Turkish Neurosurgery 2008, Vol: 18, No: 1, 70-77

Dural Sinus Thrombosis Following Head Injury: Report of Two Cases and Review of the Literature

Kafa Travması Sonrasında Gelişen Dural Sinüs Trombozu: İki Olgu Sunumu ve Literatürün Gözden Geçirilmesi

Ali DALGIÇ[1]
Mehmet SEÇER[2]
Fikret ERGÜNGÖR[3]
Önder OKAY[4]
Rıfat AKDAĞ[5]
Deniz CILIZ[6]

[1,2,3,4,5] Ankara Numune Educational and Training Hospital, II. Neurosurgery Clinic, Ankara, Turkey
[6] Ankara Numune Educational and Training Hospital, I. Radiology Clinic, Ankara, Turkey

ABSTRACT

Dural sinus thrombosis (DST) usually involves the sagittal, transverse and sigmoid sinuses and is more common in women due to pregnancy, puerperium and oral contraceptive use. Other etiologies include coagulopathies, infection and head injury. We have present two DST cases following head injury. The first case was a 35-year-old man hospitalized because of one-week history of headache and repeated vomiting after a mild head injury. Thrombosis of the superior sagittal sinus, right transverse and sigmoid sinuses and right jugular vein was determined on angiography. The second case was a 25-year-old man operated on for epidural hematoma at the posterior fossa. Meningitis developed and an abducens palsy was determined. Magnetic resonance imaging demonstrated thrombosis of the right transverse and sigmoid sinus. Low molecular weight heparin was administrated for three months. Both cases had good recovery, but one had recanalisation of the thrombosis. Intracranial hematomas, depressed skull fracture or skull fracture that cross the sinus can obstruct the blood flow in the sinus. Moreover, closed head injury may cause to DST. Because of undefinitive pathophysiology, a consensus was not obtained on overall strategy concerning conservative, radiosurgical, or surgical therapy yet.

KEY WORDS: Dural sinus thrombosis, Head injury, Complication, Surgery

ÖZ

Dural sinüs trombozu özellikle sagittal, transvers ve sigmoid sinüsleri tutar, gebelik, lohusalık dönemi ve doğum kontrol hapı kullanan kadınlarda sık rastlanır. Bunun dışında kan pıhtılaşma bozuklukları, enfeksiyon ve kafa travması etiyolojik nedenlerdir. Bu yazıda kafa travması sonrasında dural sinüs trombozu saptanmış iki olgu sunuldu. Birinci olgu, 35 yaşında erkek, 1 hafta önce geçirdiği kafa travması sonrasında başğarısı ve tekrarlayan kusma yakınması ile yatırıldı. Yapılan anjiyografisinde superior sagittal sinüs, sağ transvers ve sigmoid sinüs ile sağ juguler vende romboz saptandı. İkinci olgu, 25 yaşında erkek, posterior fossa epidural hematomu nedeni ile opere edildikten sonra menenjit gelişmiş ardından abdusens felci ortaya çıkmıştır. Manyetik rezonans görüntülemede sağda transvers ve sigmoid sinüs trombozu saptandı. Her iki olgu da 3 ay düşük moleküler ağırlıklı heparin ile tedavi edildi, klinik olarak iyileşme saptanmasına karşın olgulardan birinde trombüsün çözüldüğü görüldü. İntrakranial hematom, kafatasının çökme kırığı, dural sinüsleri çaprazlayan lineer kırıklar kan akımını engelleyerek tıkayabilir. Kapalı kafa travmaları da böylelikle dural sinüs trombozuna neden olabilir. Patofizyolojisinin henüz açıklıkla ortaya konmamış olması nedeniyle cerrahi, radyolojik veya tutucu tedavi konusunda henüz bir fikir birliği sağlanamamıştır.

