IMAGING OF SUPERIOR SAGITTAL SINUS THROMBOSIS: A REVIEW

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ABSTRACT

Ten cases of superior sagittal sinus thrombosis have been retrospectively reviewed. The focus of this study is on the CT appearance of the superior sagittal sinus thrombosis. The radiological diagnosis of superior sagittal sinus thrombosis in the present study includes either the "empty delta sign" on contrast-enhanced CT or the absence of flow on MR venography or conventional angiography. The pathognomonic empty delta sign was presented in 5 cases (50%). Seven cases developed focal parenchymal lesions, all of which were venous infarct with or without intracerebral hemorrhage at parasagittal frontoparietal region. In case of superior sagittal sinus thrombosis that lacks specific imaging sign, recognition of the pattern of hemorrhagic infarct may suggest the CT-diagnosis of this disorder. MR venography or conventional angiography has also been used to confirm the diagnosis of superior sagittal sinus thrombosis. Among the other common causes of superior sagittal sinus thrombosis, rarely oral amphetamine abuse was reported in one of our cases. Detailed findings will be presented.

INTRODUCTION

The cerebral venous and dural sinus occlusive disease is a severe disease with nonspecific clinical feature and a high index of suspicion is necessary to make an early diagnosis. Radiological investigations are mandatory for the diagnosis of cerebral sinovenous occlusion. CT findings of cerebral venous sinus occlusion can be classified as normal, suggestive, pathognomonic or abnormal but non-specific one.[1-7] The " empty delta sign " on contrast-enhanced CT that was first described by Buonamo et al in 1978 is pathognomonic for superior sagittal sinus thrombosis.[2] This sign represents the enhancement of the dura and surrounding collateral venous channels with hypodense central thrombus. The sensitivity of the empty delta sign varies from 28 to 72%.[1,4-6] On non-contrast CT, the thrombosed veins and dural sinuses can be visualized directly as foci of significantly increased density, "cord sign", for the first week after the thrombosis has formed. The radiological diagnosis of venous sinus thrombosis also includes the absence of flow on MR venography or conventional angiography. In the present study, the CT appearances of superior sagittal sinus thrombosis were retrospectively reviewed.

MATERIALS AND METHODS

Ten cases of superior sagittal sinus thrombosis diagnosed at Maharaj Chiang Mai Hospital over the 4-year period, from 1995 to 1998, have been retrospectively reviewed. There were 7 females and 3 males, the ages of which ranged from 1 month to 49 years. The imaging techniques used in this study included computed tomography, conventional film-screen or digital subtraction angiography and MR venography. Computed axial tomograms with and without contrast enhance-

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ment were performed in all cases. Additionally, conventional angiography was performed in 3 cases, MR venography was performed in 4 cases. For MR venography, the 2D TOF MR venogram was created by acquiring section 1.5 mm in thickness in the coronal plane. A pre-saturation pulse is placed inferior to the imaging volume to reduce the signal from the arterial structures. The midline sagittal 2D PC MR angiography was also performed by using velocity encoding 10-20 cm/sec. The diagnosis was based on the empty delta sign on contrast-enhanced CT or a partial or complete absence of filling of superior sagittal sinus on conventional angiogram or lack of flow in the sinus on MR venogram. The reported CT signs were divided into those visualized primarily on non-enhanced CT scans and those seen on enhanced CT scans.

RESULTS

The age, sex, symptoms and signs, predisposing conditions or associated factors, specific radiographic findings and outcomes of all ten cases are shown in Table 1. The CT findings with and without contrast enhancement are shown in Table 2.

Table 1. The age, sex, symptom and sign, predisposing factor, specific radiographic finding and outcome of ten cases

Case No. Age/sex	Symptom/sign	Predisposing condition	Diagnostic finding			Outcome
			Empty Delta sign	MRV	CA	
1. 49/F	Headache Vomiting seizure papilledema Hemiparesis	Hormone replacement therapy	Negative	+	_	Death
2. 33/F	Headache, seizure, Hemiparesis	Nephrotic syndrome	Negative	+	_	Clinically improved
3. 30/F	Headache, vomiting, Seizure	Oral contraceptive	Negative	+	_	Clinically improved
4. 40/F	Headache, hemiparesis	Idiopathic	Negative	+	_	Clinically improved
5. 29/F	Headache, vomiting, hemiparesis	Coagulation disorders	Positive	_	+	Clinically improved
6. 28/F	Headache, vomiting, seizure	Oral contraceptive	Positive	_	+	Clinically improved
7. 15/M	Headache, vomiting, seizure, hemiparesis	Oral Amphetamine	Negative	_	+	Clinically improved
8. 1mo/M	Fever, seizure	E. coli meningitis	Positive	_	_	Clinically improved
9. 40 days/F	Fever, seizure, infected scalp wound	Osteomyelitis & meningitis	Positive	_	_	Clinically improved
10. 49/M	Headache	Osteomyelitis & epidural epyema	Positive	_	_	No neurodeficit

