Cerebral venous sinus thrombosis

Cerebral venous sinus thrombosis is an under-recognized clinical condition, responsible for 0.5–1% of all strokes (Saposnik et al, 2011). Its clinical presentation is diverse, ranging from headaches to reduced conscious levels and focal neurological deficits (Coutinho, 2015). The diagnosis is often delayed, with a median interval of 7 days reported between the development of symptoms and the confirmation of diagnosis (Ferro et al, 2009). Several short- and long-term complications can develop, such as seizures and visual loss (Saposnik et al, 2011). The management of this condition is complicated, requiring a multidisciplinary approach. This article raises awareness of cerebral venous sinus thrombosis and discusses its aetiology, risk factors, management and prognosis.

What is cerebral venous sinus thrombosis?

Cerebral venous sinus thrombosis affects about 13 adults per million annually and occurs in all age groups (Coutinho, 2015). It comprises two separate entities: the thrombotic occlusion of cortical veins and of the dural sinuses. Cortical veins drain into the dural sinuses, and their occlusion leads to oedema, haemorrhage and infarction. Dural sinuses are important in the absorption and drainage of CSF, and their occlusion leads to impaired absorption of CSF and raised intracranial pressure (Coutinho, 2015). In the majority of patients, dural sinus and cortical vein thrombosis occur together. The anatomy of the cerebral venous system is illustrated in *Figure 1*.

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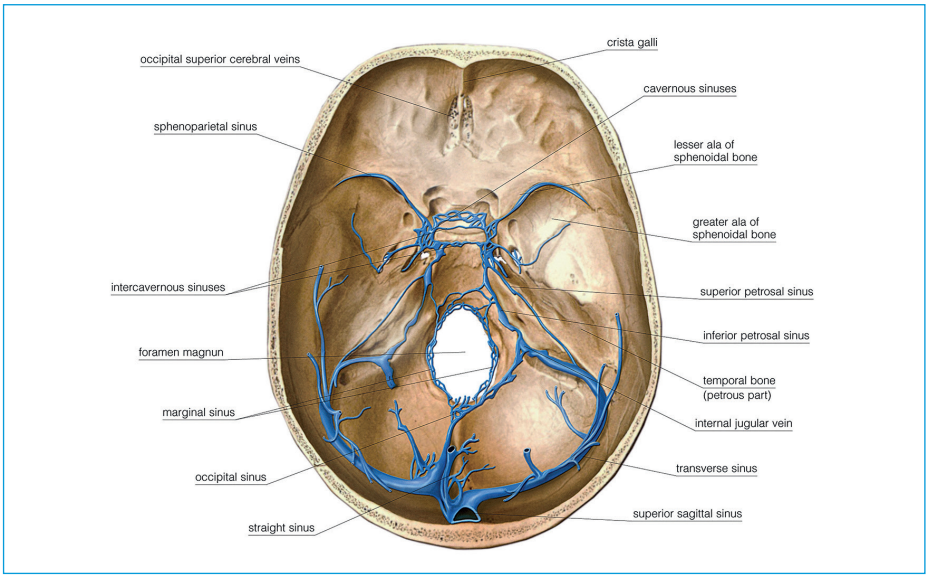

Figure 1. Anatomy of the cerebral venous system.

Clinical presentation

The presenting clinical symptoms can vary but ultimately arise as a result of the main pathological mechanisms: raised intracranial pressure, venous oedema and infarction. Headache is a key feature occuring in up to 90% of patients. While usually of gradual onset, it can present as a thunderclap onset headache (Stam, 2005). This may be the only feature. Seizures occur in 40% of patients, and can be focal or generalized, leading to status epilepticus in some cases (Stam, 2005).

Other presenting symptoms can include sensory or motor focal neurological deficits, visual disturbances and altered speech (Kumral et al, 2012). A subpopulation of patients can present with isolated intracranial hypertension. They may have single cranial nerve deficits, such as an abducens nerve palsy. Papilloedema can occur and be progressive, leading to visual loss if unrecognized (Ferro et al, 2009). Patients can also present with an altered conscious level (e.g. coma) (Kumral et al, 2012). A systemic cause of cerebral venous sinus thrombosis (e.g. middle ear infection) can lead to systemic symptoms such as fever and ear discharge. Isolated subarachnoid haemorrhage is a rare presentation of cerebral venous sinus thrombosis, and should be considered especially with a convexity subarachnoid haemorrhage not involving the basilar cisterns (Kim et al, 2016).

