CSF hydrodynamics in superior sagittal sinus thrombosis

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Abstract

Cerebrospinal fluid hydrodynamics were investigated with a constant pressure infusion method in patients with superior sagittal sinus thrombosis. Ten patients were studied with serial examinations up to 15 years after the onset of the disease. A total of 70 CSF hydrodynamic examinations were performed. A clear increase in intracranial pressure due to raised pressure in the major dural sinus was seen in all patients. A striking feature was the persistent intracranial pressure increase that declined only gradually. This had no obvious clinical impact. Change in CSF resorption facility played only a minor role in the intracranial pressure elevation. None of the patients developed hydrocephalus.

Aseptic cerebral venous thrombosis, mainly in the form of superior sagittal sinus thrombosis (SSST), is a fairly uncommon but important cause of raised intracranial pressure. Onset is often acute and dramatic. SSST is more common in younger adults and in women. There are many underlying causes but none is found in a quarter of cases. Most run a benign course in the acute phase. The mortality probably does not exceed 10%, and only about 20% of survivors are left with sequelae. Little is known about the long term outcome or risk of recurrence.¹ Recently there has been interest in unusual aspects of presentation, causes, or treatment of SSST.² Simple lumbar CSF pressure measurements have been reported from patients with sinus pathology³ but no serial investigations have been performed. CSF hydrodynamics have been studied by direct intrasinal and intraventricular pressure measurements in individual patients in special circumstances, such as during anaesthesia⁴ or neurosurgery,⁵ but fundamental pathophysiological changes in CSF hydrodynamics in SSST over time have not been reported. We present the first long term study on CSF hydrodynamics in patients with SSST.

Material and methods

Between 1975 and 1990, 13 patients admitted to this department had a selective cerebral angiography verified diagnosis of non-septic SSST. Ten of the patients (seven women and three men) were followed up over a mean of 5.8 years (range 2-15 years), and 70 CSF hydrodynamic investigations were performed

on them (17 of the investigations included only resting pressure recordings). Ethical approval and informed consent from the patients were obtained. The remaining three other patients were investigated on just one occasion, and the results of these studies are not reported. All patients had at least one CT scan, eight had two or more examinations. All patients had repeated ophthalmological examinations including visual fields (Goldman) and acuity. Fundus photographs were taken in most patients. No patient died. Neuropsychological testing included the Wisconsin card sorting test,⁶ the Halstead-Reitan neuropsychological test battery,⁷ the Claeson-Dahl's learning and retention test,⁸ the visual retention test of Benton,⁹ and a finger tapping test for motor speed.¹⁰ The first examination was performed from nine to 60 months and the last investigation from 30 to 98 months after onset of symptoms.

CSF hydrodynamic investigation was performed according to the method described by Ekstedt.¹¹ After 12 hours' bed rest, two needles were inserted in the L3-L4 interspace. CSF (2 ml) was aspirated to check for free CSF passage from each needle. The patient was then examined lying supine with the zero pressure reference level at the cranial sagittal centre. Drainage of CSF and infusion of artificial CSF was made to and from a continuously weighed bottle. The pressure in the bottle was regulated by means of an electronic control system acting on the fluid in the bottle by means of the air pressure from a pump. CSF resting pressure (P_{cl}) was determined when the resting recording had been stable for at least 10 min, which usually required 30-60 min recording. The conductance of the CSF outflow pathways (G_{op}) was determined by applying multiple pressure levels to the CSF space while recording the resulting inflow of artificial CSF into the patient. Thus within a few minutes a stable flow at a stable pressure was obtained. Usually, three different pressure/flow values were aimed at. The volume accounting method was used to calculate the pressure/flow relation. The slope for the pressure/flow values is equal to the CSF outflow conductance. The CSF formation rate (q_f) was determined by lowering the CSF pressure to a value of about 0 kPa for a sufficient period to produce a stable pressure and outflow into the bottle. Finally, the pressure difference across the CSF outflow pathways (P_{dop}) and the sagittal sinus

