Cerebral Venous Thrombosis: A Potential Mimic of Primary Traumatic Brain Injury in Infants

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OBJECTIVE. Hyperdense venous thrombi on unenhanced head CT may be misinterpreted as different types of extraaxial hemorrhages, and hemorrhagic venous infarctions may be interpreted as parenchymal contusion, leading to an incorrect diagnosis of trauma as the cause of the blood products. The purpose of this article is to show the various appearances of cerebral venous thrombosis (CVT) that mimic different types of hemorrhages and to show hemorrhagic venous infarctions that mimic parenchymal contusions.

CONCLUSION. CVT, as an entity, must be kept in the differential diagnosis when patients present with extraaxial hyperdensities on unenhanced head CT so appropriate management can be initiated to minimize potentially devastating consequences.

Cerebral venous thrombosis (CVT) is often an elusive diagnosis both clinically and radiologically despite increasing awareness of the disease process. Several review articles in the past 10 years describe this entity in detail [1–5], however, the difficulty in diagnosis lies partly with patients presenting with a variety of symptoms ranging from objective focal neurologic deficits to subjective generalized malaise or headaches. The presentation is even less specific in infants, with decreased level of consciousness and seizures being the most common presenting symptoms [3].

The basic teaching regarding differential diagnosis of extraaxial hyperdensities on unenhanced CT is often limited to hemorrhage. Yet, the appearance of clotted blood outside the vessel (hemorrhage) or inside the vessel (thrombus) is the same on imaging. CVT is an important entity that does not readily come to mind when radiologists are interpreting hyperdensities on unenhanced CT of the head.

This case series illustrates the various appearances of CVT mimicking different types of hemorrhages and hemorrhagic venous infarctions mimicking parenchymal contusions.

Cases

The cases summarized in Table 1 with corresponding Figures 1–5 were collected over a period of 3 years from 2007 through 2009. The age of the patients ranged from 0 days to 7 months. All of the patients were male. All cases had at least one unenhanced CT of the head and follow-up brain MRI. Two cases had MR venography (MRV) and one case was evaluated with CT venography. All imaging; initial radiology reports; and available medical records, including birth histories, were reviewed.

Discussion

The best way to diagnose CVT is to have a high index of suspicion and incorporate a vigorous search of the venous structures into the search pattern. Whether a blood clot is a result of hemorrhage or thrombosis, the principles dictating the appearance on CT or MRI are the same. When presented with findings of a hyperdense area on unenhanced CT of the head, it is imperative to correlate the appearance with the anatomy in the region. A thrombus within a venous structure may cause expansion, resulting in a masslike lesion mimicking either a subdural or an epidural hematoma, as was depicted in case 1. Cortical venous thrombosis may mimic subdural or subarachnoid hemorrhage depending on the course of the vessel, as illustrated by the presented cases. The association between intraventricular hemorrhage and CVT in neonates had been previously reported [6–9]. Wu et al. [7] found 31% of neonates had intraventricular hemorrhage secondary to CVT. Thalamic hemorrhage is another clue to the possibility of coexistent CVT [7, 9]. Not uncommonly, CVT leads to parenchymal venous infarctions that are often hemorrhagic.

Keywords: abusive head injury, cerebral venous thrombosis, hemorrhage, subdural hematoma, trauma

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TABLE 1: Details of the Cases