Cerebral venous sinus thrombosis is an important differential diagnosis in a young person, likely female, presenting with a headache associated with seizures, focal neurology or signs of raised intracranial pressure. The presenting neurological symptoms can be unusual because of the possibility of bilateral parenchymal involvement. This diagnosis requires a high index of suspicion.

Risk factors for cerebral venous sinus thrombosis

In 12.5% of all cases of cerebral venous sinus thrombosis studied in the ISCVT (International study on cerebral vein and dural sinus thrombosis) trial (Ferro et al, 2004), no precipitating risk factors could be identified. However, for the remainder, multiple inherited and acquired risk factors have been described (discussed below).

Inherited and acquired thrombophilia

Inherited prothrombotic states (e.g. factor V Leiden, protein C deficiency) contribute towards development of cerebral venous sinus thrombosis. In the ISCVT study, an

underlying inherited thrombophilia was identified in 22.4% of patients. Acquired thrombophilia (e.g. nephrotic syndrome, antiphospholipid syndrome) contributed to 15.7% of the remainder of the cases (*Table 1*).

Acquired risk factors

There are multiple acquired risk factors (*Table 1*), which are further described in the following sections.

Gender and associated risk factors

Cerebral venous sinus thrombosis is more common in women, in the young and middle-aged adults (Coutinho, 2015). In the ISCVT study (Ferro et al, 2004), 74.5% of patients were female.

The use of oral contraceptives is associated with an increased risk of cerebral venous sinus thrombosis (odds ratio 5.59 compared to the control population) (Dentali et al, 2006).

Cerebral venous sinus thrombosis commonly presents in pregnancy and the puerperium (10–20 per 100 000 deliveries in western Europe and north America) (Lockhart and Baysinger, 2007). It can mimic a post-dural puncture headache (Lockhart and Baysinger, 2007). The treatment in pregnancy is low molecular weight heparin as vitamin K antagonists are contraindicated.

Cerebral venous sinus thrombosis in neonates and children

The incidence of cerebral venous sinus thrombosis is estimated to be 0.67 per 100 000 per year in children (Bousser and Ferro, 2007). It occurs most commonly in neonates; 75% of neonates with cerebral venous sinus thrombosis have a concurrent underlying illness (e.g. dehydration, sepsis) (Bousser and Ferro, 2007). Maternal comorbidities (e.g. diabetes, pre-eclampsia) increase the neonatal risk of cerebral venous sinus thrombosis (Bousser and Ferro, 2007). The presenting symptoms can be seizures, respiratory distress and apnoea (Bousser and Ferro, 2007). There are no well-controlled trials studying treatment options. As well as treating concomitant illnesses, anticoagulation can be considered.

Others

Infections of the CNS, head and neck are associated with increased risk of venous sinus thrombosis (10.3% in the ISCVT study; Ferro et al, 2004) as are systemic infections (McBane et al, 2010). In the ISCVT study, 7.4% of all cases were associated with underlying malignancy (Ferro et al, 2004) with potential causative mechanisms including direct compression by tumours, sinus invasion and associated hypercoagulability syndromes. Cerebral venous sinus thrombosis is associated with a variety of inflammatory diseases including Behçet's disease, antiphospholipid antibody syndrome, systemic lupus erythematosus and the inflammatory bowel diseases (Ferro et al, 2004).

General risk factors identified in the ISCVT study include recent surgery (2.7%), dehydration (1.9%) and mechanical precipitants (4.5%) such as lumbar puncture (Ferro et al, 2004).

Known risk factors are summarized in *Table 1*.

Investigations and diagnosis

When making a diagnosis of cerebral venous sinus thrombosis it is important to identify the relevant risk factors.

Clinical examination

A full neurological examination including mental state examination and fundoscopy may provide helpful indicators for diagnosis. If detected, papilloedema urgently requires further investigation. It is important to complete a general examination looking for signs of infection and systemic illness.

Blood tests

Blood tests required include full blood count, renal and liver function, bone profile, clotting studies (prothrombin time and activated partial thromboplastin time) and inflammatory markers (Coutinho, 2015). If an underlying condition is suspected, appropriate diagnostic tests must be performed. Thrombophilia testing can be performed but is unlikely to guide the acute management of cerebral venous sinus thrombosis.