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Table 1 Normal values for CSF hydrodynamic variables (58 patients (for P_{ss} 100) and 15 to 83 years) in supine horizontal position. Reference level: cranial sagittal centre; constant pressure infusion method with artificial CSF. No sex or age differences noted

	Mean	SD	90% Confidence intervals
Lumbar resting pressure (P_{cl})	1.4 kPa	0.2	1.2 to 1.8
Conductance of CSF outflow pathways (G_{op})	18.0 mm ³ kPa ⁻¹ s ⁻¹	4.0	12.0 to 26.0
CSF formation (qf)	6.7 mm ³ s ⁻¹	1.4	4.5 to 9.5
Pressure difference across outflow pathways (P_{dop})	0.4 kPa	0.1	0.3 to 0.6
Sagittal sinus pressure (P_{ss})	1.0 kPa	0.2	0.7 to 1.4

pressure (P_{ss}) were calculated according to the formula¹²:

$$P_{dop} = qf/G_{op} \text{ and } P_{ss} = P_{cl} - P_{dop}$$

The calculated values of P_{ss} express the mean value of the pressures in the major dural sinuses. The normal values for CSF hydrodynamic variables (table 1) were obtained from patients in whom medical history, as well as medical and neurological investigation, indicated no organic neurological or circulatory disorder. This control group included 58 patients.¹³

SI units have been used, and the following conversion factors may be useful: pressure (1 kPa = 102 mm H₂O = 7.5 mm Hg); conductance (1 mm³ kPa⁻¹sec⁻¹ = 6×10^{-3} ml (cm H₂O)⁻¹ = 8×10^{-3} (mm Hg)⁻¹ min); and resistance (the inverse of conductance).

Results

The clinical findings are summarised in table 2. Six patients (patients 1, 3, 4, 5, 6, and 8) had a well defined syndrome with symptoms related to intracranial hypertension (headache, papilloedema, impaired consciousness), with or without generalised epileptic seizures. The others had focal symptoms, with or without signs of intracranial hypertension. Two patients were left with sequelae (table 3). Only one of

Table 3 Outcome of 10 patients with superior sagittal sinus thrombosis

Patient No	Recovery	Time for resolution of papilloedema (months)	Sequelae
1	+	12	
2	+	5	
3	+	9	
4	+	27	
5	+	84	
6	+	6	
7	—		Discrete motor dysfunction, Partial complex seizures
8	+	11	
9	+	12	
10	—		Partial complex seizures

the four patients who had generalised epileptic seizures in the acute phase had later recurrence (patient 7). The others have stopped anti-epileptic drugs.

Neuroradiology Selective carotid angiography identified seven patients with an isolated SSST (no involvement of other sinuses). The occluded sinus could not be visualised in five patients, indicating complete thrombotic occlusion (table 4). The three patients investigated at least one month after onset all had a partial, isolated thrombosis. Each patient's CSF hydrodynamic variables are shown in figure 1. All patients had raised sagittal sinus (P_{ss}) and intracranial pressure (P_{cl}) at their first investigation. The pressure difference across the CSF outflow pathways (P_{dop}) was preserved or slightly increased. Six patients were studied within two months after onset. The highest pressure was recorded at either the first or second measurement. Pressures gradually declined (P_{ss} and P_{cl}) over time in all patients but returned to normal in only two patients (case 7 and 10) after eight and 15 years, respectively. The presence of total or partial thrombotic occlusion on initial angiography did not appear to correlate with the long term development of pressure or to clinical outcome. Conductance was normal or slightly to moderately decreased. A more pronounced

Table 2 Clinical characteristics of 10 patients with superior sagittal sinus thrombosis. Mode of onset indicated as time interval elapsed between appearance of first symptom and date of entry to hospital

