also made to relate the MR findings to the underlying pathophysiology in VSOD.

Materials and Methods

A retrospective review was made of the clinical history and MR examinations of 25 patients with VSOD. Patients ranged in age from 4 days to 72 years; 12 were male and 13 were female. There were predisposing factors known to be associated with sinus thrombosis in 19 patients, including oral contraceptives (five patients), otitis media/mastoiditis (four patients), the postpartum period (three patients), hypercoagulable states (two patients), sickle cell anemia (one patient), postoperative complications (two patients), dehydration (one patient), and pseudotumor cerebri (one patient). No predisposing factors were identified in the remaining six patients.

Examinations were performed on either a mid- (0.35 to 0.5 T) or high-field (1.5 T) superconductive scanner. All patients had a noncontrast MR examination including relatively T1- and T2-weighted conventional spin-echo images in at least two planes. Twelve patients were also evaluated with contrast-enhanced studies (0.1 mmol/kg intravenous gadopentetate dimeglumine), and 11 patients had follow-up examinations.

On the noncontrast MR studies, special attention was given to the presence or absence of mass effect, hemorrhage, parenchymal signal on T2-weighted images, and signs of venous sinus occlusion including absence of flow void phenomena. A subjective comparison of the area of abnormal brain involved with swelling versus signal change and hemorrhage was made. Mass effect was defined as effacement of sulci or the ventricular system on either T1- or T2-weighted images. Areas of abnormal parenchymal signal change were judged on T2-weighted images. Assessment was also made for areas with swelling with no abnormal signal, and for areas with greater mass effect than signal abnormality. Hemorrhage was defined by the characteristic MR findings of blood in its various forms. Deoxyhemoglobin during the acute phase or hemosiderin during the chronic phase was defined as hypo- to isointense on T1-weighted images. On T2-

weighted images, acute oxy- or deoxyhemoglobin was iso- or hypointense, whereas hemosiderin was hypointense. The contrast-enhanced studies were evaluated for abnormal arterial, parenchymal, and cortical venous enhancement. The degree of dural sinus enhancement was not included as venous enhancement. Enhancing veins can be differentiated from arteries by location and orientation. For example, in the temporal lobe, on axial images, veins appear as structures in the coronal plane (arteries tend to run in the sagittal plane) with caliber increasing from deep to superficial (see Fig 2B).

The follow-up examinations (12 patients) were evaluated for MR evidence of persistent brain swelling, atrophy, signal changes, and hemosiderin.

Results

Mass effect and cortical sulcal effacement were found in 25 of 26 patients. Eleven of the 26 patients had only mass effect without abnormal signal changes. The remaining 15 patients had hyperintense signal on T2-weighted image changes, but these were always much less extensive than the area involved in the brain swelling (cortical sulci effacement/mass effect). Nine of these 15 patients also had intraparenchymal hemorrhage with abnormal signal on T2-weighted images located predominantly peripheral to the hematoma in eight. None of the 13 patients studied with contrast-enhanced examinations had abnormal parenchymal or arterial enhancement; however, 10 had abnormal cortical venous enhancement. Five of the 12 patients with follow-up MR examinations had persistent swelling without T2 abnormality ranging from 2 months to 24 months; eight patients showed partial (two patients) or complete (six patients) resolution of signal on T2-weighted image abnormality, two showed diffuse atrophy, and one had a focal infarction. Three of the 12 follow-up examinations showed focal areas of hemosiderin. Two of these were caused by resolving hematoma, but the third patient did not initially have MR evidence of frank hematoma suggesting petechial hemorrhage. Only one patient with a follow-up examination had a contrast-enhanced study. This patient had chronic venous enhancement for 2 years, but no parenchymal or arterial enhancement.

Discussion

The characteristic MR appearance of arterial occlusive disease including brain swelling, vasogenic edema, and abnormal arterial and parenchymal enhancement has been well described (14–16). The MR appearance is related to ischemic changes caused by temporary or permanent arterial occlusion resulting in flow abnormalities within the artery and a breakdown of the blood-brain barrier. Vasogenic edema and abnormal parenchymal enhancement (with intact delivery of intravenous contrast agent) develop as consequences of the breakdown of the blood-brain barrier. With slow or absent arterial flow, the disappearance of flow-void phenomena and abnormal arterial enhancement can be seen in some patients during the acute phase of arterial occlusive disease.