The D-dimer test is well established in the diagnosis of pulmonary embolism and deep vein thrombosis. In cerebral venous sinus thrombosis, there is no clear scoring system to define high and low risk cases. In a study which defined low risk patients as those with a normal neurological examination and computed tomography, the D-dimer test was useful in excluding cerebral venous sinus thrombosis (100% negative predictive value). However, patients who are deemed high risk should be imaged, regardless of D-dimer results (Alons et al, 2015).

Table 1. Risk factors for cerebral venous sinus thrombosis

Inherited risk factors	Factor V Leiden mutation
	Prothrombin G20210A factor 11 mutation
	Hyperhomocysteinaemia
	Protein C and S deficiency
Acquired risk factors	Systemic disease: malignancy, systemic lupus erythematosus, inflammatory bowel disease, Behçet's disease
	Intracerebral causes: internal jugular vein cannulation, head trauma, intracranial hypotension, neurosurgery
	Haematological diseases: polycythaemia rubra vera, anaemia
	Medications: oral contraceptive pill, steroids, hormone replacement therapy
	Infections: CNS, head and neck infections
	Pregnancy, puerperium

Imaging

Venous sinus imaging should be considered in patients presenting with:

- Headache and risk factors for thrombosis
- Features of raised intracranial pressure
- Focal neurological features with acute or sub-acute headache
- Papilloedema and risk factors for thrombosis
- Altered mental state associated with suspicion of bilateral thalamic infarction.

If the following features are present on initial imaging, consider venous sinus imaging:

- Bilateral infarction or haemorrhage
- Infarction crossing arterial boundaries.

The main imaging modalities used to diagnose cerebral venous sinus thrombosis are magnetic resonance imaging and computed tomography. Non-contrast computed tomography is often used as the preferred imaging modality in the acute setting, given its accessibility (*Figure 2*). Signs noted include the cord sign or dense vein sign, which represent a homogenous, hyperattenuated clot seen in either the sinuses or veins. The sensitivity of the cord sign is between 25% and 65% and the dense vein sign has a specificity of >99% if seen (Caprio and Bernstein, 2012). False positives occur

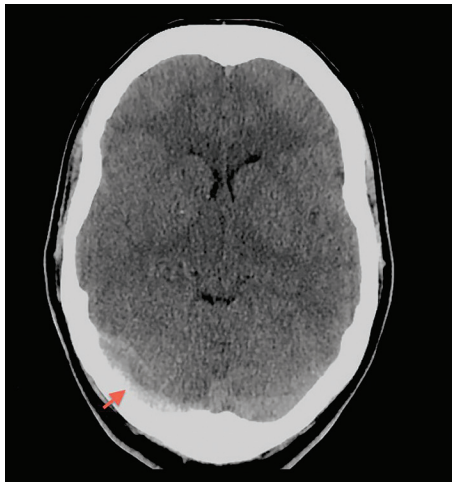

Figure 2. Non-contrast computed tomography. Cord sign representing hyperattenuation within a dural venous sinus, and marked by red arrow.

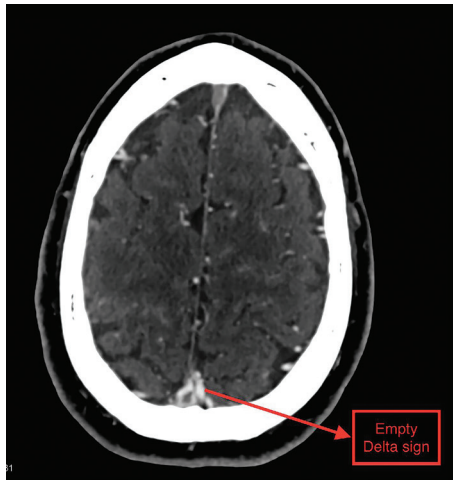

Figure 3. Contrast-enhanced computed tomography with empty delta sign labelled.

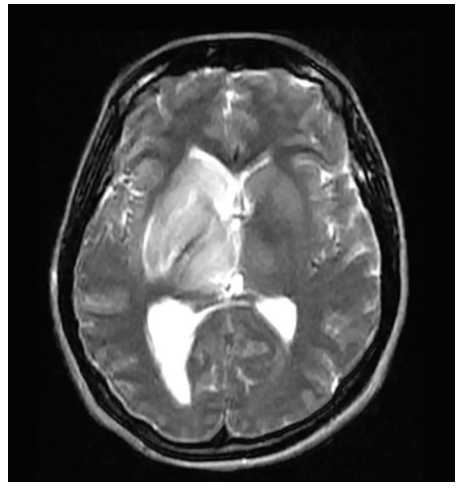

Figure 4. Axial T2 image with venous infarction secondary to straight sinus thrombosis.

from interference from surrounding bony tissue. False negatives occur if the imaging is delayed, and the clot appears isodense.