MRV = MR venogram, CA = conventional angiogram

Table 2. The CT findings

CT findings in case No.	1	2	3	4	5	6	7	8	9	10	Total
On non-contrast CT:											
Low attenuation of white matter	+	+	+	+	+	+	+	-	-	-	7
Location	f	fp	fp	fp	fp	fp	f				
Unilateral		+	+	+		+	+				5
Bilateral	+				+						2
Intracerebral hemorrhage	+	+	-	+	+	-	+	-	-	-	5
Cord sign	+	-	+	-	-	-	-	+	-	-	3
Effacement of sulci or ventricles	+	+	+	+	+	+	+	H	+	-	9
On contrast-enhanced CT:											
Empty delta sign	-	-	-	-	+	+	-	+	+	+	5
Leptomeningeal enhancement	-	-	-	-	-	-	-	+	+	_	2

f = frontal region, p= parietal region, H = hydrocephalus

All adult cases presented with symptom of headache. Seizure developed in 7 cases. Five cases had focal neurological deficit (hemiparesis). Three cases had septic causes, included meningitis in 2 pediatric patients and skull osteomyelitis with epidural abscess in a 49 year-old man. The diagnosis of superior sagittal sinus thrombosis in these 3 cases were based on CT findings of empty delta sign alone (Fig. 1). The radiographic finding of associated infections (i.e., Leptomeningeal enhancement due to meningitis, osteomyelitis of skull and epidural abscess) were clearly demonstrated on CT without evidence of cerebral infarction or hemorrhage. The other three cases were associated with oral contraceptive and hormonal replacement therapy. In each of the remainder cases, the underlying causes were idiopathic, nephrotic syndrome, coagulation disorder, and oral amphetamine abuse.

Patient No.7 was 15-year-old man presenting with severe headache and vomiting. Three days later he had seizure followed by left hemiparesis. His CT scan showed right frontal intraparenchymal hemorrhage with white matter edema (Fig. 2A). The initial suspected diagnosis was ruptured arteriovenous malformation. The digital subtraction angiogram revealed occlusion of superior sagittal sinus (Fig. 2B), that was the cause of hemorrhagic infarct. He was later found to have a history of oral amphetamine abuse.

On non-contrast enhanced CT, brain swelling was the most common finding (90%), demonstrated by effacement of cortical sulci or ventricles. Low attenuation of frontal or frontoparietal white matter which was not confined to arterial territories is seen in 7 cases (5 unilateral and 2 bilateral). Intraparenchymal hemorrhage was seen in five cases with hemiparesis. On contrast-enhanced CT, empty delta sign was seen in 5 cases. Leptomeningeal enhancement was demonstrated in 2 cases with underlying meningitis. Hydrocephalus secondary to E. coli meningitis was seen in patient No.8.

Four cases of superior sagittal sinus thrombosis were diagnosed by MR venography. The MR venogram revealed absence of flow in the superior sagittal sinus (Fig. 3). Before MR imaging became available in our hospital, conventional film-screen angiography was performed in two of our cases to confirm the diagnosis of superior sagittal sinus thrombosis.

As a result of superior sagittal sinus thrombosis, one patient died, where as the remainders recovered fully or with neurological deficits.