In our patients with VSOD, abnormal arterial and parenchymal enhancement was not demonstrated during the acute or chronic phases of the disease. The expected hyperintense vasogenic edema seen on T2-weighted acquisitions in arterial ischemia or infarction was not always seen in patients with VSOD. Twenty-five of 26 had areas of brain swelling demonstrated by sulcal effacement. Frequently, however, there was no associated T2 abnormality. Also, when there was T2 abnormality, it was never as extensive as the area of brain swelling. These findings suggest a distinctly different pathophysiology from that of arterial occlusive disease.

In VSOD, the vascular obstruction is on the venous side. Theoretically, this could result in slow arterial flow. However, arterial enhancement was not demonstrated in our patient population (0%). Abnormal enhancement of the cortical or deep venous structures, however, was seen in most patients (10 of 13), which is consistent with the underlying venous stasis. Because the delivery of arterial oxygenated blood to the affected tissue is intact in VSOD, despite poor venous drainage that could diminish arterial supply, the breakdown of the blood-brain barrier, which is frequently seen in arterial ischemia, may not occur in VSOD. This is supported by the lack of abnormal parenchymal enhancement (0%) seen in our patients despite the intact arterial delivery of contrast agent to the abnormal, swollen brain tissue. In addition, the frequent observation of brain swelling without associated vasogenic edema (high signal on T2-weighted images) also suggests an absence of blood-brain barrier breakdown in the areas affected by VSOD.

The MR findings in our patients can generally be grouped into three patterns: 1) brain swelling without signal on T2-weighted image abnormalities, 2) brain swelling with signal on T2-weighted image abnormalities (interstitial edema) but no hematoma, and 3) brain swelling with signal on T2-weighted image changes compatible with hematoma and edema. The reason for these MR patterns is unknown and cannot be explained fully without the support of animal data. In view of the differences in MR findings between VSOD and arterial occlusive disease, we propose the following mechanism to explain the pathophysiology of the three distinctive MR patterns in VSOD.

In the normal brain, there is a constant shift of bulk water (transudate) through the interstitium from the capillary space into the ventricle (Fig 1A) (17), accounting for approximately 10% to 60% of cerebrospinal fluid (CSF) production (18). In hydrocephalus, this process can be reversed causing abnormal increased interstitial fluid. When intraventricular pressure is much greater than the capillary bed (Fig 1B), the CSF is driven from the ventricular space into the interstitium through the ependyma, resulting in an accumulation of excessive interstitial fluid, which can be readily detected as periventricular increased signal on T2-weighted images. The distribution of the "transependymal effusion" is greatest adjacent to the ventricular ependyma and gradually diminishes away from the ventricle, suggesting a reverse pressure gradient from the ventricle (high pressure) to the capillary space (low pressure).

We speculate that the patterns of VSOD may be explained by a disturbance of this hypothetical model of the normal physiologic shift of bulk interstitial fluid from the capillary bed toward the ventricle. The consequences of elevated venous pressure from venous occlusion can result in a spectrum of changes detectable by MR including a dilated venous and capillary bed (Fig 1C), abnormal shift of bulk water, development of interstitial edema (Fig 1D), increased CSF production, decreased CSF absorption, and rupture of venous structures (hematoma) (Fig 1E). All of these pathophysiologic changes may explain the MR patterns observed in patients with VSOD. MR findings of these three patterns with mild to severe sequelae likely depend on the degree of elevation in the venous pressure.

The first pattern seen with VSOD is brain swelling (sulcal effacement) without T2 evidence of edema (Fig 2). This may represent the earliest

