CASE REPORT Open Access



A brief review of dural venous sinus injury with a short case series

Ajaya Kumar Ayyappan Unnithan*

Abstract

Background: Dural venous sinus injury results in torrential hemorrhage and exsanguination. The initial review by Harvey Cushing had shown high mortality. The depressed fractures over sinuses were managed conservatively, because of the risk of massive hemorrhage during elevation. The consequences of sinus injury are hemorrhage, intracranial hypertension, brain swelling and venous infarct. Topical hemostatic agents such as gelatin sponge, oxidized cellulose, hemostatic matrix, bioresorbable plate, tissue-glue-coated collagen sponge, and fibrin glue help in controlling bleeding during surgery. The aim of the study is to analyze five cases of traumatic dural venous sinus injury. The literature review was done from Pubmed, Scopus and Google Scholar. A retrospective and descriptive analysis is done.

Case presentation: Among the five cases described, four had injury to superior sagittal sinus and one had injury to the transverse sinus. Two patients had open wounds. Three patients had continuous bleeding and two had intracranial hypertension. Decompressive craniectomy was done in three patients, wound hemostasis and closure in one, and craniotomy and evacuation of hematoma in one. Compression with Gelfoam was the main method for hemostasis and thrombin–gelatin hemostatic matrix had to be applied in two patients, with continued pressure for one hour.

Conclusions: Injury to the dural venous sinus should be suspected in fracture over the venous sinus. Surgery is indicated in active bleeding, expanding hematoma, compression or occlusion of the sinus and intracranial hypertension. Topical hemostatic agents help in controlling the bleed. Morbidity and mortality are more with involvement of middle and posterior third of superior sagittal sinus.

Keywords: Cranial venous sinus, Craniocerebral trauma, Craniotomy, Gelatin sponge, Oxidized cellulose

Background

Injury to the dural venous sinus is a challenge to the neurosurgeon. The importance of venous sinus injury is the possibility of rapid and torrential hemorrhage, especially if it is from an open wound [1]. The essential task is to identify the injury early to prevent hypovolemic shock. Harvey Cushing's observations during the first World war was that surgery might prevent the progressive venous thrombosis in wounds involving the major venous sinuses [2]. Cushing's review of 219 military head injuries had 14 cases of dural sinus injury with a mortality

rate of 79% [3]. An analysis of cases with dural sinus injuries by Meier et al. showed a high mortality rate of 41% and intra-operative mortality rate of 20% [4]. The main causes of mortality are presence of severe head injury and profuse bleeding [1]. During surgery, there is chance of exsanguination. Early effective hemostasis is the main aim of surgery. Venous infarction and subsequent brain edema can delay neurological recovery.

The aim of the study is to analyze five cases of traumatic dural venous sinus injury, treated by the author. The study design is descriptive and retrospective analysis. The presentation, imaging findings, surgical management, method of hemostasis and outcome are studied. A literature search was done in Pubmed, EMBASE, Scopus and Google Scholar for articles describing traumatic dural

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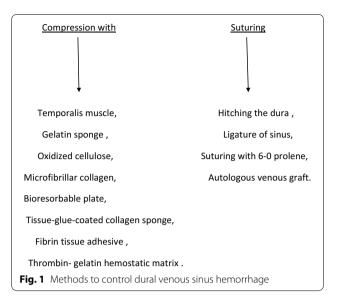


venous sinus injury, imaging modalities and the methods of treatment. English was the language selected. Available articles and the articles in the reference list were analyzed, without any restriction of period. Reviews, case series, case reports, diagnostic studies and technical notes were assessed. The five cases of venous sinus injury are analyzed in the light of the review.

Review of the literature: The chance of hemorrhage is high in fractures or sutural diastasis over the dural venous sinuses [1, 3]. Depressed fracture over a sinus should be considered to have caused injury to the sinus. It can cause occlusion and thrombosis of the sinus also [5, 6]. The incidence of significant dural sinus injury was 1-5% of head injuries in earlier series [1, 3]. But it has come down to 1.7%, in the series of Kim et al. [1]. The anterior and middle third of superior sagittal sinus (SSS) is involved most frequently (60-80%), followed by transverse sinus and then posterior third of SSS [1–6]. Torcular Herophili and sigmoid sinus also were involved in the series of 112 patients published by Meirowsky [2]. The anterior third of SSS can be ligated safely [1, 3]. The consequences of sinus injury are hemorrhage, intracranial hypertension, brain swelling and venous infarcts [5-8].