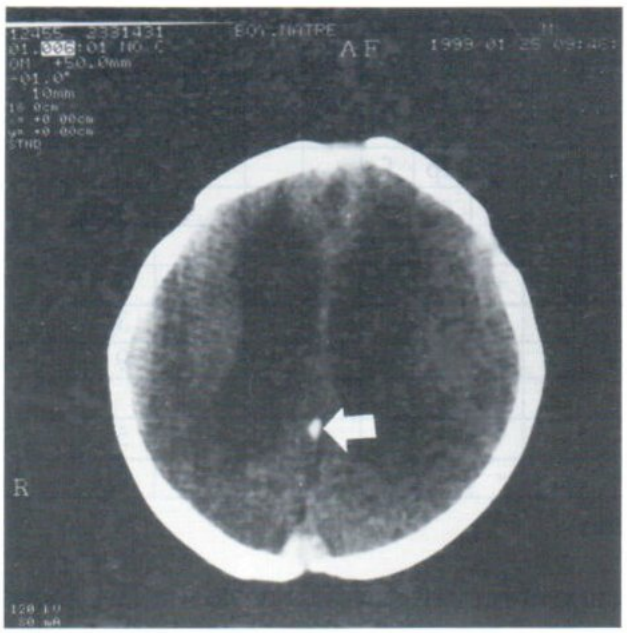

Fig. 1A Axial NECT scan shows intraluminal thrombus, cord

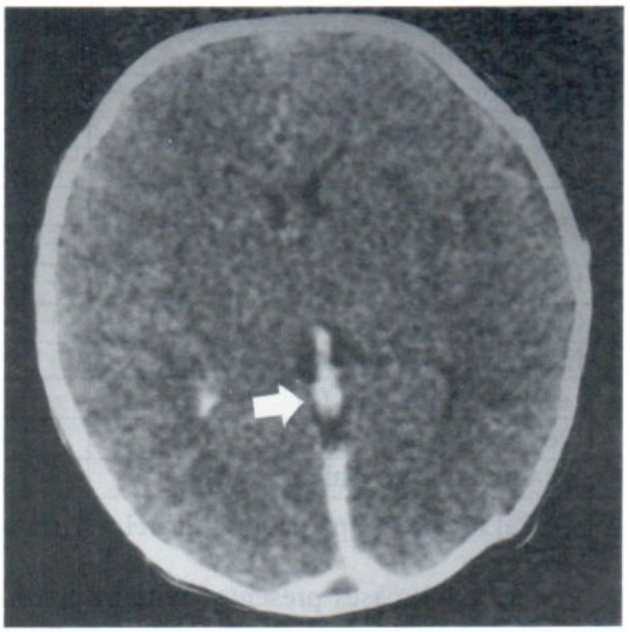

Fig. 1B Axial CECT scan reveals an empty delta sign which is caused by enhancing dura surrounding intraluminal clot.

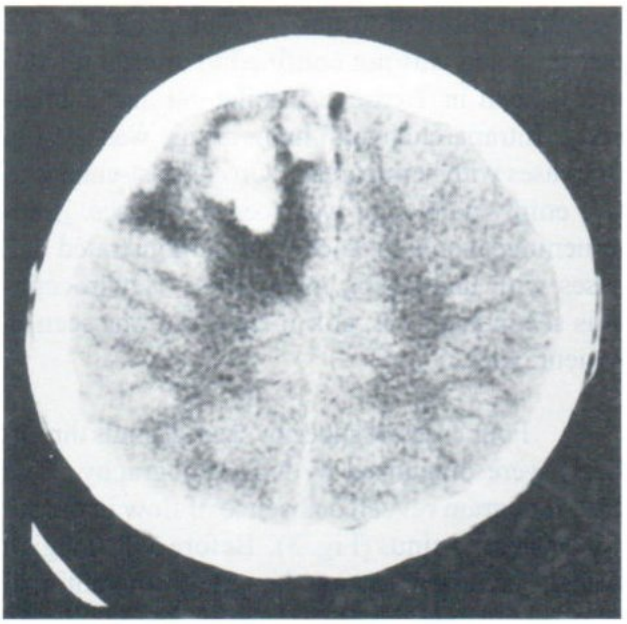

Fig. 2A 15-year-old man, with oral amphetamine abuse, presented with headache, vomiting, seizure and left hemiparesis. A, Axial NECT scan shows low-attenuation of right frontal white matter with central area of hemorrhage.