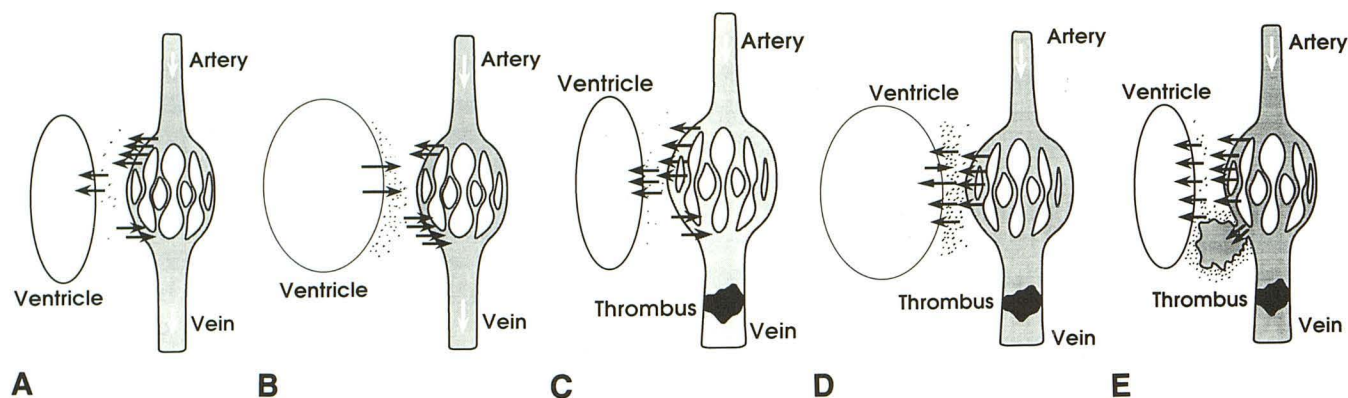


Fig. 1. A, Normal brain has a constant shift of bulk water from the capillary space into the ventricle contributing to a small percentage of CSF production.

B, In hydrocephalus, increased intraventricular pressure causes a reversal of the normal transependymal flow resulting in periventricular edema.

C, Initially, when venous drainage is blocked, the compliant venous system dilates. A slight increase in transudate can be handled by drainage into the ventricle without significant edema developing.

D, As venous pressure and transudate volume increase, the transependymal drainage route becomes saturated, and a larger percentage of CSF production is secondary to transependymal flow. In addition, CSF absorption is decreased because of increased venous pressure. The result is mild hydrocephalus and periventricular edema. These are all reversible changes if venous drainage is restored and venous pressure is reduced.

E, When venous pressure exceeds the integrity of the vascular wall, intraparenchymal hemorrhage results. Edema surrounds the hematoma. Hemorrhage causes irreversible tissue damage.

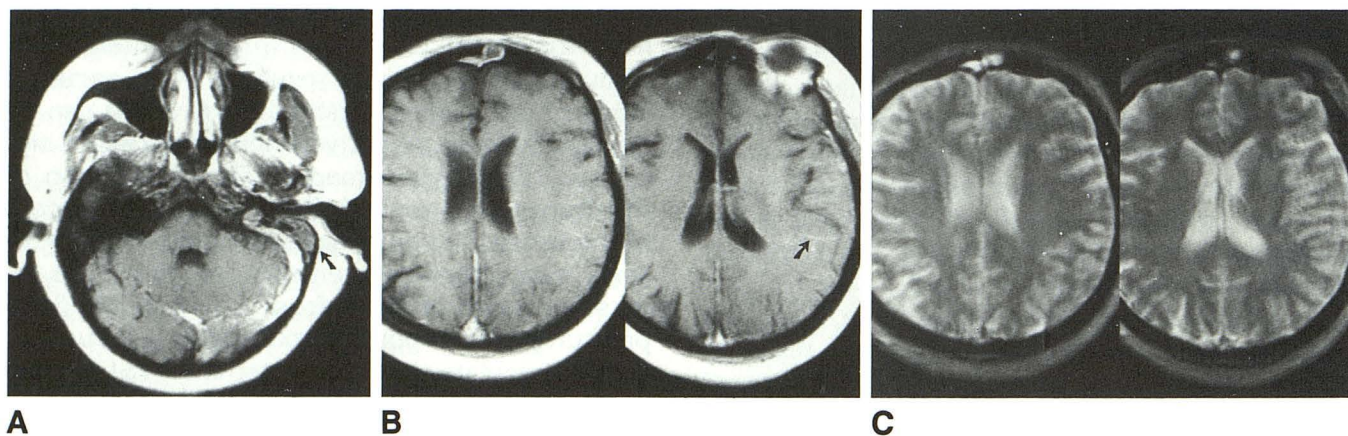


Fig. 2. This 41-year-old woman developed left transverse and sigmoid sinus thrombosis as a complication of mastoiditis.

A, Spin-echo 500/20 (repetition time/echo time) axial T1-weighted image demonstrates the lack of flow void in these sinuses as well as mastoid disease (arrow).

B, Spin-echo 500/20 gadolinium-enhanced axial T1-weighted images show left occipital parietal sulci effacement indicating brain swelling. Faint cortical venous enhancement (arrow) is also seen. This is the first pattern of VSOD diagrammed in Fig 1C.