Imaging: Multi-detector tomography(MDCT) scan and the recent advancement of three dimensional reconstruction(3 D CT) can show depressed fractures and sutural diastasis over sinuses very well [9, 10]. Intracranial air bubbles adjacent to the fracture line, contusions, and hemorrhages can be seen in CT scans [9]. The 'swirl' sign, hypodensity within the hyperdense bleed, indicates active bleeding and chance of further expansion of haematoma [11, 12]. CT venography (multislice contrast-enhanced CT)can show occlusion of venous sinus [9, 13]. The 'empty delta' sign, in CT venography (CTV), represents a filling defect within a venous sinus [9]. Extrinsic compression of venous sinus secondary to a depressed skull fracture or extra-axial hematoma can be distinguished from venous sinus thrombosis by CT venography [13]. Magnetic resonance imaging can differentiate venous extradural haematoma [14]. MR venography (MRV) is a less invasive method to evaluate dural venous sinuses. Digital subtraction angiography (DSA) is the standard, but more invasive, investigation to know the patency of the venous sinus [6].

Indications for surgery: The depressed fractures over sinuses were managed conservatively, because of risk of massive hemorrhage during elevation [15]. But surgery is necessary in open fracture to clear the contamination and to repair the dura to reduce the chance of infection [1, 5, 16]. Conservative treatment can be tried if the dura is intact, and bone displacement is less and the wound can be cleaned [17]. Active bleeding from an open wound can produce rapid exsanguination and should



be intervened rapidly [10]. An expanding hematoma in serial CT scans and the 'swirl sign' are indications for emergency surgery [11]. Other conditions necessitating surgery are occlusion of sinus and intracranial hypertension [5, 6, 8, 16–20]. The venous thrombosis can lead to venous infarction, venous hypertension, and neurological deficits [16, 18]. Relieving the compression and repair of sinus injury will bring neurological improvement.

Surgery: The surgeon should be prepared to deal with bleeding from the sinus. Sufficient packed red blood cell (PRBC) units and fresh frozen plasma (FFP) should be arranged. Blood pressure should be monitored continuously and shock should be avoided. Surgery carries high morbidity and mortality. Craniotomy should be done with extra care to avoid further injury to the sinus. The evacuation of hematoma also should be gentle. Compression with gelatin sponge (Gelfoam) or temporalis muscle can reduce the bleeding [1, 3, 6, 21]. Other agents useful for topical hemostasis are oxidized cellulose (Surgicel), microfibrillar collagen and bioresorbable plate [22]. Topical hemostatic agents containing thrombin can be used. Tissue-glue-coated collagen sponge (TachoSil) and fibrin tissue adhesive (fibrin glue) also have been used successfully [23]. Hitching the dura to the adjacent periosteum and application of ligature or clip in the rostral part of sinus also may be needed to reduce the bleeding [1, 3, 4]. Suturing of the laceration with fine materials such as 6-0 Prolene can be done if proximal and distal control of sinus can be achieved [24]. Autologous venous graft also can be used for the repair [1, 3]. Decompressive craniectomy has to be done in case of intracranial hypertension due to venous edema or infarction [25].

The methods of hemostasis are summarized in Fig. 1.

Outcome: In the series of 112 cases of dural sinus injury, Meirowsky reported 13 deaths (11.6%) [2]. Meier et al. reported in a series of 39 cases that mortality ranged from 17% for injury to the anterior one-third of the superior sagittal sinus, to 100% for injury to the posterior part of the superior sagittal sinus [4]. The mortality for combined injuries of different sinuses was 67% and for transverse sinus injuries was 29%. In the report of surgical treatment of intracranial venous sinus injury for 111 cases by Huang SQ, the cure rate was 77.5% [26]. Elkatatny et al. reported mortality of 15%, and morbidity as moderate disability for 5%, in a series of 20 cases of traumatic dural venous sinus injuries [27]. The causes of morbidity are coagulopathy, intracerebral hemorrhage, and sinus occlusion [1, 3, 27].

Case presentation

Case1

Clinical condition: A 35-year-old male was brought after a two-wheeler accident, with an open wound in the midline anterior to the vertex. There was profuse bleeding. He was in shock with systolic blood pressure of 80 mm of Hg and was unconscious. Glasgow Coma Scale (GCS) was E1M1V1 (eye opening, motor response, verbal response) and pupils were 2 mm and not reacting to light. He was intubated and ventilation was started. Fluid resuscitation was started. The wound was padded.