Patient No	Sex	Age at onset (years)	Follow up time (months)	CSF hydrodynamic investigations (No)	Duration between onset and first investigation (months)	Mode of presentation	Neurological features	Cause
1	M	20	52	4	2	Acute	Somnolens, Ha, Pe, GES	Hereditary AT III-deficiency
2	M	23	136	9	0.1	Subacute	Partial motor seizures, Hp, Ha, Pe	Colitis ulcerosa
3	F	22	70	9	1	Subacute	Ha, Pe	Idiopathic
4	F	36	64	6	9	Chronic	Ha, Pe	Idiopathic
5	F	53	184	12	24	Chronic	Ha, Pe	Head injury
6	F	23	25	2	5	Acute	Somnolens, Ha, Pe, GES	Post partum
7	F	35	99	8	2	Acute	Coma, Hp, Pe, GES	Post partum, hereditary dysfibrinogenemia
8	F	21	122	12	1	Subacute	Ha, Pe	Oral contraceptives
9	M	18	60	6	2	Acute	Somnolens, Hp, Pe, GES	Idiopathic
10	F	40	180	2	108	Chronic	Partial complex seizures, Ha	Sarcoidosis

GES = generalised epileptic seizures; Ha = headache; Pe = papilloedema; Hp = hemiparesis.

Table 4 Angiographic findings in 10 patients with superior sagittal sinus thrombosis

Patient No	SSS		Other sinus involvement		Cerebral veins	Collaterals
	Total	Partial	Yes	No		
1	+		LS + SS	+		Transcerebral
2		+				Cortical
3		+		+		
4		+		+		Cortical
5		+	SS	+		Meningeal
6	+					Cortical
7	+			+	+	Transcerebral
8	+		LS		Cortical	
9	+			+	+	Transcerebral + extracranial
10		+		+	Cortical	Transcerebral + cortical

SSS = superior sagittal sinus; SS = straight sinus; LS = lateral sinus.

decrease was seen in patients with a complete sagittal sinus thrombotic occlusion. No consistent changes in conductance were seen although in some cases an initial decreased conductance was later normalised. The CSF formation rate (qf), which was normal in all patients, did not vary over time. No optic nerve damage could be detected except for slightly enlarged blind spots three patients (1, 2, and 4). Hydrocephalus was not found in any of the patients on repeated CT scans (table 5). When seen, signs of cerebral oedema resolved in all cases with time. No patient had a recurrence of thrombosis.

Neuropsychological assessment Four patients (3, 4, 7, and 8) were evaluated at least twice in the chronic stage with standardised neuropsychological testing; one patient had four examinations. No overt clinical symptoms related to cognitive dysfunction were found in these four. Patient 2 had a normal test result five years after onset. A slight decrease in fine motor speed in the right hand and in the ability for perceptual analysis was noted in patient 4, 18 months after the onset of symptoms. In patient 7 a discrete impairment was seen at the first investigation and a minor deterioration involving inductive logistics and learning of audioverbal information had occurred at final testing seven years later. In patient 8 a minor decline in tests of recent memory with sub-clinical dysfunction of higher adaptive capability was detected six years after baseline testing.

Table 5 Findings on CT scans in first (early) and last (late) investigation. Time in brackets marks interval from onset of symptoms to scan

Patient No	Early	Late
1	Empty delta sign (1 day)	Normal (3 weeks)
2	Diffuse oedema, slit ventricles (2 days)	Normal (12 months)
3	Normal (1 month)	Normal (23 months)
4	Normal (9 months)	
5	Normal (36 months)	Normal (140 months)
6	Empty delta sign + central oedema (1 week)	Normal (2 months)
7	Two frontal left sided haemorrhagic infarctions (3 days), small ventricles	Minor left sided substance defect (21 months)
8	Normal (1 month)	Normal (46 months)
9	Haemorrhagic left sided frontal infarction (3 days)	Left sided hypodensity (15 months)
10		Normal (108 months)

