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The Recognition of Traumatic Cerebral Venous Thrombosis

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One of therapeutic dilemmas of neurosurgeons is the recognition and possible management of traumatic cerebral arterial injury. Similar concerns can arise when there is trauma to the cerebrovenous system. On the one hand, its clinical presentations are nonspecific, and when the diagnosis is delayed, there is an increased risk of morbidity. Moreover, the initiation of anticoagulation is still a matter of debate because of the risk of worsening traumatic hemorrhage. According to Netteland et al., cerebral venous thrombosis is associated with four-times higher 30-day mortality (16% versus 4%) [1]. After traumatic brain injury, the *trans*-sinus fracture might explain the early genesis of thrombosis by damaging the endothelial lining of the sinus wall. The most affected areas are, in order, the following: transverse sinus, sagittal sinus, and sigmoid sinus [2].

Since when did clinicians recognize thrombosis of the cerebrovenous system? Despite the neurologist Gowers describing clinical features in the final years of the nineteenth century [3], cerebral venous thrombosis was an autopsy finding until the second half of the twentieth century and was not made durante vita. For centuries, thrombosis of the cerebral veins was known either as phlebitis or in puerperium. However, Gowers astutely recognized the clinical features in his book: "Cerebral symptoms are chiefly general—apathy, somnolence, and coma; vomiting and convulsions, usually general, but sometimes local; rigidity of the neck, and sometimes of the muscles of the back" [3]. He described progression as follows: "Unilateral symptoms are usually due to the extension of the thrombus into veins over one hemisphere, and then there may be unilateral convulsions, often beginning locally, and loss of power on one side" [3].

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The main symptom is the abrupt onset of seizures, usually beginning as focal epilepsy, but often within minutes, focal twitches spread on the same side or opposite side as in Jacksonian epilepsy. In the preceding century, purulent infections in general were rampant, and most cases were associated with infections, including sepsis or triggered by middle ear infection, mastoiditis, or cellulitis of the scalp. Gowers was also the first to point out trauma (and dehydration in a medically fragile patient) as a cause of cerebral venous thrombosis. In his 1898 two-volume *A Manual of Diseases of the Nervous System*, he often linked it to a linear skull fracture crossing a longitudinal or lateral sinus [3].

First Mentions

> *The vascular lesions of the brain met with in civil life, for instance, are most commonly due to arterial disease, to thrombosis, hemorrhage, or embolism, while primary affections of the cerebral veins are uncommon; on the other hand, in gunshot injuries of the head, especially when tangential or superficial, disturbance of the cerebral venous circulation by depression of fragments of the skull is frequent, owing to the superficial course of the cerebral veins and the fact that their thinner walls and the lower pressure of the blood that flows through them make them more liable to be blocked by pressure than the arteries* [3].

Holmes and Sargent would describe in great detail the clinical features of the longitudinal sinus syndrome:

> *The early onset of this rigidity is another striking point; we have seen it well marked within twenty-four hours of the infliction of the wound, and in some instances at least it las been noticed by the patient almost at once.... The rigid limbs generally*

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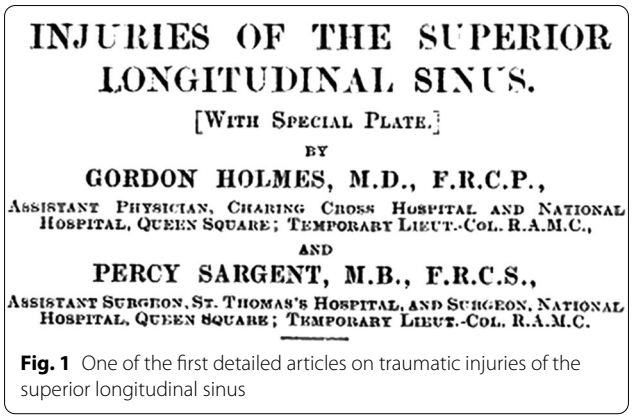

INJURIES OF THE SUPERIOR LONGITUDINAL SINUS.

[WITH SPECIAL PLATE.]