Imaging: Computerized tomography (CT) scan of brain was done and it showed multiple comminuted fractures in midline with interhemispheric bleed and subarachnoid hemorrhage over the underlying parenchyma (Fig. 2a). Bone fragments were seen piercing down in the midline and into the parenchyma (Fig. 2b). Anterior third of superior sagittal sinus (SSS) was injured and it was the cause of profuse bleeding.

Surgery: He was shifted to operation theater. Wound was opened and the fragments were removed rapidly. Gelfoam (absorbable gelatin sponge) pieces were placed. Wound was closed in two layers with non-suction drain. Six packed red blood cell units (PRBC) were given and dopamine infusion also was started. Blood pressure got normalized on the second day.

Outcome: On the third day he began to respond. His G.C.S became $E2M5V_{ET}$. CT brain was repeated and showed bifrontal hypodensities suggestive of venous infarcts. He was weaned from ventilator slowly. He improved to a state of consciousness.

Follow-up: He had impairment of short-term memory, behavioral changes and emotional disturbances such as increased rage. Magnetic resonance imaging (MRI) revealed bifrontal gliosis (Fig. 2c). Magnetic resonance venogram (MRV) showed narrowing of anterior SSS (Fig. 2d). He is on follow-up for 12 years. He did not

develop any symptoms of raised intracranial pressure in this period.

Case 2

Clinical condition: A 24-year-old male was brought in unconscious state after a high-velocity two-wheeler accident. He had facial injuries and profuse oral bleed. He had only flexion response to pain and pupils were 2.5 mm and not reacting to light. G.C.S. was E1M3V1. He was intubated and ventilated.

Imaging: CT head showed multiple facial bone fractures, fracture in right parietal bone extending through coronal suture to bilateral frontal bones and bilateral extra-axial hematoma extending from midline (Fig. 3a, b). The hematoma was mainly on right side with small component on left side. Pneumocephalus was seen inside, from midline and laterally from the fracture site. Diffuse brain edema was seen.

Surgery: Right frontotemporoparietal decompressive craniectomy and evacuation of extradural hematoma was done. Active bleeding was seen from fracture in the midline and from the anterior and middle third of superior sagittal sinus (SSS). The duramater was hitched to periosteum with Gelfoam, to reduce bleeding. Subdural hematoma was evacuated after opening the duramater. Duraplasty was done with artificial substitute (G patch), to reduce the brain edema. Two PRBCs were given. He was ventilated. Pupil size reduced bilaterally. Postoperative CT showed near total evacuation of extra-axial hematoma, except on left side (Fig. 3c). On third day left pupil dilated, suggestive of intracranial hypertension. CT brain was repeated and it showed left frontotemporal hemorrhagic contusions with edema (Fig. 3d). Left decompressive craniectomy and removal of hemorrhage was done. He was ventilated continuously till he slowly improved.

Outcome: By 2 weeks, he was weaned from ventilator. And he had cognitive improvement by 2 months. Bilateral cranioplasty was done after 6 months, with Titanium mesh.

Follow-up: There are no significant posttraumatic sequelae on follow-up after 2 years.

Case 3

Clinical condition: A 30-year-old male had a high-velocity two-wheeler accident. He had multiple long bone fractures—bilateral femur fractures and left tibia and radius fractures. Scalp contusions were there. He was drowsy, but responsive to call. G.C.S. was E3M6V5.

Imaging: CT head showed diastasis of coronal suture and underlying extradural hematoma (EDH) from midline to left (Fig. 4a, b). Initial orthopedic stabilization was done. Follow-up CT brain after 4 h showed enlargement