ANAHTAR SÖZCÜKLER: Dural sinüs trombozu, Kafa travması, Komplikasyon, Cerrahi

Received: 22.08.2007
Accepted: 20.11.2007

Presented In: This paper was reported in XIX. Annual Meeting of Turkish Neurosurgical Society in 2005

Correspondence address:
Ali DALGIÇ
E-mail : dalgica@superonline.com

INTRODUCTION

Dural sinus thrombosis (DST) usually involves the sagittal sinus (70-80%), transverse and sigmoid sinuses (70%), and may extend to the cerebral veins. In approximately one third of all cases, more than one sinus is involved, and in 30-40 % of them, cerebellar and cortical vein thrombosis is associated (18, 23). The signs and symptoms are extremely varied and nonspecific, ranging in severity from mild headache to progressive neurological deficit, deterioration of consciousness, progressive coma and death related to intracranial hemorrhagic infarction and increased intracranial pressure (1, 4, 11, 23).

DST was first diagnosed post-mortem by Ribes in 1825. DST diagnosis using autopsy or invasive X-ray angiography was difficult until the end of the last century. However, DST has been diagnosed more frequently with the help of the availability of better non-invasive diagnostic techniques, such as Computerized Tomography (CT) and Magnetic Resonance Imaging (MRI) (4,18,23).

The estimated annual incidence is 1.5 to 3 cases per million in adults and 6.7% per million in children (23). DST is responsible for 1-2 % of all strokes in adults (18). Even though DST may affect all age groups, it is more common in women, particularly in the age group of 20 to 35, due to pregnancy, puerperium, and oral contraceptive use. Other etiologies also commonly include coagulopathies, intracranial infection and head injury. Particularly, DST may occur after head injury, usually in the cases of depressed skull fracture, epidural or subdural hematomas (13, 17, 28, 30, 31). In addition, all surgical attempts such as craniotomy, head and neck infections, cranial tumors, deep venous thrombosis, severe dehydration, inflammatory bowel diseases, connective tissue disorders (CTD), sarcoidosis, nephrotic syndrome, lumbar puncture and parenteral injections, and neonatal asphyxia have been reported as the causes of DST (3,17,18,23,27, 29).

In this paper, we present two cases of DST following head injury and review the reported cases in the literature.

Case 1

A 35-year-old man was hospitalized because of one-week history of headache and repeated vomiting after having a mild head injury. Except for the trauma, he did not have a significant history of illness. On examination, he had no abnormalities except left hemiparesis and any dehydration findings. CT scans revealed that there was a hyperdense appearance like intracerebral hematoma on right post-parietal cortical area and contiguous hyperdensity on right tentorial region (Figure 1A-B). At once, anticonvulsant and antiedema therapy were prescribed.

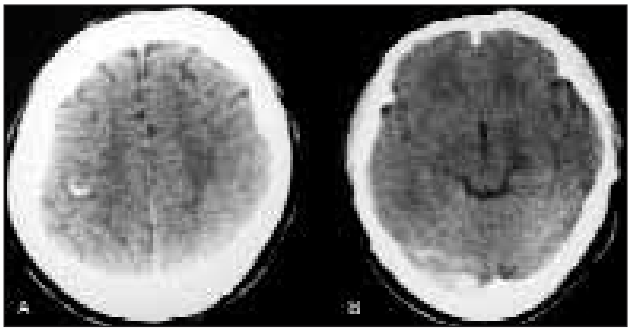

***Figure 1: A** The dense vein sign was seen on the right parietal cortical region on axial CT scan without contrast. **B** Hyperdense changes over right occipital region probably denotes to right transverse sinus on axial non contrast CT scans.*

Lumbar puncture had normal pressure and normal protein and glucose ratio and the cerebrospinal fluid (CSF) was clear. His complete blood count and routine electrolyte measurements were normal. All coagulative and hemostatic function studies, including protein C and S, were normal and rheumatologic parameters such as rheumatoid factor and antinuclear antibody were also normal. Digital subtraction angiography (DSA), performed to explain the causes of both the intracerebral hematoma-like structure in the post-parietal region and the right tentorial area hyperdensity, showed a thrombosis of the superior sagittal sinus, right transversal and sigmoid sinuses and right jugular veins with increased blood flow increased in the cortical collateral vein (Figure 2A-B). The patient was treated with low molecular weight heparin (LMWH) continued for three months. He was discharged at the tenth day without any neurological deficit. After fifteen months follow-up, he was entirely normal on neurological examination, and magnetic resonance angiography (MRA) revealed blood flow in the superior sagittal, right transverse and sigmoid sinus and the right jugular vein (Figure 3).