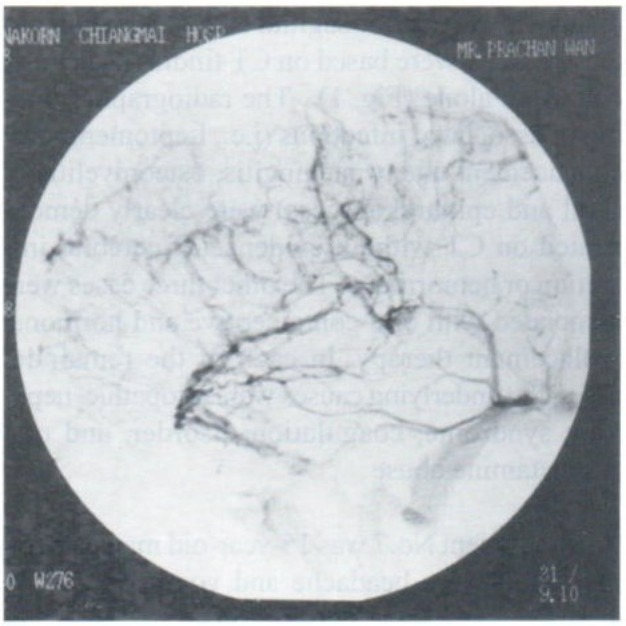

Fig. 2B Lateral view of digital internal carotid angiogram, late venous phase, shows nonfilling of the superior sagittal sinus.

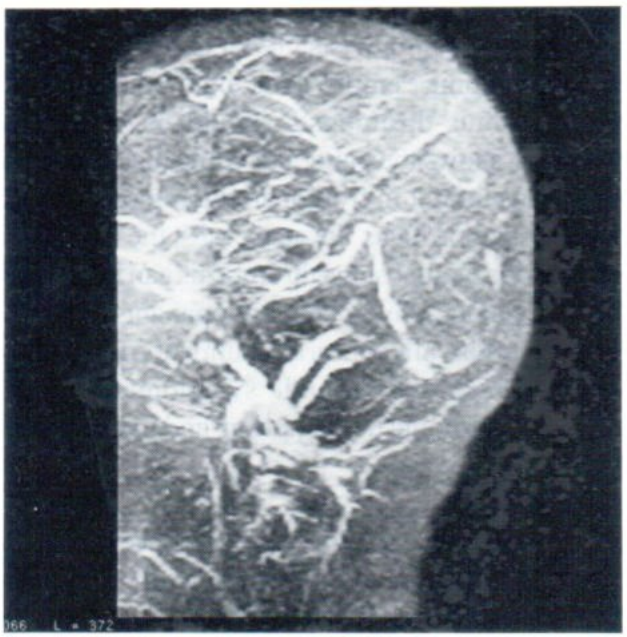

Fig. 3 2D TOF MR venogram shows lack of flow in the superior sagittal sinus.

DISCUSSION

Cerebral venous sinus occlusion is a serious, potentially lethal disease that was described for the first time in a French literature in 1825. In a review of 306 articles on dural sinus thrombosis published between 1983 and 1990, the incidence is uncertain, range from 0.1% of consecutive autopsies to 10% of all deaths due to cerebrovascular disease.[1] Occlusion of the dural sinuses or cortical veins can obstruct blood flow through the arteriocapillary bed and, consequently, lead to ischemia and infarction. In contrast to arterial infarct, venous infarct is primarily affect the white matter rather than cortex and is not confined to arterial territories. Considering the locations of occluded dural sinus, the superior sagittal sinus is the most common one, followed by the transverse, sigmoid and cavernous sinuses respectively. The mortality rate of superior sagittal sinus thrombosis was about 57% (59 of 103 cases) in a study of Schell and Rathe in 1988. In our study, the mortality rate was 10%; one died and all of the survivors recovered with and without neurological sequelae.

In general, cerebral venous occlusion may occur from primary aseptic causes, such as oral contraceptives, pregnancy, puerperium, dehydration, hematological disorders, Behcet's disease, antiphospholipid syndrome and idiopathic conditions. The infectious process that involves the soft tissue of the face, vascular structures, or meninges, can be secondary causes of cerebral veonus thrombosis.[1] Interestingly, amphetamine abuse, which is a well-established cause of cerebral vasculitis, is also reported in the present study as the cause of superior sagittal sinus thrombosis. Previously, there have been reports concerning the association between cerebral vasculitis and intracerebral hemorrhage in cases of oral amphetamine abuse.[8-10] To our best knowledge, therefore, this is the first report of superior sagittal sinus thrombosis and hemorrhagic infract associated with oral amphetamine abuse.