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Fig. 1 One of the first detailed articles on traumatic injuries of the superior longitudinal sinus

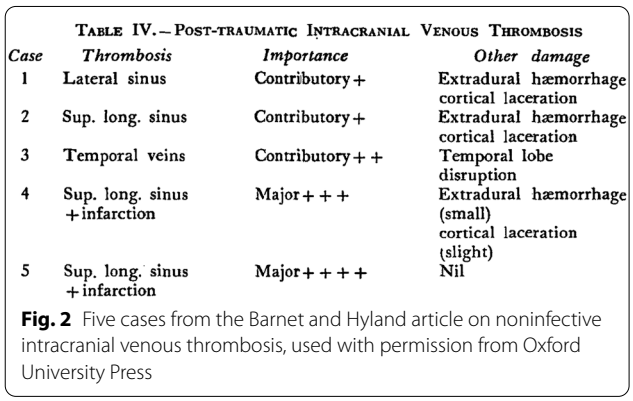

TABLE IV.—POST-TRAUMATIC INTRACRANIAL VENOUS THROMBOSIS

Case	*Thrombosis*	*Importance*	*Other damage*
1	Lateral sinus	Contributory +	Extradural hæmorrhage cortical laceration
2	Sup. long. sinus	Contributory +	Extradural hæmorrhage cortical laceration
3	Temporal veins	Contributory + +	Temporal lobe disruption
4	Sup. long. sinus + infarction	Major + + +	Extradural hæmorrhage (small) cortical laceration (slight)
5	Sup. long. sinus + infarction	Major + + + +	Nil

Fig. 2 Five cases from the Barnet and Hyland article on noninfective intracranial venous thrombosis, used with permission from Oxford University Press

assume very characteristic attitudes; when the arms are affected, they lie closely adducted to the sides and rotated inwards, with the elbows flexed and pronated, and in severe cases the wrists and fingers in moderate flexion. Even the trunk muscles may be involved, and then the abdominal wall is unnaturally rigid, and respiration is mainly thoracic; in a few cases, indeed, there has been slight difficulty in coughing, and phonation has been monotonous and toneless owing to the poor inspiratory intake [2] (Figs. 1, 2).

In assessing 37 fatal cases, Barnett and Hyland suggested that "venous thrombosis should be carefully looked for at autopsy in all these traumatic cases with 'pulped brain' because the gross appearance with hemorrhagic destruction extending deeply into the cerebral substance leads us to believe that venous thrombosis may be a contributing factor. Post-traumatic lesions of this type are usually attributed entirely to contre-coup or sheering forces and to the noxious effect of blood from ruptured vessels producing extensive local and generalized oedema" [4]. Their contribution was substantial to the understanding of trivial trauma to the head: "The main result of the trauma was the production of sagittal sinus and cortical vein thrombosis with accompanying haemorrhagic brain softening" [4]. They reported on a 50-year-old woman who sustained a fall 4 weeks before death, receiving a fractured humerus and a bump on the head that was initially considered trivial. Three weeks afterward, she first complained of headache, which was followed 3 days later by a sudden onset of hemiplegia; she then gradually slipped into a fatal coma. The necrotic and hemorrhagic lesion in the hemisphere contralateral to the hemiplegia was found at autopsy [4].

The first description of thrombosis of the cerebral veins and sinuses is attributed to the French physician Ribes in 1825. Among other cases of phlebitis, he observed thrombosis of the sagittal sinus and cerebral veins in a man who had had seizures and delirium [5].

In 1942, the British gynecologist Stansfield introduced the anticoagulant heparin [6]. Erik Jorpes initially clarified the chemical structure. Crafoord and Jorpes' second publication appeared in 1941, with 325 treated patients [7], and heparin entered the clinical armamentarium. Knowing how often puerperium thrombosis could be fatal, Jorpes decided to try intravenous heparin:

> *Dr. Ronald Jones saw the patient the next morning and agreed with the diagnosis. After discussion heparin therapy was tried, as it seemed to be a rational line of attack and it had previously given good results in cases of thrombophlebitis of the lower extremities in the gynecological ward. An intravenous drip of 5% glucose saline was started and was continued at the rate of a pint every four hours. Each pint contained 200 mg of heparin, equivalent to 16,000 Toronto units.... Heparin therapy was continued on the above lines for four days, by which time the patient had apparently fully recovered* [7].