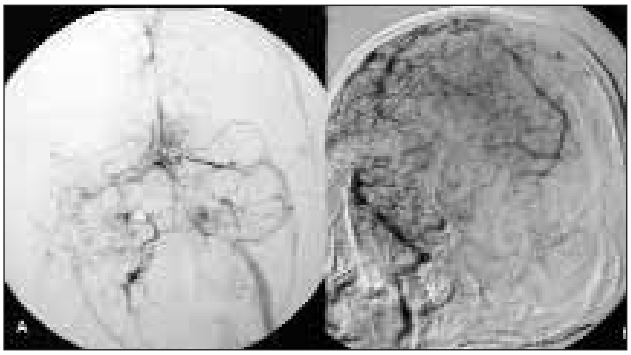

Figure 2: *DSA demonstrated occlusion of right jugular vein, superior sagittal, right transverse and sigmoid sinuses (**A**) while the cortical and deep collaterals are augmented (**B**).*

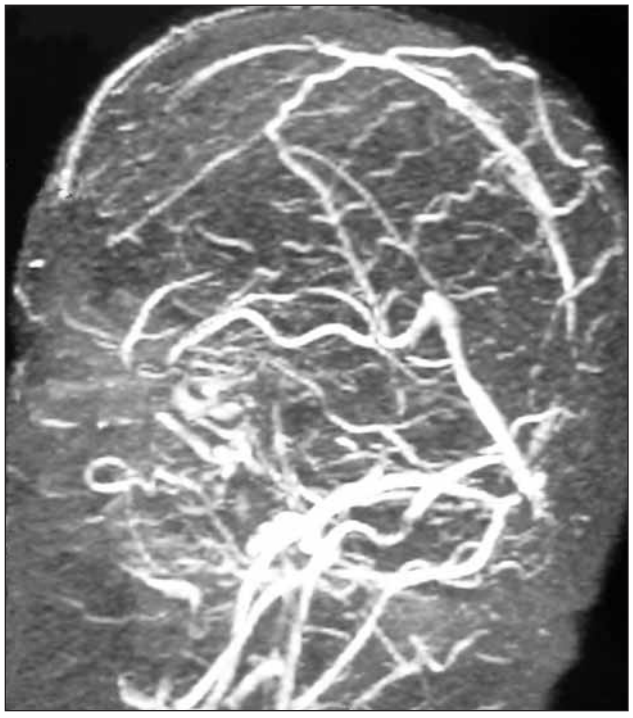

Figure 3: *Superior sagittal, right transverse and sigmoid sinuses and the right jugular vein are all open on MRA after 15 months.*

Case 2

A 25-year-old man was admitted to our emergency department after a traffic accident. The Glasgow Coma Score was 12 points and there was no neurological deficit except right facial palsy. On radiological investigation, there were fractures on the right mandible arc and the right occiput extending to the temporal region on X-rays and there was epidural hematoma contiguous to right cerebellar hemisphere on posterior fossa extending to the occipital region on CT scans (Figure 4A). An urgent operation was performed to remove the hematoma.

He was discharged on the postoperative sixth day with continued right facial palsy. Five days later, he submitted to the clinic due to deteriorated consciousness and high temperature. The examination revealed that he had neck stiffness; lumbar puncture revealed 800 leukocytes in the CSF. Meningitis was diagnosed and antibiotics were administered. Next day, he said that he had been seeing double and abducens palsy was diagnosed. There were no abnormalities apart from the changes due to the surgery in follow-up CT scans (Figure 4B).

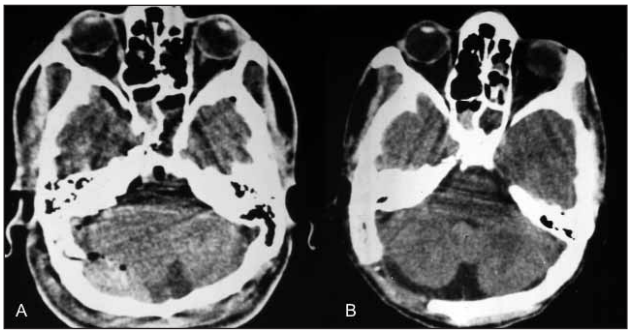

Figure 4: A: *Axial CT scan demonstrating epidural hematoma over right suboccipital area exerting a mass affect on right cerebellum.* ***B:*** *Follow-up CT scan showed complete removal after suboccipital craniectomy.*

MRI was done to evaluate the abducens palsy and demonstrated thrombosis of the right sigmoid and transverse sinuses (Figure 5). LMWH was added to his treatment. After two weeks, he was discharged with relatively improved right facial palsy. At the fourteen months follow-up, he was normal except for mild right facial palsy on neurological examination, and follow-up MRA showed that there was no flow in the right transverse and sigmoid sinus (Figure 6).