Patient No.	Age	Initial Presentation	Initial Imaging Report	Actual Imaging Findings	Birth History	Comments
1	10 d	Irritability, poor feeding, seizure	CT: hyperdensities representing right occipital EDH, ISDH, SAH, IVH, contusions	CT: hyperdensities representing DVS thrombosis (initially as EDH and ISDH), cortical venous thromboses (initially as SAH), IVH, hemorrhagic venous infarcts (initially as contusions), no extraaxial hemorrhage MRI–MRV (at > 29 h): confirmed CVT	Vacuum-assisted vaginal delivery without complications	Mother positive for antithrombin III deficiency, patient not tested at the time of admission
2	7 wk	Altered consciousness, emesis, poor feeding	CT: hyperdensities as SDH and contusions, right frontoparietal skull fracture. MRI (at > 4 wk) after traumatic encephalomalacia	CT: hyperdensities corresponding to diffuse CVT involving all DVS, no extraaxial hemorrhage, age-indeterminant skull fracture away from DVS, left parietal calcified cephalohematoma MRI: residual CVT and encephalomalacia from venous infarction	NSVD, 1 wk in specialty nursery for poor feeding and emesis	No coagulopathy workup performed
3	7 mo	Lethargy, seizures, recent diarrhea, poor feeding	CT and MRI (at > 62 h): bilateral SDH with acute component on right as linear hyperdensity	CT and MRI: small bilateral hygromas following CSF signal and mildly prominent subarachnoid spaces, no extraaxial hemorrhage, thrombosed cortical vein along the right frontal lobe representing linear hyperdensity on CT	Preterm labor at 30 wk, mother treated with magnesium sulfate and bed rest, delivery at 38 wk	No coagulopathy workup performed
4	2 wk	ALTE and hypothermia	CT: questioned thrombus vs hemorrhage adjacent to vermis	CTV (at > 3 h), MRI–MRV (at > 12 h): confirmed thrombus in the straight sinus, no extraaxial hemorrhage	Unknown	Diagnosed with group B streptococcal meningitis
5	0 d	Immediately after delivery with low Apgar score: 0, 0, and 3 at 1, 5, and 10 min, respectively	CT: questioned linear hyperdensity in the sulcus as SAH vs cortical venous thrombosis	MRI (at > 3 d): thrombosed cortical vein with adjacent venous infarct, no extraaxial hemorrhage	Low Apgar score	No coagulopathy workup performed

Note—EDH = epidural hemorrhage, ISDH = interhemispheric subdural hemorrhage, SAH = subarachnoid hemorrhage, IVH = intraventricular hemorrhage, DVS = dural venous sinuses, MRV = MR venography, CVT = cerebral venous thrombosis, NSVD = normal spontaneous vaginal delivery, ALTE = acute life threatening event.

These focal hemorrhages can be misinterpreted as traumatic parenchymal contusions, especially if extraaxial hemorrhage is reported instead of CVT, leading to a misdiagnosis of trauma as the cause of the blood products.

The hyperdense nature of a thrombus on unenhanced CT will usually resolve within 7 days but may be present longer in cases with larger clots. Beyond this subacute phase, CVT may not be easily identified on CT. Awareness of the time frame and appearance of the degradation of blood products is again a key step in the appropriate diagnosis and further workup. MRI with MRV is a more definitive study to evaluate for CVT. CT venography is an excellent modality to depict thrombi in the dural venous sinuses, but because of radiation exposure, it should be reserved for cases where MRI–MRV cannot be obtained. However, cortical and medullary venous thromboses may not be conspicuous on conventional MRI sequences or MRV because of anatomic variations, asymmetries, and small size of the vessels. To better evaluate the abnormalities of these veins, a gradient-recalled echo sequence or a newer susceptibility weighted sequence should be added to the protocol to help identify paramagnetic blood products along the course of thrombosed veins.

The underlying causes of CVT are numerous, with infection and dehydration identified as the most common causes. There are only a few studies, mostly case reports and small series, that discuss CVT in the setting of trauma [10–16]. In the great majority of published cases, traumatic CVT was focal, most commonly in the transverse or sigmoid sinus on the side of a skull fracture. In one of the larger studies with 195 patients looking at blunt trauma and CVT [13], CVT was found only in patients with a skull fracture crossing one of the dural venous sinuses, and no CVT was found in groups with a fracture away from sinuses or in the group without skull fractures. Although a few case reports have attributed CVT to trauma in patients without a skull fracture, such conclusions should be drawn carefully in cases in which there is no history, physical examination, or radiologic studies that reveal definitive signs of acute trauma.