The clinical manifestations and prognosis of cerebral venous sinus occlusion depend on the region and extent of venous occlusion, the rapidity of the developing occlusions, and the available collateral venous flow.[1-2] Typically, in cases of superior sagittal sinus thrombosis, the patients usually present with symptoms and signs of increased intracranial pressure, with or without focal signs. It is theoretically believed that focal infarction or intracerebral hemorrhage arises from extension of thrombus into the cortical vein and, consequently, causes seizure and/or focal neurological deficits. Common symptoms and signs of these patients are headache, confusion, seizures, papilledema, and hemiparesis. In our study, headache was the most common and earliest symptom, being followed in incidence by convulsion and hemiparesis. We found that hemiparesis was highly associated with frontoparietal intracerebral hemorrhage (100%) and usually carried poor prognosis. Bilateral hemorrhagic infarctions were found on the CT of patient No.1 who died. Four

cases had had hemiparesis that were incompletely recovered following the treatment.

In our study, the empty delta sign was presented in five cases (50%). This sign may not be apparent in the extreme cases with acute or chronic stages of superior sagittal sinus thrombosis.[7] Several pitfalls in the use of the empty delta sign have been mentioned elsewhere.[4] Briefly, the empty delta sign must be seen on adjacent sections at different levels in order to exclude high splitting of the superior sagittal sinus, which may mimic the presence of this sign. Another pitfall is the failure to review the study with a variety of window settings. Partial volume effect or artifact may limit visualization of thrombus in the superior sagittal sinus because the sinus is immediately adjacent to the skull. In patients with head trauma or non-traumatic subarachnoid hemorrhage, the "pseudo-delta sign" is caused by acute subarachnoid or subdural hemorrhage along the falx and tentorium surrounding the unclotted blood in the sinus. The cord sign was presented in three cases of our study (30%). However, CT may visualize mild hyperdense unclotted blood in the venous sinuses that resemble thrombus, owing to high hematocrit and low-density unmyelinated brain of the newborn.

When CT shows normal or nonspecific findings, the diagnosis of superior sagittal sinus thrombosis is quite difficult. Non-specific signs are diffuse or localized brain swelling and focal parenchymal lesions, mainly infarcts and occasionally hemorrhage.[1] In our study, these lesions were found in 7 cases that developed focal parenchymal lesion (venous infarction with or without hemorrhage) in parasagittal frontoparietal region (Fig. 4). Unilateral involvement was seen in 5 cases and bilateral asymmetrical involvement was seen in only 2 cases. Hemorrhagic infarction was seen in 5 cases with hemiparesis. Even in the absence of pathognomonic "empty delta sign", the superior sagittal sinus thrombosis should be suspected when CT shows the pattern of white matter edema or venous infarct in frontoparietal region, with or without hemorrhage. In such cases, the diagnosis of superior sagittal sinus thrombosis can be proved by MR venography or conventional angiography. This therefore allows early treatment of this condition, which may provide symptomatic relief, prevent neurologic deterioration, and increase survival.

MR venography has been accepted as a rapid, noninvasive and effective method to confirm the diagnosis of venous thrombosis.[11-13] The most commonly used method to obtained MR venogram is to create a 2D TOF venogram by acquiring section 1.5-2 mm in thickness in the coronal plane. The phase-contrast MR angiogram is also useful for differentiating flow from thrombus formation.[12] The midline sagittal 2D PC MR angiogram using velocity encoding of 10-20 cm/sec can displays flow in the sagittal sinus, internal cerebral vein, vein of Galen and straight sinus. The axial 2D PC MR angiogram also demonstrates flow in the transverse sinuses, sigmoid sinuses and jugular veins. The diagnosis of superior sagittal sinus thrombosis is based on a partial or complete absence of flow in the superior sagittal sinus. On conventional SE images the occlusion of superior sagittal sinus is seen as absence of flow void and intra-luminal intermediate-to-high signal of clot. Conventional angiography is an accurate but invasive method for assessment of the cerebral vessels. Where MRI and MRA are not available or contraindicated, conventional angiography remains the standard tool for the diagnosis of thrombosis.[14]