DISCUSSION

In 1946, Ecker described the first case of blunt head injury to the skull associated with DST. Since then, other trauma-induced DSTs have been reported in cases of head injuries (8, 12, 28-31). Ochagavia announced that the incidence of DST was 4% after penetrating head trauma (15). However, Stiefel reported that he found DSTs with an incidence of 6.8% in the pediatric age group (25). There are two series on posttraumatic CST in children but sporadic case reports in adults and in children have been

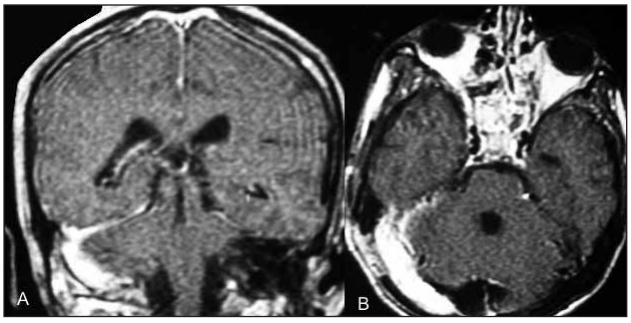

Figure 5: *Coronal (**A**) and axial (**B**) MR images show thrombosis of the superior sagittal sinus, right sigmoid and transverse sinuses.*

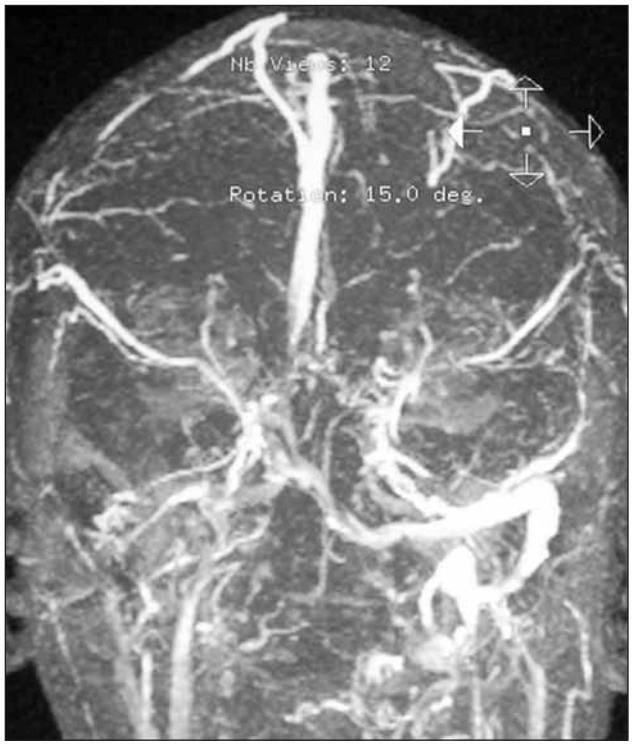

Figure 6: *MRA control shows resolution of thrombosis while flow is stil not visualized on right transverse and sigmoid sinuses.*

published (1,5,6,8,13-16,19-21,25,27,32). Overall, there are 25 cases including 19 children and 6 adults. The higher number of children can be explained by the fact that the venous collateral system is not completely mature in their cerebrum. The superior sagittal sinus is involved more often with thrombosis than the other sinuses in CST patients. Reports of posttraumatic DST have stated that the sigmoid sinus and transverse sinus are generally involved (21 cases) and the superior sagittal sinus affected less often (3 cases). The deep venous system can also rarely be obstructed due to head injury (2 cases).