C, Spin-echo 2000/100 axial T2-weighted images show no associated signal abnormality. Follow-up MR imaging 10 days later showed chronic brain swelling without signal abnormalities.

and/or mildest degree of venous occlusion in which the compliant venous bed dilates without significant elevation of venous pressure. Although the intravascular volume of the brain is normally only 4% to 7% of total brain volume, an increase in blood volume in the distended venous bed can result in a significant increase in total brain volume. The fixed intracranial volume of the skull may potentially cause sulcal effacement even with a small overall increase in brain blood vol-

ume. This is supported by the studies of Bradley et al (19, 20), who demonstrated periodic changes in brain volume without apparent signal abnormality during the different phases of the cardiac cycle. The brain expands with systole and decreases in volume during diastole. Similarly, in VSOD, the mass effect of the brain may simply be attributable to venous congestion. Because the venous pressure is not sufficiently elevated, there should be no significant increase in shift of

interstitial fluid from the vascular bed toward the ventricle and therefore no accompanying signal abnormality on T2-weighted images. This is similar to the mechanism of penile erection, which is also a venous occlusive process with intact arterial supply. This distention of the venous bed without associated interstitial edema may result in persistent brain swelling without abnormal signal on T2-weighted images for months or years, as was seen in some of our patients.

In the second pattern of VSOD, the mass effect of the brain is accompanied by abnormal signal on T2-weighted images that is always less extensive than the overall area of mass effect (Fig 3). Frequently, ventricular dilation is also seen. As the compliance limit of the venous bed is reached, a further increase in venous pressure develops. As the venous pressure continues to rise, it can cause an increased shift of bulk water, driven by the pressure gradient, from the capillary bed into the interstitium. This transudate results in an abnormal accumulation of interstitial fluid in areas with poor venous drainage (Fig 3B). A periventricular distribution of increased signal on T2-

weighted images develops as the transependymal route of drainage becomes saturated. Ferris (21) suggested that a high venous pressure alters the fluid exchange between the blood compartment and the tissue compartment of the brain with the production of edema. This appears to correlate with the MR findings in the second pattern of VSOD, in which the distribution of abnormal signal is periventricular, as well as in poorly drained areas, such as the basal ganglia and thalamus (Fig 4).

With increased shift of the bulk water from the capillary space into the ventricle, communicating hydrocephalus may develop as a result of an increase in overall CSF production. These findings may be related to the degree of venous pressure elevation. Tsai et al (22) reported an elevation of venous pressure in the sagittal and transverse sinuses associated with VSOD, which can be as high as 32 to 47 mm Hg. The MR findings of these patients were of the pattern-2 type.

Hydrocephalus caused by elevated venous pressure in the dural sinuses (23–25) and superior