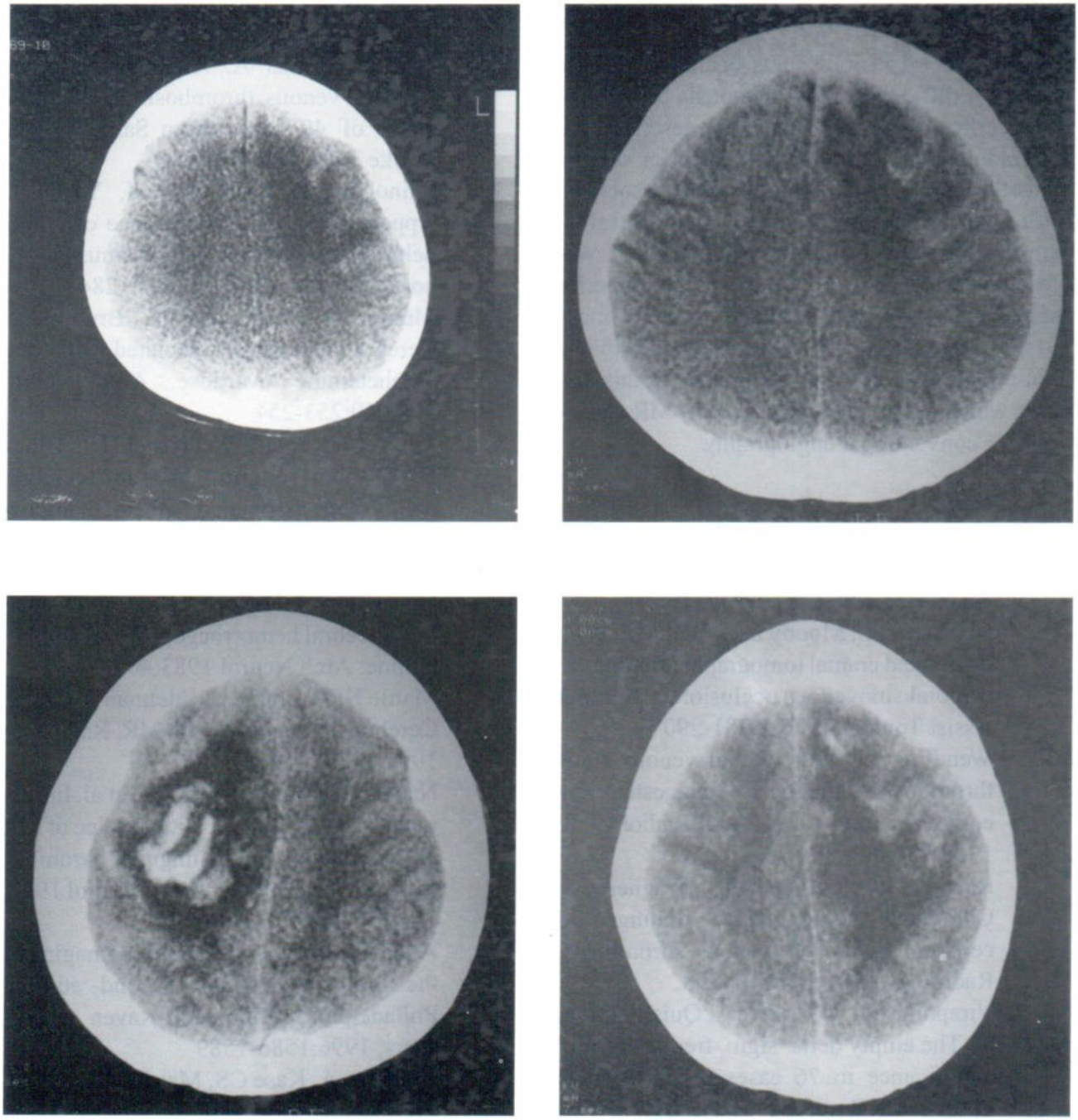

Fig. 4 CT findings of venous infarcts with and without associated intraparenchymal hemorrhage in four different cases.

CONCLUSION

Cerebral venous sinus occlusive disease remains a difficult clinical diagnosis because of nonspecific symptoms. This study described the CT findings in 10 patients with superior sagittal sinus thrombosis, including one case associated with oral amphetamine abuse. The classical "empty delta sign" on contrast-enhanced CT was presented in 5 cases (50%). In case of superior sagittal sinus thrombosis that lacks specific imaging sign, recognition of the pattern of hemorrhagic infarct may suggest the CT-diagnosis of this disorder, which can be, later on, proved by MR venography or conventional angiography.

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