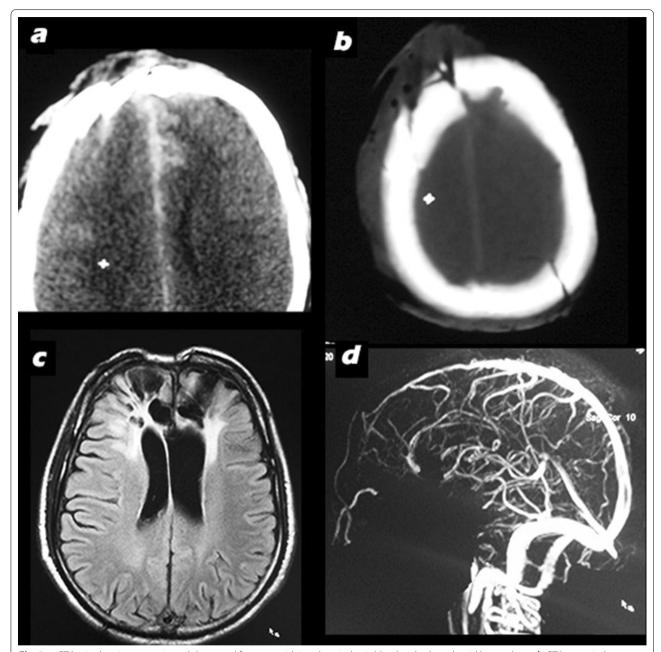


Fig. 2 a CT brain showing comminuted depressed fractures with interhemispheric bleed and subarachnoid hemorrhage; **b** CT bone window showing depressed bone fracture segment in midline; **c** MRI brain showing bifrontal gliosis; **d** MR venogram showing narrowing of anterior third of superior sagittal sinus

of EDH with 'swirl sign' near midline suggestive of active bleeding. Coronal reformat of CT showed hematoma crossing midline to right (Fig. 4c).

Surgery: Left frontoparietal decompressive craniectomy was done under general anesthesia. EDH was evacuated. There was active bleeding from draining veins and the middle third of superior sagittal sinus (SSS). Bleeding

could be stopped only after digital compression for one hour, with Gelfoam soaked in thrombin–gelatin hemostatic matrix (Surgiflo) (Fig. 4d). Bone was not kept back in the wound, to aid decompression. Four units of PRBC were given. Postoperative CT brain showed minimal residual bleed in midline and in right.

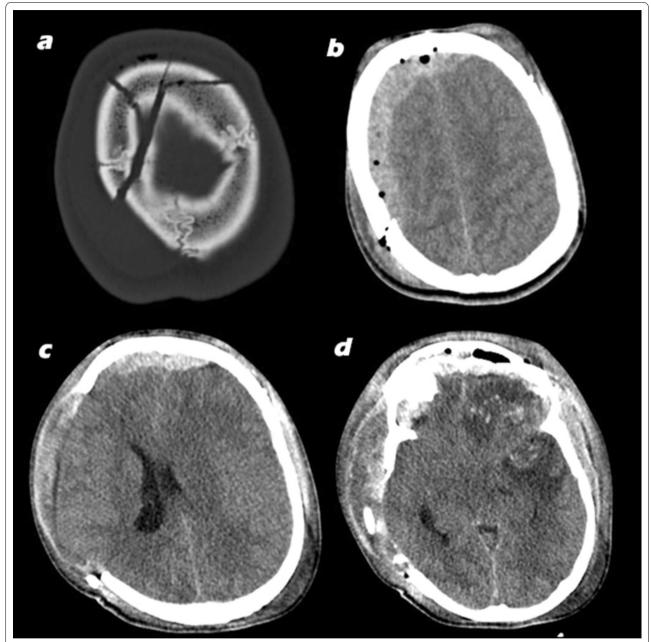


Fig. 3 a Fracture of right parietal bone extending through coronal suture to bilateral frontal bones; **b** extra-axial hematoma extending from midline, mainly on right side with pneumocephalus; **c** CT after decompressive craniectomy showing edema; **d** left frontotemporal contusions with edema

Outcome: Fixation of long bone fractures were done and he improved without any neurological deficits.

Follow-up: He recovered. Cranioplasty was done after 1 month.

Case 4

Clinical condition: A 59-year-old male was brought with bleeding from large open wound of scalp and right pinna

laceration following a three-wheeler accident. He was drowsy, responsive to call. His G.C.S. was E3M6V5. The wound of scalp was large, from right temporal region to left parietal region, with bleeding and clots. Large skull defect was seen (Fig. 5a). The comminuted skull pieces were seen at the site of accident.