Discussion

The natural history of SSST has not been well documented.¹ We have studied CSF hydrodynamics over a mean of 5.8 years from the acute or subacute to the chronic phase in 10 patients with SSST, and our patients may thus reflect the natural course of the condition. None of our patients underwent shunting or other neurosurgical procedures, and only two patients were optimally treated with anti-coagulants (heparin in the acute phase) and some had short term medical treatment aimed at reducing brain oedema in the acute phase (including steroids, mannitol, or diuretics). The main limitation of this study was that the patients studied were highly selected. Most were suspected of having intracranial hypertension and were referred to our clinic, which offers the facilities for CSF hydrodynamic monitoring. Thus neither acute cases with a fulminating course nor the benign forms which recover rapidly and completely are likely to have been referred. The clinical characteristics of our patients do not, however, essentially differ from contemporary clinical materials.¹

There are three intracranial components or compartments—brain tissue, blood, and CSF—that if pathologically altered could cause intracranial hypertension. Our view of the predictable hydrodynamic consequences of a superior sagittal sinus thrombosis is illustrated in figure 2. In our patients, the main hydrodynamic features were increase of P_{ss} and P_{ci} . Animal studies have shown a direct relation between the increase in sagittal sinus pressure and CSF pressure,¹⁴ and CSF pressure will increase by an amount equal to the increase of P_{ss} .¹⁵ When sagittal sinus pressure is raised, engorgement of cortical veins is likely to cause an increase in total intracranial blood volume consequent on the impaired venous flow into the sagittal sinus. This excess intracranial volume may further increase intracranial pressure. The increase of intercerebral venous blood pressure elevates hydrostatic capillary pressure, thereby producing an increase in net capillary filtration and the possibility of a progressive cerebral oedema. Ventricular compression may be seen on CT scan in up to half of patients in the acute phase of SSST.¹⁶ The increase in brain volume may in itself lead to an additional increase in P_{ss} . Direct compression of the sinuses and lateral lacunae may follow

Figure 1 Course of various pressure values ($P_{c,p}$, $P_{i,p}$, P_{dop}) and conductance (G_{op}) plotted against time. Dotted line in pressure value curves marks 90% confidence limit for $P_{c,i}$ upper normal value. Low conductance values are defined as values falling within shaded area on G_{op} curves.

Figure 2 Overview of mechanisms and development of CSF hydrodynamic events after SSST, according to findings in present study.

elimination of the parasagittal subarachnoid space, further compromising venous outflow.

If further pressure elevation occurs or is not relieved, coma and death may ensue. In most patients, however, the deterioration is transient and spontaneous improvement is the rule. This improvement is probably more a reflection of the adequacy of collateral channels than to restoration of blood flow through a recanalised occluded sinus lumen.¹⁷ In case 9 a first angiogram three days after onset of symptoms showed a complete thrombosis but no collaterals, and he was in a critical phase for 10 days. Two weeks later a second angiogram clearly showed collaterals but the thrombotic occlusion was completely unchanged. The persistently high P_{ss} in our patients may well indicate that thrombotic occlusion is commonly permanent. It should be emphasised that P_{ss} represents an indirect calculated mean pressure of the major dural sinuses and is presumably raised in the case of obstructed venous outflow due to thrombosis of the superior sagittal sinus or its bridging veins, or both. In patients who survived for a considerable period after SSST but were examined postmortem the lumen of the superior sagittal sinus has been reported to be almost com-

pletely occluded by fibrous tissue with only small sinusoidal channels present which would not have permitted adequate drainage of venous blood from the brain.¹⁷ In individual patients the amount of blood flowing out through collaterals or a partially patent sinus probably varies. Collaterals were present in all our patients who had a complete thrombosis. Those patients with no collaterals on angiography would have to rely on sufficient outflow via the sinus (patient 3). Boussier *et al* reported that all patients with complete SSST in a group with cerebral vein thrombosis had anastomoses on angiography whereas two patients with partial thrombosis did not.¹ In our series one patient (patient 10) whose pressure levels returned to normal had a repeat angiogram five years after onset which showed a more extensive collateral system that might explain the decrease in pressure levels.