The severity of trauma in the publications varied greatly: a head hitting a table [12], a 1-m fall from a chair [11], being hit by an object falling from a 2-m height [16], and a motor vehicle accident [14, 15]. The extent of CVT did not seem to correlate with severity of trauma. It

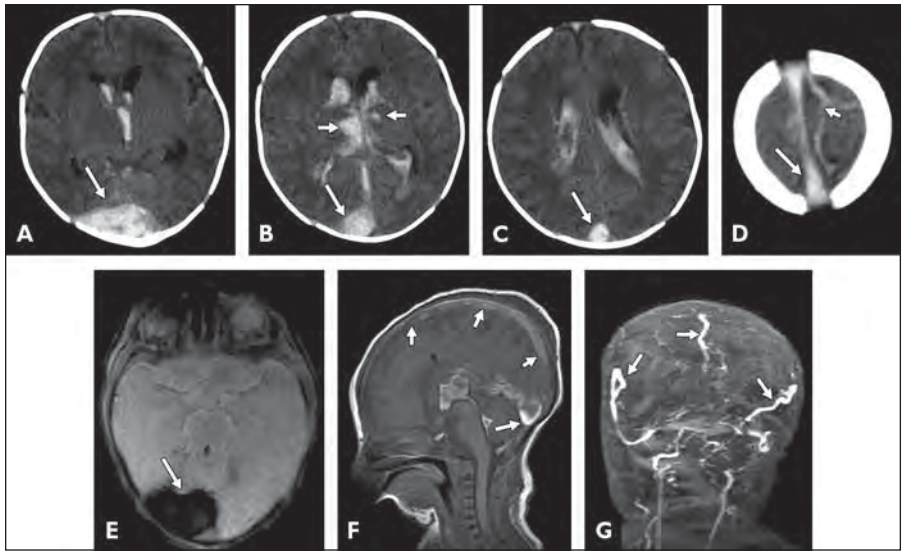

Fig. 1—10-day-old boy with irritability, poor feeding, and seizure (case 1).
A–D, Axial CT images show hyperdense thrombus (**A**) expanding right transverse sinus (*long arrow*). Bilateral thalamic hemorrhage and edema is present (*short arrows*, **B**). Additional thrombus is seen in superior sagittal sinus (SSS) (*long arrows,* **B–D**). Smaller curvilinear hyperdensities at vertex are thrombosed cortical veins (*short arrow*, **D**).
E and **F,** T2-weighted gradient-recalled echo images show thrombosed right transverse sinus (*arrow*, **E**) that could be traced into thrombosed torcula (*long arrow*, **F**) and thrombosed SSS (*short arrows*, **F**) in which normal flow voids are replaced with abnormal hyperintense signal from thrombus.
G, Image from 3D projection from 2D time-of-flight MR venography shows complete lack of flow-related enhancement in dural venous sinus, with only patent dominant veins laterally, likely veins of Labbé and parasagittal vein (*arrows*).