Imaging: CT head showed large skull defect in middle and bilateral parietal regions, and extradural bleeding

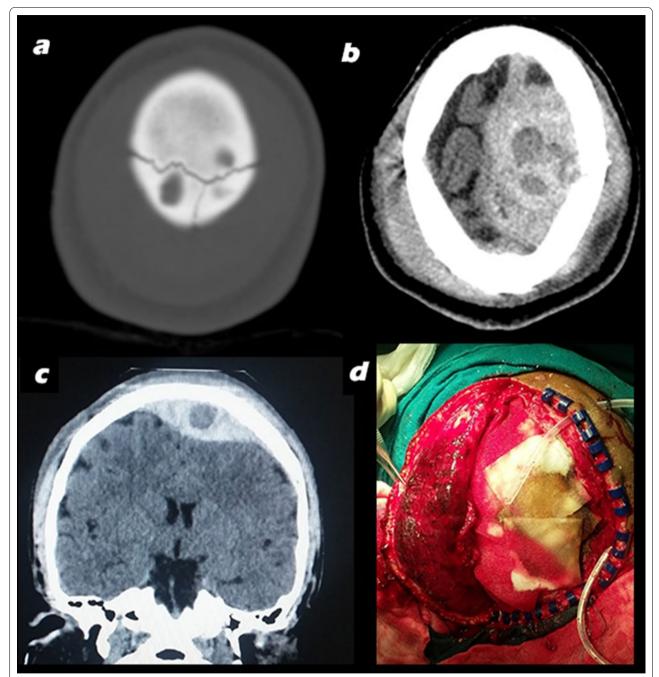


Fig. 4 a Diastasis of coronal suture; **b** extradural hematoma from midline; **c** coronal reformat of CT showing hematoma crossing midline to right and swirl sign near midline suggestive of active bleeding; **d** bleeding controlled by Gelfoam soaked with thrombin–gelatin hemostatic matrix

from midline to left side (Fig. 5b). 3D CT showed the large defect (Fig. 5c).

Surgery: Wound exploration was done under general anesthesia. Lavage was done with saline and hydrogen peroxide. Clots were removed. Active bleeding was seen from the middle third of superior sagittal sinus (SSS). Thrombin–gelatin hemostatic matrix (Surgiflo)

was applied and compression was done with Gelfoam. Wound was closed with drain. Ear repair was done.

Outcome: He improved without any deficit. He had right renal injury, which was managed conservatively.

Follow-up: After 6 weeks, cranioplasty was done with contoured Titanium mesh (Fig. 5d).

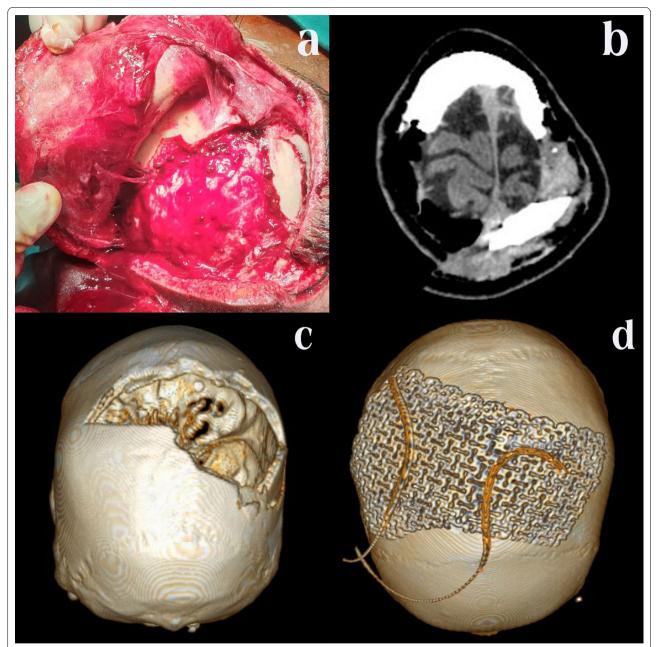


Fig. 5 a The open wound of scalp with bleeding, and large skull defect; **b** CT head showing skull defect, and extradural bleeding from midline to left side; **c** 3D CT showing the large defect; **d** 3D CT after cranioplasty with titanium mesh

Case 5

Clinical condition: A 6-year-old girl was admitted with headache and vomiting after fall at school hostel, from a height of around 8 feet. Her G.C.S. was E4M6V5 on admission.

Imaging: CT brain showed right occipital bone fracture and extradural hematoma (EDH) extending down to posterior fossa. Initial conservative management was done. But she had continuous headache, drowsiness and

vomiting, suggestive of intracranial hypertension. G.C.S. became E3M6V4 on next day. MRI was done on next day. EDH had increased and was compressing right transverse sinus lower end of superior sagittal sinus (Fig. 6a, b). MR Venogram (MRV) showed nonvisualization of right transverse sinus (Fig. 6c).