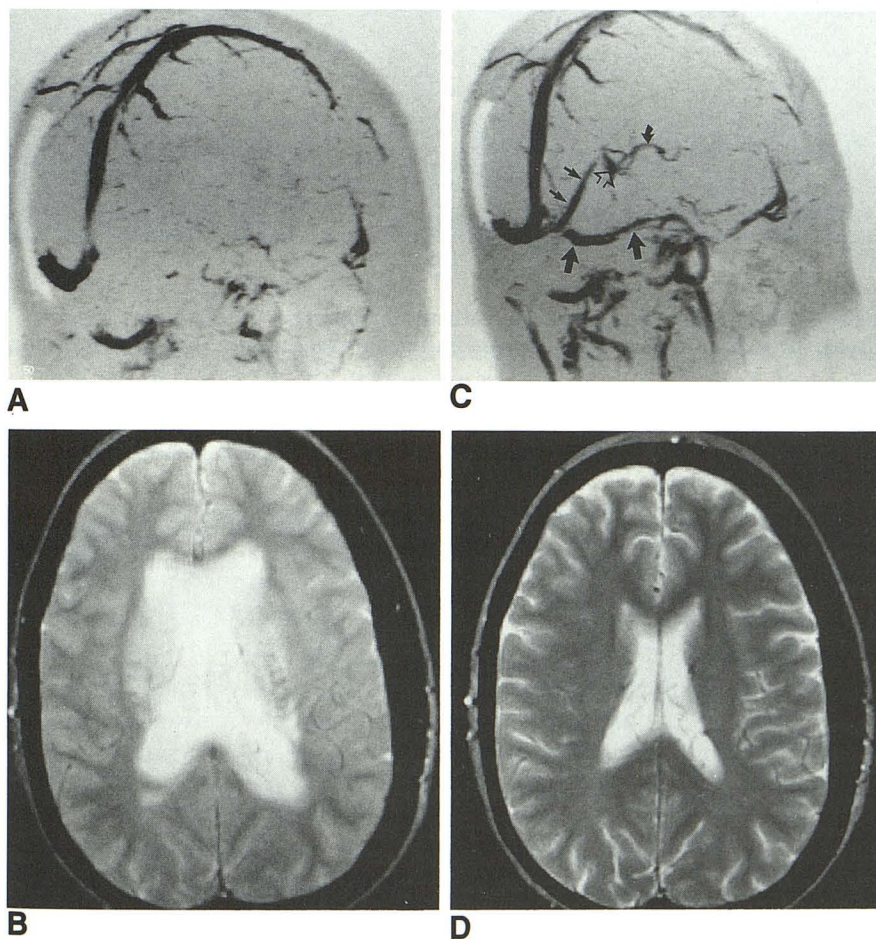


Fig. 3. This 22-year-old woman developed left transverse and deep venous occlusion 3 months postpartum.

A, A two-dimensional sequential oblique parasagittal gradient-echo 31/10/1 (repetition time/echo time/excitations) MR angiogram with flip angle of 50° shows lack of flow in the left transverse and deep veins.

B, Spin-echo 2500/100/2 T2-weighted image shows periventricular edema, mild hydrocephalus, and sulcal effacement. This is the second pattern of VSOD diagrammed in Fig. 1D.

C, Gradient-echo MR angiogram (32/8/1, flip angle 40°) performed 2 weeks after successful thrombolytic therapy shows flow in previously absent deep veins. *Large straight arrows* indicate transverse sinus; *small straight arrows* indicate straight sinus; *open arrow* indicates vein of Galen; *curved arrow* indicates internal cerebral vein.

D, Axial T2-weighted spin-echo image (2500/100/2) shows complete resolution of the periventricular edema and mild hydrocephalus accompanied by a complete neurologic recovery.