Prior extensive experiences were based on treatment of pulmonary embolism and arterial occlusions [7–9]. Kendall combined treatment with heparin and dicumarol [10].

Modern Experiences

Clinical trials in the 1990s finally resolved the concern about using anticoagulants in most cases of cerebral venous system thrombosis. However, in traumatic brain injury, the need to balance risk of thrombus extension against risk of expansion of any concurrent traumatic intracranial hemorrhages is considered. Limited data support that risk of hemorrhage is higher with earlier initiation of anticoagulation treatment. Others suggest initiating low molecular weight heparin after an interval

scan documents relative stability of traumatic intracranial hemorrhages. In general, one should consider starting with a reduced dose and gradually increasing dose over the next days to subtherapeutic/therapeutic levels under surveillance of repeat computed tomography scans to monitor stability of traumatic intracranial hemorrhages [1, 11–13].

Traumatic cerebral venous thrombosis is not a medical diagnosis that frequently appears in the news, but in 2013, *The New York Times* reported that "Mrs. Clinton, 65, was admitted to New York-Presbyterian/Columbia hospital on Sunday after a scan discovered the blood clot" [14]. The scan was part of her follow-up care for a concussion she sustained more than 2 weeks earlier, when she fainted and fell, striking her head. According to the State Department, the fainting was caused by dehydration, brought on by a stomach virus. The concussion was diagnosed on December 13, although the fall had occurred earlier that week. (The reported diagnosis was right transverse sinus venous thrombosis.) She appeared later with prism glasses to correct transient diplopia. Astute neurologists were not surprised. In fact, as was noted in the very first articles, trauma to the cerebral venous system could be trivial but significant and, before anticoagulation, could lead to morbidity.

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Conflicts of interest

There are no conflicts of interest.

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References

1. Netteland DF, Mejlænder-Evjensvold M, Skaga NO, et al. Cerebral venous thrombosis in traumatic brain injury: a cause of secondary insults and added mortality. J Neurosurg. 2020;134(6):1912–20.
2. Holmes G, Sargent P. Injuries of the superior longitudinal sinus. Br Med J. 1915;2(2857):493–8.
3. Gowers WR. A manual of diseases of the nervous system. Berlin: P. Blakiston, Son & Company; 1898.
4. Barnett HJM, Hyland HH. Noninfective intracranial venous thrombosis. Brain. 1953;76(1):36–49.
5. Ribes F. Exposé succinct des recherches faites sur la phlébite. Revue Médicale Française et Étrangére et Journal de Clinique de l'Hôtel Dieu et de la Charité de Paris. 1825;3:5–41.
6. Stansfield FR. Puerperal cerebral thrombophlebitis treated by heparin. Br Med J. 1942;1(4239):436–8.
7. Crafoord C, Jorpes E. Heparin as a prophylactic against thrombosis. JAMA. 1941;116(26):2831–5.
8. McClure RD, Lam CR. Experiences in heparin administration. JAMA. 1940;114(21):2085–9.
9. Murray G. Heparin in surgical treatment of blood vessels. Arch Surg. 1940;40(2):307–25.
10. Kendall D. Thrombosis of intracranial veins. Brain. 1948;71(4):386–402.
11. Chtara K, Bradai S, Baccouche N, et al. Post-traumatic cerebral venous sinus thrombosis in an intensive care unit: a case series of ten patients. J Med Vasc. 2023;48(2):62–8.
12. Rivkin MA, Saraiya PV, Woodrow SI. Sinovenous thrombosis associated with skull fracture in the setting of blunt head trauma. Acta Neurochir (Wien). 2014;156(5):999–1007.
13. Wang WH, Lin JM, Luo F, et al. Early diagnosis and management of cerebral venous flow obstruction secondary to transsinus fracture after traumatic brain injury. J Clin Neurol. 2013;9(4):259–68.
14. Grady D, Landler M. Clinton out of hospital after treatment for clot. *The New York Times*. January 2, 2013. https://www.nytimes.com/2013/01/03/us/politics/hillary-clinton-is-discharged-from-hospital-after-blood-clot.html