Recently, it has been reported that a prothrombotic risk factor or a direct cause, such as pregnancy, head trauma, infections and deep venous thrombosis, was identified in about 85 percent of the patients (23). Infections showing local propagation, such as sinusitis, otitis, and mastoiditis, and systemic infections like sepsis or meningitis are known to cause DST (10, 23). Similarly, a case of SSS thrombosis was reported in a case that suffered a furuncle of the scalp and superimposed closed head injury (8). Otherwise, the clinical picture of CTD consists of symptoms and signs related to the vasculitis that occurs on the arterial vascular wall and especially on the venous wall (3). Vasculitic destruction of the vascular wall, especially in the arteries, is similarly found in meningitis. Kastenbauer and Pfister have reported that they diagnosed DST in 9 of 87 cases with meningitis (10). Accordingly, all of these mechanisms, disturbances in the blood flow, damage to the capillary endothelium and vasculitis, facilitate and result in DST. Additionally, factors such as obstruction of the dural sinuses from traumatic rifts of the thin venous channels, compression of the sinuses from intracranial bleeding, and skull fractures crossing the sinuses cause DST (14,27). Owler and Beser have reported a case in which extradural haematoma caused venous sinus obstruction (17) and depressed skull fracture may also cause DST following head injury (13,28,30,31). Bagley reported a case of DST in mild head injury without skull fracture for the first time in 1934. Since then, sporadic case reports (Table I) and two series (Table II) have been published in the literature. The presented case 2 had additional risk factors, such as a skull fracture and epidural haematoma for which he had undergone surgery following which he developed meningitis. Even though head trauma is an independent risk factor for DST, we suspected that DST might have been due to the meningitis since abducens palsy developed after this infection.

The pathogenesis of DST has not been well established yet in head injury. Various hypotheses for the occurrence of DST imply intramural hemorrhages caused by the rupture of small sinusoids, injury to the endothelial lining, extension of the thrombus from injured emissary veins and compression of the sinuses from intracranial edema in the development of DST in closed head injury patients (14,15,17,28,32). Furthermore, abnormalities

Table I: Summary of reported cases with cerebral sinus thrombosis due to head injury in the last two decades.

Author & year	Age, sex	Symptom	Skull fracture	Intracranial lesion	Diagnosis	Thrombosis	Treatment	Follow-up	Outcome
Hesselbrock et al. 1985	44, M	IICPS, seizure	?	contusion	CT, DSA	SSS	Supportive	unknown	MR
Ochagavia et al. 1996	27,M	herniation due to IICPB	-	edama	autopsy	LSS and TLTS	-	-	EX
Ferrara et al. 1998	24, M	IICPS	+	Venous infarct	CT, operative	SSS	Surgery	ligated	WR
Meena et al. 2000	40, M	IICPS, hemiparesi, seizure	-	-	CT, MRV	SSS	AC	unknown	GR
Satoh et al. 2000	2, F	IICPS	-	-	CT, MRI MRV, DSA	LSS	supportive	RC	GR
Brors et al. 2001	32, M	Cranial nerve palys	+	Contusion	CT, MRV	RSS, RTS	AC	RC	GR
Erdogan et al.	1, M	IICPS	-	Venous infarct, SH	CT, MRV	Galen, Rosenthal ICV	supportive	unknown	WR
Owler et al. 2004	18, M	IICPS, hemiparesi	-	Venous infarct	CT, MRI DSA	ICV, pericallosal vein	supportive, surgery	unknown	GR
Sousa et al. 2004	7, F	IICPS	-	-	CT	RSS	supportive	unknown	GR
Muthukumar 2005	7, F	IICPS	+	-	MRV	RTS	AC	unknown	GR
Saad et al. 2005	10, F	IICPS	-	-	CT	RTS	AC	unknown	GR
Yuen et al. 2005	4, F	IICPS	SS	-	MRV	RTS, RSS	supportive	RC	GR
overall	7M, 5F mean age 18	11 IICPS 2seizure	fracture 3 nofracture 7 SS1	lesion 6, nolesion 6	BT 9, DSA 3, MRI 2, MRV 7	SSS 3 RTS 4 RSS 3 LSS 2 LTS 1 ICV 2	Supportive 6, AC 4	RC 3	GR 8 MR1 WR 2 ex 1