Surgery: Induction of anesthesia was done. Craniotomy was done, both supratentorial and infratentorial, preserving bone in the region of torcula (Fig. 6d). EDH was

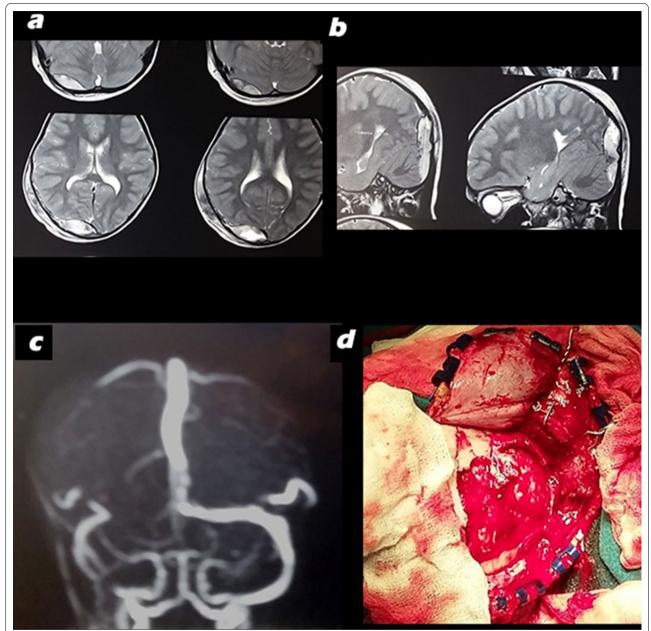


Fig. 6 a MRI showing EDH compressing lower end of superior sagittal sinus; **b** sagittal MRI showing EDH centering transverse sinus; **c** MR venogram showing nonvisualization of right transverse sinus; **d** bleeding from the region of transverse sinus controlled with Gelfoam

evacuated. Oozing from the region of transverse sinus was controlled with Gelfoam.

Outcome: She improved without any deficits.

Follow-up: She did not have any symptoms on follow-up up to 6 months.

Peculiarities of the cases: Among the five cases described, four were having injury to SSS and one injury to transverse sinus. Three patients had continuous bleeding and two had intracranial hypertension.

Decompressive craniectomy was done in three, only wound hemostasis and closure in one, and craniotomy and evacuation of hematoma in one. Among the hemostatic methods in literature, compression with gelatin sponge was the main method for hemostasis and thrombin—gelatin hemostatic matrix had to be applied in two, with continued pressure for one hour. Hitching of the duramater to the periosteum was done in one patient. The methods used are similar to those described

in the literature [1, 3, 4, 6, 21, 23]. The patient who had open fractures with profuse bleeding and hypotension had bifrontal infarction and behavioral changes. Wide decompressive craniectomy had to be done for involvement of the middle third of SSS. The final outcome was good in all patients in the background of the literature.

Conclusions

Injury to the dural venous sinus and the resultant massive hemorrhage is a challenge to the neurosurgeon. Early diagnosis can be done if high index of suspicion is kept in cases of fractures over the sinuses. Serial CT scan, CT venography and MR venography can diagnose bleeding from the sinus and compression of sinus. The current literature shows that surgery is indicated in active bleeding, expanding hematoma, compression or occlusion of the sinus and intracranial hypertension. Now with the advancements in topical hemostatic agents (such as hemostatic matrix, bioresorbable plate, tissueglue-coated collagen sponge, fibrin glue etc.), bleeding can be controlled more effectively. Repair with autologous venous graft or muscle has to be done in cases with uncontrollable bleeding. Occlusion of middle or posterior third of the superior sagittal results in venous infraction. Five cases were discussed. The patients with involvement of the middle third of SSS required decompressive craniectomy. All of them had good recovery.

Abbreviations

GCS: Glasgow Coma Scale; CT: Computerized tomography; SSS: Superior sagittal sinus; MRV: MR venography; EDH: Extradural hematoma; MDCT: Multi-detector computed tomography; CTV: CT venography; DSA: Digital subtraction angiography.

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Author contributions

Treated the patients, analyzed the cases, and prepared the manuscript. The author read and approved the final manuscript.

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Declarations

Ethics approval and consent to participate

Taken for a retrospective analysis.

Consent for publication

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Competing interests

The author declares that there is no competing interest.

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