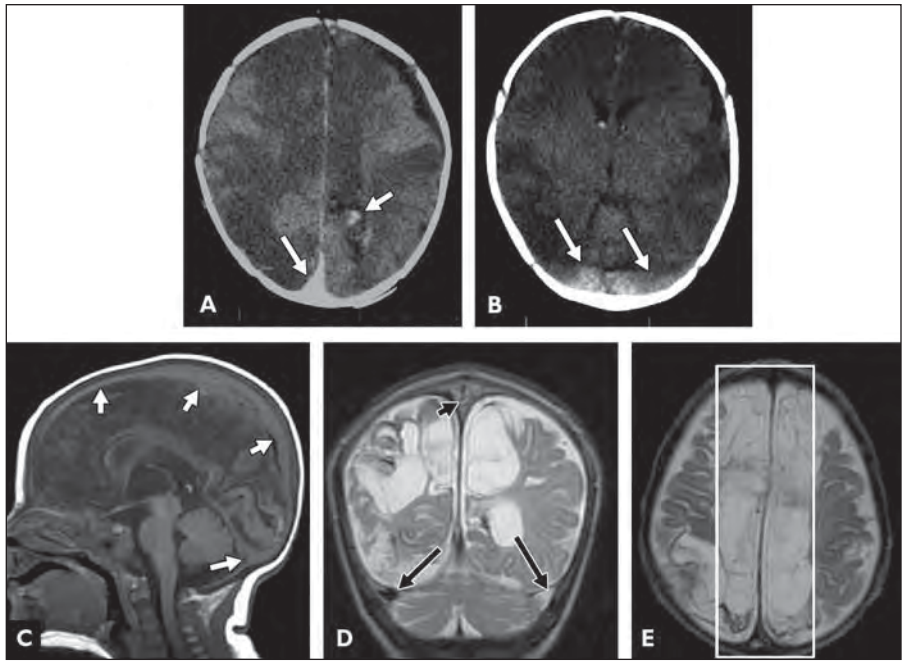

Fig. 2—7-week-old boy with altered consciousness, emesis, and poor feeding (case 2).
A and **B,** Axial CT images show hyperdense triangular configuration of superior sagittal sinus (SSS) (*long arrow*, **A**) as well as hyperdensities along transverse sinuses (*arrows*, **B**), consistent with cerebral venous thrombosis (CVT). Small hyperdense region in left parietal lobe (*short arrow*, **A**) is most likely hemorrhagic venous infarction given diffuse CVT.
C, T1-weighted MR image shows abnormal signal along SSS (*short arrows*) and in torcula (*long arrow*).
D and **E,** Coronal and axial T2-weighted images show thrombus in long SSS (*short arrow*, **D**) and in transverse sinuses (*long arrows*, **D**). Right transverse sinus appears to have flow void, likely from interval recanalization. Initial CT showed thrombus in that location. Encephalomalacia from venous infarction is seen bilaterally, predominantly in distribution drained by SSS (*box*, **E**).

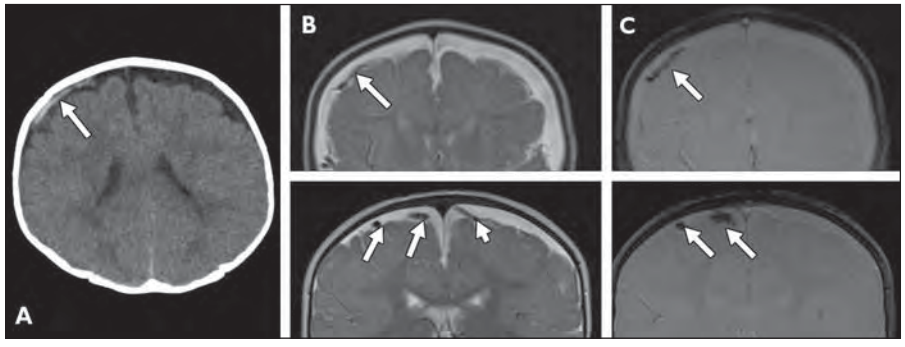

Fig. 3—7-month-old boy with lethargy, seizures, recent diarrhea, and poor feeding (case 3).
A, Axial CT image shows curvilinear hyperdensity in right frontal extraaxial space (*arrow*), most compatible with thrombosed cortical vein rather than acute hemorrhage.
B, Two consecutive coronal T2-weighted images show course of right cortical vessel (*long arrows*). Note left cortical vessel (*short arrow*), which is patent and therefore has no abnormal signal on gradient-recalled echo (GRE) images.
C, Corresponding consecutive coronal GRE images show susceptibility artifact from intravascular thrombus (*arrows*).