Abbreviations= M: male, F: female, SS: suture separation, IICPS: increased intracranial pressure symptoms, CT: computerize tomography, DSA: digital subtraction angiography, MRI: magnetic resonance imaging, MRV: magnetic resonance venography, SSS: superior sagittal sinus, LTS: left transverse sinus, LSS: Left sigmoid sinus, RTS: right transverse sinus, RSS: right sigmoid sinus, ICV: internal cerebral vein, AC: anticoagulation, RC: recanalization, GR: good recovery, MR: moderate recovery, WR: worse recovery, EX: exitus

in the clotting mechanism, alteration in the coagulation after head injury, disturbances in the blood flow or damage to the capillary endothelium may predispose and lead to thrombosis (28). The normal brain is rich in thromboplastin and it is generally postulated that cerebral contusion may result in the release of large quantities of this substance (8). Huber and colleagues reported from a postmortem study that microthrombi formation was seen in the contusion areas and there were also found in the contralateral hemisphere without contusion including postcapillary venules and spastic arteries (9). Similarly, intravascular coagulation and microvascular occlusion were exhibited in both experimental models and fatal traumatic brain injury (24). After the cascade of blood coagulation within the brain triggered by closed head injury, disrupted coagulation may result in the thrombosis of veins and/or sinuses.

The clinical presentation can be extremely varied

Table II: Summary of reported cases with cerebral sinus thrombosis due to head injury in the last two decades.

Author & year	Age, sex	Symptom	Skull fracture	Intracranial lesion	Diagnosis	Thrombosis	Treatment	Follow-up	Outcome
Taha et al. 1993	5(3m/2f) children	various	3 cases	3 cases contusion	BT, MRI	LSS, LTS	Supportive	4 RC 1 noRC	5 GR
Stiefel et al. 2000	8(5f/3m) children	IICPS	all cases	5 cases (EH or SH)	BT	1 RSS, 1 LTS, 3 RTS, 1 RSS+JV 1 RSS+RTS	-	6 RC 1 no RC 1 out	4 GR 4 MR
our cases	35 M	IICPS	-	-	BT, DSA	RTS, RSS	AC	RC	GR
	25 M	cranial nerve	+	EH	BT, MRI	RTS, RSS	AC	noRC	GR

Abbreviations= M: male, F: female, SC: suture ceparation, IICPS: increased intracranial pressure symptoms, EH: epidural hematoma, SH: subdural hemato1 ma CT: computerize tomography, DSA: digital substraction angiography, MRI: magnetic resonance imaging, SSS: superior sagittal sinus, LTS: left transverse sinus, LSS: Left sigmoid sinus, RTS: right transverse sinus, RSS: right sigmoid sinus, JV: jugular vein AC: anticoagulation, RC: recanalization, GR: good recovery, MR: moderate recovery.

in DST. Commonly, increased intracranial pressure signs such as nausea, vomiting and headache are present. The compensatory function of the venous collateral system is the main factor that determines the DST diagnosis and also affects prognosis (5). When compared to adults, incomplete growth of venous collaterals in children may facilitate the diagnosis of DST. These explain the relatively high frequency in children and the reported cases in the literature are consequently commonly in the pediatric age group.

The confirmation of the diagnosis relies on the demonstration of thrombus by neuroimaging techniques (1,18,20,23). For instance, CT scanning may show the thrombus itself when there is acute thrombosis or a high density in the affected sinus region and may present a specific delta sign, dense vein sign or cord sign, in addition to secondary signs such as congested cortical veins or cerebral edema (25). However, the most sensitive examination technique is MRI in combination with MRA. If the diagnosis is still uncertain after these investigations, DSA may be used and provides better details of the cerebral veins (3,18,23).

There is no consensus on the overall strategy concerning surgical, radiosurgical, or conservative therapy in DST (5,22,23,25,27,31). Firstly, if there is any compression to the dural sinus by depressed fracture or hematoma, it should be removed (28,30,31). If these pathologies were not diagnosed, the priority of treatment in the acute phase is to stabilize the patient's condition. Hence, supportive therapies are hydration, anticonvulsants, steroids, mannitol, acetazolamide and craniectomy for decreasing intracranial pressure. Additionally, the progress of known risk factors, such as deep venous thrombosis, infection and coagulopathy it must be prevented (22, 25).