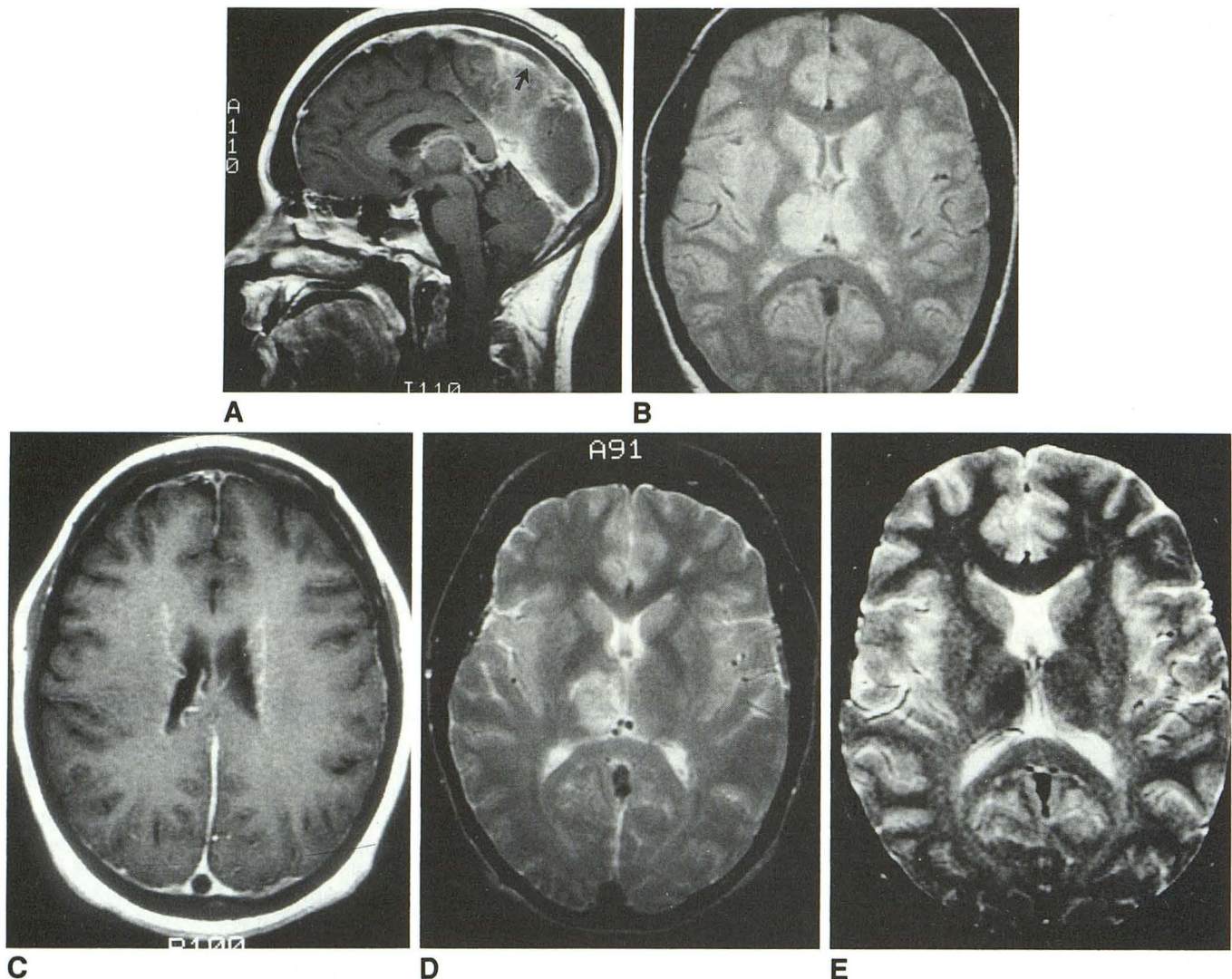


Fig. 4. This 29-year-old woman taking oral contraceptives developed superior sagittal sinus thrombosis.

A, Midsagittal gadolinium-enhanced T1-weighted spin-echo image (350/20/2) demonstrates a filling defect (*arrow*) in the sinus posteriorly.

B, Initial T2-weighted spin-echo image (2000/30/2) shows focal periventricular and thalamic abnormal signals seen on T2-weighted imaging.

C, Gadolinium-enhanced axial T1-weighted spin-echo image (350/20/2) demonstrates deep venous enhancement in the periventricular region. There was no associated arterial or parenchymal enhancement.

D, T2-weighted spin-echo image (2000/80/2) obtained 5 days after B shows partial resolution of the signal abnormality seen on the T2-weighted images.

E, Three months after B, the edema has resolved, and residual hemosiderin (dark signal) is present in the thalamus, suggesting previous petechial hemorrhages (spin-echo 2500/100/2).

vena cava (26) is a known phenomenon. Also, hydrocephalus is associated with otitis media: the so-called otitic hydrocephalus (27–31). This was more common in the preantibiotic era. Bering and Salibi (23) produced hydrocephalus in 74% of dogs by occluding cephalic venous drainage. They postulate that the mechanism responsible for ventriculomegaly relates to 1) decreased CSF absorption caused by elevated dural sinus pressure and 2) increased CSF production caused by

increased pulsation of the choroid plexus. Both of these mechanisms may result in a communicating hydrocephalus, which may explain the mild ventriculomegaly seen in our patients with the second pattern of VSOD.

The cause of the phenomenon of increased shift of interstitial bulk fluid from the vascular bed toward the ventricle because of impaired venous drainage, as suggested by the MR findings in our patients, is unknown. We suggest that the

second stage of VSOD develops because of increased venous pressure causing an increase in CSF production through the choroid plexus or shift of interstitial fluid into the ventricle. Also, absorption of the CSF in the arachnoid granulations may be depressed because of elevated venous sinus pressure. All of these mechanisms can result in a communicating hydrocephalus and periventricular edema.

Eventually, when the elevated venous pressure exceeds the structural limit of the venous wall, rupture occurs resulting in a parenchymal hematoma. This is the third MR pattern seen in our patients with VSOD. Associated high intensity signal on T2-weighted images is usually observed near the periphery of the hematoma (Fig 5). Because the onset and severity of clinical symptoms is usually much milder in VSOD than in arterial occlusive disease, and because symptoms and signal abnormalities are often reversible if venous drainage is restored, tissue damage appears to be predominantly the result of hematoma rather than simple venous obstruction or ischemia. The mechanism of tissue damage is probably similar to that of lacunar infarction when intraparenchymal hemorrhage occurs secondary to abnormally elevated arterial blood pressure. Although VSOD can result in a decrease in arterial blood flow because of poor venous drainage, the degree of ischemic injury is not as severe as expected. This is further supported by the infrequent occurrence of infarction or atrophy in the follow-up studies in our patients.