Computed tomography with contrast can show an empty delta sign – an area of low attenuation surrounded by high attenuation contrast (*Figure 3*).

Computed tomography angiography is an accessible and accurate imaging technique. It is relatively fast, permits the use of contrast to image both the arterial and venous systems, and has a sensitivity and specificity of 100% (Caprio and Bernstein, 2012). Cerebral venous sinus thrombosis appears as a filling defect in the vein in question. The main drawback is the exposure to ionizing radiation, which is especially relevant in children and pregnant women (Leach et al, 2006).

Magnetic resonance imaging is also commonly used to diagnose cerebral venous sinus thrombosis. However, the appearance of the thrombus depends on the chosen sequence and the timing of the imaging relative to clot formation. T2-weighted imaging is valuable especially in the acute phase of the disease, where the clot may not be well imaged by T1-weighted imaging (*Figure 4*). The thrombus is seen as a tubular structure which is said to 'bloom'. This is seen in up to 90% of all cases of cerebral venous sinus thrombosis (Caprio and Bernstein, 2012). Diffusion weighted imaging shows a diffusion defect with a hyperintense signal. However, the sensitivity has been described as only 4–40% (Caprio and Bernstein, 2012).

A common modality to image cerebral venous sinus thrombosis is the venous time of flight magnetic resonance angiogram. Flow gaps occurring in the non-dominant transverse sinus, transverse sinus hypoplasia can be misinterpreted as clot formation.

Magnetic resonance imaging is also better able to detect associated parenchymal changes than computed tomography (Leach et al, 2006).

Invasive diagnostic tests

Invasive cerebral angiography and venography has been largely superseded by magnetic resonance venography and computed tomography venography. However, they are still occasionally used when the diagnosis is unclear or another invasive procedure is being planned (Stam, 2005).

Other tests

Lumbar puncture is not routinely used in the diagnosis of cerebral venous sinus thrombosis. However, if infection is suspected, it is recommended. The lumbar puncture should be performed after imaging is obtained, and the opening pressures must be measured. The main concerns for not performing a lumbar puncture are the fear of transtentorial or transforaminal herniation or coning. Lumbar punctures are contraindicated if there are computed tomography signs of lateral shift of midline structures, loss of the suprachiasmic and basilar cisterns, loss of or shift of the fourth ventricle and the loss of the superior cerebellar plate and quadrigeminal cisterns (Joffe, 2007). These features are all signs of raised intracranial pressure, but their absence does not exclude it. If the intracranial pressure is raised significantly (e.g. >30 cmH_2O), CSF can be removed as a temporizing measure.

Figure 5 outlines the investigation and management of cerebral venous sinus thrombosis.

Management

Anticoagulation

The main treatment for thrombotic disease is anticoagulation (Stam, 2005), to recanalize the occluded vessel, and prevent thrombus propagation and recurrence. The main therapeutic dilemma is the risk of haemorrhagic transformation associated with anticoagulation (de Bruijn and Stam, 1999). Clinical guidelines support the use of heparin anticoagulation, and this is widely used as the initial treatment of cerebral venous sinus thrombosis. Intracranial haemorrhage with cerebral venous sinus thrombosis is not an automatic contraindication for anticoagulation, as patients treated with heparin who also had an underlying haemorrhage did not have worsening symptomatic intracranial bleeding post treatment (de Bruijn and Stam, 1999). In practice, it is often used even in the presence of haemorrhage on imaging if the diagnosis of cerebral venous sinus thrombosis is felt to be secure.

Low molecular weight heparin is recommended for the initial treatment of cerebral venous sinus thrombosis. It is at least as effective as unfractionated heparin, with fewer side effects (Coutinho et al, 2010). Patients are then converted on oral anticoagulation.