Anticoagulation therapy is the first choice although it remains controversial in traumatic cases (6,22,25,27,31). LMWH, which has a longer life than unfractionated heparin, can also be administered since it has a more predictable response at standard doses and lower incidence of thrombocytopenia and hemorrhagic complications (22). However, since the patients have a hemorrhagic lesion related to head injury, anticoagulant therapy should be applied carefully with adequate monitoring for complications such as new bleeding. Urokinase and the recently used recombinant tissue-type plasminogen activator cause thrombolysis and can generally be administered via an endovascular route, but they are limited to case reports in head injury patients (2, 22).

The mortality ranges between 4.3 and 30%, and furthermore survival cases may have permanent neurological deficits, with a rate of about 12-25% in all DST patients (4,7,23,26). In the monitored posttraumatic DST cases (Table 1 and 2), better recovery has been observed in right-localized sinus thrombosis than the left-localized ones because generally the left hemisphere is dominant in the population. Moreover, the outcome of patients

would be worse if the superior sagittal sinus or deep venous system of the brain has been involved.

Studies following recanalization of venous sinuses have shown that it may be incomplete in some cases (7,26). Stolz et al have presented a prospective study, in which significant functional improvement appeared about 89% in 12-month follow-up period; on the other hand, the recanalization rate was determined to be about 60% (26). Published papers of DST cases following closed head injury showed a high recanalization rate (25,27) Furthermore, recurrence of DST is estimated to occur in approximately 12% and the patients also have an increased risk (14%) of deep venous thrombosis. Because of these reasons, the authors feel anticoagulation should be continued after the patients are discharged (7,23,26).

CONCLUSION

Since head trauma itself is a main DST factor, a head injury patient, particularly younger cases that suffer from headache or symptoms of increased intracranial pressure must be analyzed on suspicion of DST. In addition, additional diagnostic investigations should be performed in terms of DST in head trauma cases that have other risk factors show an unexpected clinical condition. Early diagnosis can contribute to preventing morbidity or even mortality.