Treatment in the acute phase should aim to induce thrombolysis and prevent further thrombosis. Effective thrombolysis in the early stage might prevent the persistent intracranial pressure elevation that we saw in our patients. Several studies have reported the use of fibrinolytic treatment with varying success.^{18,19} Early heparin treatment may improve outcome

by preventing thrombus extension²⁰ which further compromises the outflow of venous blood, additionally increasing intracranial pressure. Total sagittal sinus occlusion is said to be often fatal.²¹ In our study, no obvious difference in the clinical course and hydrodynamic variables was detected between cases with partial and complete thrombosis, probably reflecting the limited size and selectivity of our material.

An occlusion of the superior sagittal sinus may, to some degree, obstruct resorption of CSF by the arachnoid villi (a lower conductance), leading to increased CSF pressure. In our patients, conductance was normal or only slightly to moderately decreased (moderate reduction occurring in patients with a complete thrombosis). Conductance, when failing to improve during follow up, may be a consequence of a permanent disturbance of arachnoid villus function. As the CSF formation rate did not vary P_{dop} will be preserved or increased when conductance is lowered according to the classic relation $P_{\text{dop}} = q_{\text{fr}}/G_{\text{op}}$. The maintenance of the cerebrospinal-sagittal sinus gradient encourages CSF resorption. Alternative CSF pathways through venous collateral vessels may also play a part in CSF drainage. Therefore, a fairly small disturbance occurs in the CSF absorption process with only a minor contribution to intracranial pressure elevation. Under these circumstances treatment by serial lumbar punctures or various shunting procedures can hardly produce more than a marginal pressure lowering effect.

Only a few studies have described the hydrodynamic effects of sagittal sinus obstruction. One of these reported a reversed or nullified P_{dop} but conductance was normal or slightly reduced.⁴ Simultaneous recording of sagittal sinus pressure and intracranial pressure in humans and experimental animals, however, has shown that a positive CSF-sagittal sinus pressure gradient is almost always maintained despite gross elevations in both the intracranial and sinus pressures.^{5 13 22 23}

In animal studies a chronically increased intracranial venous pressure has produced a communicating hydrocephalus.²⁴ Others, however, have failed to produce hydrocephalus after experimental venous outflow obstruction.^{25 26} Clinically, ventricular enlargement rarely occurs in SSST.²⁷ Serial enlargement of the ventricles over the course of one to two weeks has been shown as cerebral oedema resolved.²⁸ In another study, six patients with sagittal sinus pathology underwent CT scanning once, a few days to a year after onset. No ventricular enlargement was found.³ In our patients CT scans were performed over a much longer time span; no patient developed communicating hydrocephalus, which supports the results of their hydrodynamic profile. In some cases CSF outflow conductance was only moderately decreased compared to other conditions characterised by hydrocephalus.^{29 30} Furthermore, the preservation of the pressure gradient between the subarachnoid space and the venous side of the dural sinuses might be the main factor preventing hydrocephalus. A

transcerebral pressure difference between the ventricular system and the subarachnoid cortical space as a cause of ventricular enlargement should not be present in those patients in whom the freely patent CSF spaces prevent the development of any such pressure gradients.

In the event of acute intracranial pressure elevation, alterations in consciousness disappear as cerebral oedema resolves. Survivors generally recover completely. Hydrodynamic abnormalities may still be seen many years after SSST but when oedema has resolved a high intracranial pressure does not seem to substantially impair cerebral function. Neuropsychological testing, however, showed some patients to have discrete signs of cognitive dysfunction. No serious visual complications were found in our patients despite their persistently elevated intracranial pressure. The limited ability to compensate for a possible additional increase in intracranial pressure may mean that patients with SSST who develop a stroke or who receive a head injury have a higher risk.

Our results show that after superior sagittal sinus thrombosis, hydrodynamic abnormalities, particularly persistently raised CSF pressure mainly due to raised P_{ss} , remain for many years. The change in conductance or CSF resorption facility plays only a minor part in the increase in intracranial pressure. Despite the persistently raised CSF pressure hydrocephalus does not develop and the long term clinical course is generally benign.

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