When patients are treated for the underlying venous-occlusive process by successful thrombolytic treatment (22, 32, 33) or by a sinojugular venous graft (34), the abnormal signal, dilated ventricles, and clinical symptoms may rapidly

resolve. This was seen in five of our patients. For example, one of our patients presented with grave clinical symptoms and abnormal signal on T2-weighted images associated with sinus thrombosis and brain swelling without evidence of a hematoma (Fig 3). After endovascular thrombolytic treatment with urokinase successfully restored venous sinus flow, this patient had prompt and complete recovery with resolution of signal abnormalities. Thus, the high intensity signal on T2-weighted images should not always be considered as an infarction. It could represent interstitial edema: a reversible process. This is further supported by a recent report of complete or nearly complete symptomatic recovery after urokinase treatment in five VSOD cases with evidence of edema on CT (19). As an example, one patient had a sinus pressure of 27 mm Hg before treatment and a pressure of 8 mm Hg after successful endovascular therapy. The associated edema resolved after treatment. Therefore, cortical sulcal effacement and abnormal signal on T2-weighted images likely represent reversible venous congestion and pressure-driven interstitial edema. Such abnormalities associated with VSOD should not be considered venous infarctions. Early intervention may resolve clinical symptoms and normalize the MR appearance (22, 32, 33).

In conclusion, the MR findings of VSOD are quite different from those of arterial occlusive disease. These include the absence of abnormal arterial or parenchymal enhancement, persistent brain swelling that is greater than the associated signal abnormality, periventricular effusion with associated ventriculomegaly (both can be reversible), and a greater incidence of hematomas than seen with ischemic infarction. Breakdown of the blood-brain barrier does not appear to occur in

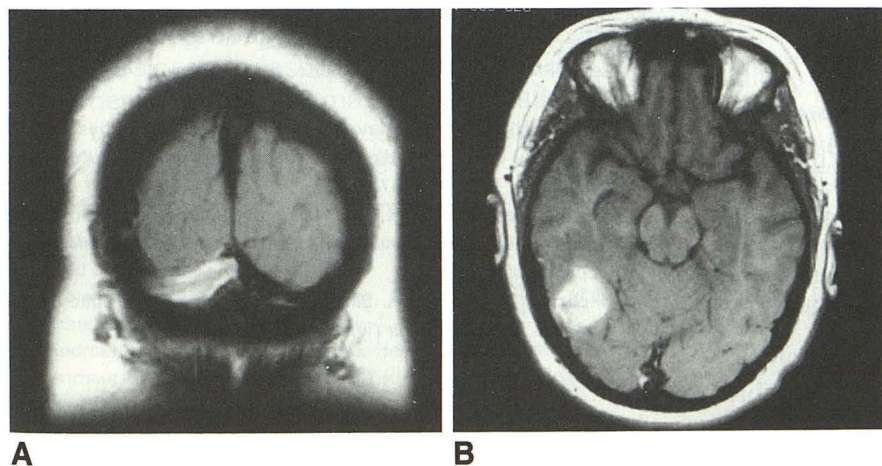


Fig. 5. This 72-year-old man developed right transverse and sigmoid sinus occlusion as a complication of mastoiditis. Spin-echo 300–500/20/2.

A, Coronal T1-weighted image shows lack of flow in the right transverse sinus.

B, T1-weighted image demonstrates the intraparenchymal hematoma with surrounding edema. This is the third pattern of VSOD diagrammed in Fig 1E.

VSOD; therefore, abnormal parenchymal enhancement may not be seen. The causes for the different patterns seen in VSOD remain unknown and require further study. However, from our limited data, these findings may be explained in part by the degree of elevation in venous pressure and the hypothetical model of bulk water shift through the interstitium from the capillary bed to the ventricle. The abnormal signal on T2-weighted images, particularly involving the thalamus and basal ganglia, likely represents a reversible change caused by increased interstitial fluid driven by a pressure gradient and should not always be considered as an infarction. Irreversible tissue damage appears to be more related to hemorrhage and to a lesser degree to ischemia. Early intervention to resolve the underlying venous occlusion can be beneficial, particularly in those patients with the first or second pattern of MR findings.

Acknowledgment

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