REFERENCES

1. Brors D, Schafers M, Schick B, Dazert S, Draf W, Kahle G: Sigmoid and transverse sinus thrombosis after closed head injury presenting with unilateral hearing loss. Neuroradiology 43: 144-146, 2001
2. Chow K, Gobin YP, Saver J, Kidwell C, Dong P, Vinuela F: Endovascular treatment of dural sinus thrombosis with rheolytic thrombectomy and intra-arterial thrombolysis. Stroke 31: 1420-1425, 2000
3. Cruz BA, Santos LA, Damasceno RP Ribeiro LS, Xavier GA Jr, Nunes SV, Campos GB, Crivellari H: Dural sinus thrombosis in childhood systemic lupus erythematosus. J Rheumatol 28: 2140-2141, 2001
4. de Bruijn SF, de Haan RJ, Stam J: Clinical features and prognostic factors of cerebral venous sinus thrombosis in a prospective series of 59 patients for The Cerebral Venous Sinus Thrombosis Study Group. J Neurol Neurosurg Psychiatry 70: 105-108, 2001
5. Erdogan B, Caner H, Aydin MV, Yildirim T, Kahveci S, Sen O: Hemispheric cerebrovascular venous thrombosis due to closed head injury. Childs Nerv Syst 20: 239-242, 2004
6. Ferrera PC, Pauze DR, Chan L: Sagittal sinus thrombosis after closed head injury. Am J Emerg Med 16: 382-385, 1998
7. Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F: ISCVT Investigators Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis. Stroke 35: 664-670, 2004
8. Hesselbrock R, Sawaya R, Tomsick T, Wadhwa S: Superior sagittal sinus thrombosis after closed head injury. Neurosurgery 16: 825-828, 1985
9. Huber A, Dorn A, Witzmann A, Cervos-Navarro J: Microthrombi formation after severe head trauma. Int J Legal Med 106: 152-155, 1993
10. Kastenbauer S, Pfister HW: Pneumococcal meningitis in adults: Spectrum of complications and prognostic factors in a series of 87 cases. Brain 126: 1015-1025, 2003
11. Kuehnen J, Schwartz A, Neff W, Hennerici M: Cranial nerve syndrome in thrombosis of the transverse/sigmoid sinuses. Brain 121: 381–388, 1998
12. Meena AK, Jayalaxmi SS, Murthy JMK: Cortical sinovenous thrombosis following closed head injury. Neurol India 48: 195-196, 2000
13. Meltzer H, LoSasso B, Sobo EJ: Depressed occipital skull fracture with associated sagittal sinus occlusion. J Trauma 49: 981, 2000
14. Muthukumar N: Uncommon cause of sinus thrombosis following closed mild head injury in a child. Childs Nerv Syst 21: 86-88, 2005
15. Ochagavia AR, Boque MC, Torre C, Alonso S, Sirvent JJ: Dural venous sinus thrombosis due to cranial trauma. Lancet 347: 1564, 1996
16. Owler BK, Warrier S, Besser M: Traumatic unilateral deep cerebral venous infarction. J Clin Neurosci 11: 767-770, 2004
17. Owler BK, Besser M: Extradural hematoma causing venous sinus obstruction and pseudotumor cerebri syndrome. Childs Nerv Syst 21: 262-264, 2005
18. Renowden S: Cerebral venous sinus thrombosis. Eur Radiol 14: 215-226, 2004
19. Saad DF, Crawford GI, Wulkan ML: Cerebral venous thrombosis after closed head injury in a child.. J Trauma 58: 1066-1067, 2005
20. Satoh H, Kumano K, Ogami R, Nishi T, Onda J, Nishimura S, Kurisu K: Sigmois sinus thrombosis after mild closed head injury in an infant: Diagnosis by Magnetic resonance imaging in the acute phase. Neurol Med Chir 40: 361-365, 2000
21. Sousa J, O'Brien D, Bartlett R, Vaz J: Sigmoid sinus thrombosis in a child after closed head injury.. Br J Neurosurg 18: 187-188, 2004
22. Stam J: The treatment of cerebral venous sinus thrombosis. Adv Neurol 92: 233-240, 2003
23. Stam J: Thrombosis of the cerebral veins and sinuses. N Engl J Med 352: 1791-1798, 2005
24. Stein SC, Chen XH, Sinson GP, Smith DH: Intravascular coagulation: a major secondary insult in nonfatal traumatic brain injury. J Neurosurg 97: 1373-1377, 2002
25. Stiefel D, Eich G, Sacher P: Posttraumatic dural sinus thrombosis in children. Eur J Pediatr Surg 10: 41-44, 2000
26. Stolz E, Trittmacher S, Rahimi A Gerriets T, Rottger C, Siekmann R, Kaps M: Influence of recanalization on outcome in dural sinus thrombosis: a prospective study. Stroke 35: 544-547, 2004
27. Taha JM, Crone KR, Berger TS, Becket WW, Prenger EC: Sigmoid sinus thrombosis after closed head injury in children. Neurosurgery 32: 541-545, 1993

28. Tamimi A, Abu-Elrub M, Shudifat A, Saleh Q, Kharazi K, Tamimi I: Superior sagittal sinus thrombosis associated with raised intracranial pressure in closed head injury with depressed skull fracture. Pediatr Neurosurg 41: 237-240, 2005
29. Umit H, Asil T, Celik Y Tezel A, Dokmeci G, Tuncbilek N, Utku U, Soylu AR: Cerebral sinus thrombosis in patients with inflammatory bowel disease: a case report. World J Gastroenterol 11: 5404-5407, 2005
30. Uzan M, Ciplak N, Dashti SG, Bozkus H, Erdincler P, Akman C: Depressed skull fracture overlying superior sagittal sinus as a cause of benign intracranial hypertension: Case report. J Neurosurg 88: 598–600, 1998
31. Yokota H, Eguchi T, Nobayashi M, Nishioka T, Nishimura F, Nikaido Y: Persistent intracranial hypertension caused by superior sagittal sinus stenosis following depressed skull fracture. Case report and review of the literature. J Neurosurg 104: 849-852, 2006
32. Yuen HW, Gan BK, Seow WT, Tan HKK: Dural sinus thrombosis after minor head injury in a child. Ann Acad Med Singap 34: 639-641, 2005