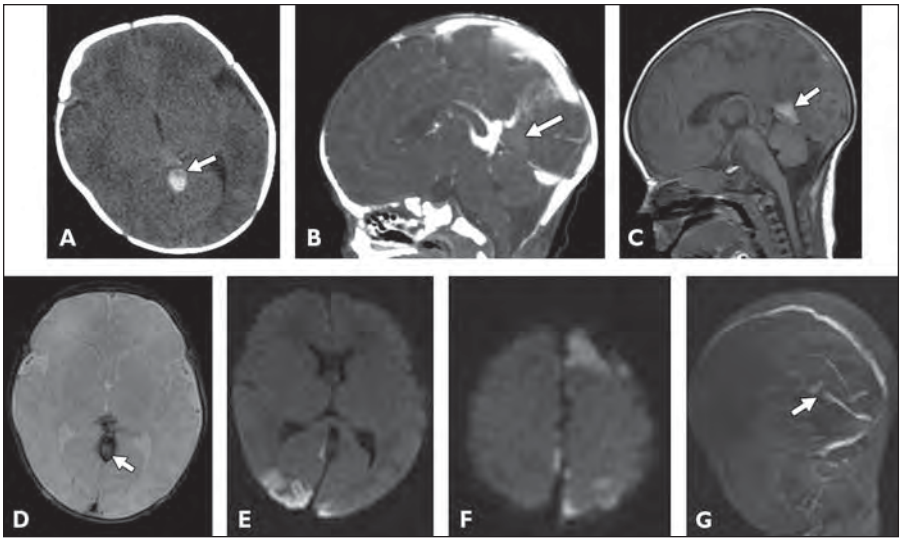

Fig. 4—2-week-old boy who had acute life-threatening event and hypothermia (case 4).
A, Axial CT image shows focal hyperdensity in midline just superior to cerebellar vermis (*arrow*).
B, Reformatted sagittal image from CT venogram shows thrombus in straight sinus (*arrow*) corresponding to CT abnormality.
C and **D,** Sagittal T1-weighted (**C**) and axial gradient-recalled echo (**D**) images show thrombus in straight sinus (*arrows*).
E and **F,** Diffusion-weighted MR images show abnormal cortically based signal, compatible with infarctions from septic vasculitis in setting of bacterial meningitis.
G, Image from 3D projection from 2D time-of-flight (TOF) MR venography (MRV) shows absence of flow-related enhancement in region of thrombus (*arrow*). Flow defect on TOF MRV is not as conspicuous because of presence of methemoglobin in thrombus.