There are no randomized control trials of the duration of treatment specific to cerebral venous sinus thrombosis. Thus, many treatment recommendations are extrapolated from the treatment of systemic venous thromboembolism, are based on

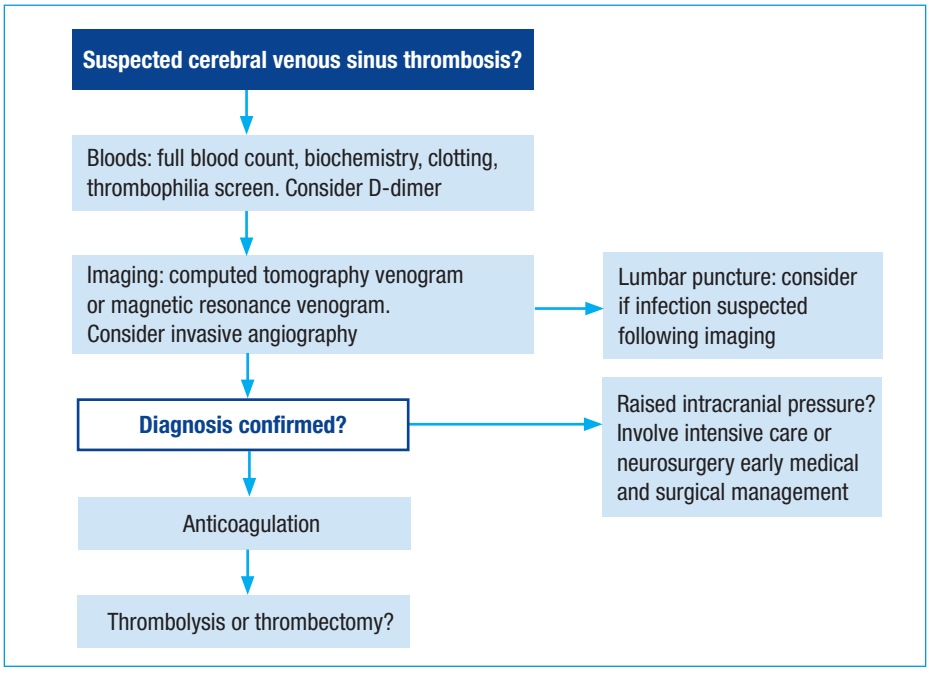

Figure 5. Investigations and management flowchart.

expert opinion and are determined by whether it is provoked by risk factors or unprovoked. Risk factors can be permanent or transient. In cerebral venous sinus thrombosis, if a transient risk factor (e.g. dehydration) is thought to co-exist, anticoagulation is continued for 3 months. If the cause is malignancy, anticoagulation is continued indefinitely or up till the point of cure. Cerebral venous sinus thrombosis in pregnancy requires anticoagulation throughout the pregnancy and into 6 weeks of the puerperium. Mild thrombophilia (e.g. heterozygous factor V Leiden mutation) or oral contraceptive pill use requires 3–6 months of anticoagulation. Severe thrombophilia (e.g. homozygous factor V Leiden, protein C/S and antithrombin III deficiency) requires up to 12 months of anticoagulation (Caprio and Bernstein, 2012).

Thrombolysis or thrombectomy

Thrombolysis or thrombectomy has been described as a treatment for cerebral venous sinus thrombosis. There are multiple reports of the successful use of endovascular thrombolysis in cerebral venous sinus thrombosis (Ciccone et al, 2004). However, a randomized control trial or large study is required to study these treatments further.

Decompressive surgery

A small group of patients suffer from cerebral herniation as a result of venous haemorrhagic infarctions. These patients can be managed with decompressive surgery. A small retrospective multicentre registry studying decompressive surgery in cerebral venous sinus thrombosis showed some promising results. Although 18% of the 38 patients included died post operation, only 8% of the survivors were severely dependent at follow up. Thus, analogous to arterial stroke, there is a role for surgery in the management of cerebral venous sinus thrombosis (Ferro et al, 2011).

Complications

Early complications

The main early complications of cerebral venous sinus thrombosis are seizures, hydrocephalus and raised intracranial pressure.

Seizures

Seizures are more common in cerebral venous sinus thrombosis than in arterial strokes. In studies, this varies between 36 and 44% of the recruited populations (Kwan and Guenther, 2006). Currently, there are no data to support the administration of antiepileptic agents in primary and secondary seizure prevention (Kwan and Guenther, 2006). However, antiepileptics can be initiated for a defined time frame in patients with parenchymal lesions, cerebral venous sinus thrombosis and after a single seizure (Saposnik et al, 2011).