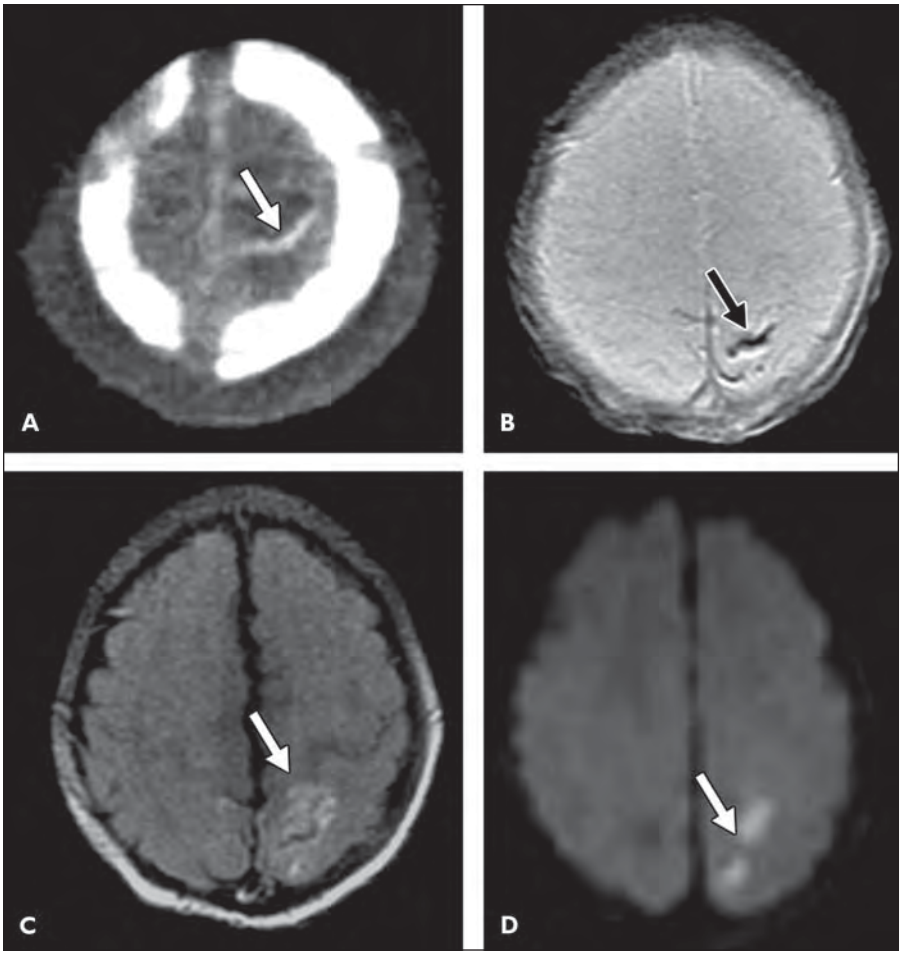

Fig. 5—Newborn boy immediately after delivery with low Apgar score (case 5).
A, Axial CT image shows linear hyperdensity within sulcus (*arrow*), suggestive of thrombosed cortical vein rather than small focal subarachnoid hemorrhage.
B, Gradient-recalled echo (GRE) image shows susceptibility artifact (*arrow*), corresponding to CT abnormality.
C and **D,** Axial FLAIR image (**C**) has signal abnormalities (*arrow*, **C**) compatible with small venous infarction in region of occluded cortical vein. Diffusion-weighted image (**D**) shows diffusion abnormality (*arrow*) that can be seen with venous infarctions. Note mild relative hyperdensity of falx on CT image with background of low density brain, which is normal appearance in newborn and does not represent thin subdural hemorrhage. This normal falx is supported by absence of susceptibility along falx on GRE sequence.

is important to recognize that a significant number of patients in these reported cases presented with delayed signs and symptoms, as long as 2 weeks after a traumatic event [12]. This has significant implications for police and child protective services investigations if CVT is suspected to be a result of abusive head trauma. It is difficult to establish the precise timing between the inciting event and the development of CVT as well as the time between the onset of CVT and symptom onset. Berfelo et al. [5] recently reported a range from 0 to 28 days before onset of symptoms from CVT that consisted mainly of seizures.

Another complicating factor is a high prevalence of prothrombotic risk factors in the population that develops CVT after any inciting event. Heller et al. [17] published one of the larger series on children with CVT and showed that more than 50% of children who developed CVT after an inciting event had a prothrombotic risk factor and many had more than one. A case report by Rich et al. [12] revealed a protein S deficiency in a 4-year-old child who developed CVT after a minor head bump. In another case report, an antiphospolipid antibody was found in a 7-year-old child with CVT after a car accident [10]. Therefore, it is imperative to screen for specific abnormalities such as Factor V Leiden, *G2021A* mutation, lipoprotein (a), protein C, protein S,

antithrombin, and antiphospholipid antibodies. In patients with these risk factors, even a minor or unnoticed trauma may increase the likehood of developing CVT. Perhaps, in some cases, it may be prudent to screen parents, siblings, and other close relatives.

Conclusion

Radiologists must keep CVT within the differential diagnosis when a blood clot is identified on head CT. The importance of distinguishing between hemorrhage and thrombosis is paramount because delay in the appropriate management can have devastating consequences.

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