Hydrocephalus

A communicating hydrocephalus can occur as a result of the impaired function of the arachnoid granulations (Stam, 2005). An obstructing hydrocephalus occurs less often, as a result of intraventricular bleeding.

Intracranial hypertension

Patients with cerebral venous sinus thrombosis can present with raised intracranial pressure. Strategies to reduce intracranial pressure include acetazolamide therapy and serial lumbar punctures to remove CSF (Einhäupl et al, 2006). Anticoagulation must be commenced as well to treat the cerebral venous sinus thrombosis. If the intracranial pressure remains refractory to these measures, consider a neurosurgical referral for lumboperitoneal shunting (Einhäupl et al, 2006).

Late complications

The late complications of cerebral venous sinus thrombosis are recurrence, headache, seizures, visual loss and the formation of a dural arteriovenous fistula.

Recurrence

The ISCVT trial studied the risk of recurrence post-cerebral venous sinus thrombosis: 2.2% had a recurrent cerebral venous sinus thrombosis and 4.3% had a recurrent venous thrombosis (Ferro et al, 2004).

Headache

Severe, recurrent headaches are a well-recognized complication of cerebral venous sinus thrombosis. Up to 50% of all patients have ongoing headaches when followed up (Saposnik et al, 2011). It is important to be vigilant for recurrent cerebral venous sinus thrombosis and raised intracranial pressure when a patient with a previous cerebral venous sinus thrombosis presents with a headache.

Seizures

Seizures can occur both immediately and later in cerebral venous sinus thrombosis. Seizures occurred in up to 32% of patients in one study, mainly in the first year after diagnosis (Saposnik et al, 2011).

Visual loss

This is rare – a late sign but possible in patients presenting with raised intracranial pressure. It is important to perform continuous neuro-ophthalmology evaluation on these patients, as visual loss can be subtle (Saposnik et al, 2011). Typically, enlargement of the blind spot is the first sign. Transient visual obscurations can occur, for example with changes in position.

KEY POINTS

- Cerebral venous sinus thrombosis is the obstruction of cerebral veins or venous sinuses by thrombus, causing venous hypertension and oedema.
- Risk factors for cerebral venous sinus thrombosis can be categorized into inherited and acquired.
- Computed tomography or magnetic resonance imaging do not exclude venous sinus thrombosis. A magnetic resonance venogram or computed tomography venogram is essential. Formal angiography may be considered if clinical suspicion is strong and these results are negative.
- Acute complications include hydrocephalus, seizures and raised intracranial pressure.
- Cerebral venous sinus thrombosis is treated acutely with therapeutic dose low molecular weight heparin, with options for thrombolysis or thrombectomy in severe, refractory cases.

Dural arteriovenous fistulae

Dural fistulae occur after persistent dural sinus occlusion. The presence of a pre-existing fistula can also increase the risk of developing a cerebral venous sinus thrombosis (Saposnik et al, 2011).

Prognosis

Death

Early death occurs in cerebral venous sinus thrombosis mainly as a result of transtentorial herniation after cerebral haemorrhage, status epilepticus and pulmonary embolism and other medical causes. In the ISCVT study, 30-day mortality was 3.4% (Ferro et al, 2004). Late death is usually caused by the underlying medical condition (e.g. malignancy).

Long-term outcomes

In the ISCVT, complete recovery was seen in 79% of the patients (Ferro et al, 2004), and 13% of patients had an unfavourable outcome (8% death, 5% dependence). Risk factors for poor outcome include coma, cerebral haemorrhage, malignancy, CNS infection, male sex, age >37 years and mental status disorder (Ferro et al, 2004). Post-cerebral venous sinus thrombosis, the most common complications were seizures and repeated thrombosis (Ferro et al, 2004).

Conclusions

Cerebral venous sinus thrombosis is a rare, treatable condition, which requires a high index of suspicion to diagnose. It presents in multiple ways, with highly variable severity. As magnetic resonance imaging and computed tomography have advanced, imaging for diagnosis has become less invasive. Treatment involves managing complications and anticoagulating. There are many questions to answer for the future. These include the role of anti-epileptics, thrombolysis, thrombectomy and the novel oral anticoagulants. Novel oral anticoagulants are well established in other thrombotic conditions (e.g. deep vein thrombosis) but can they be successfully used in cerebral venous sinus thrombosis?

As progress is made in the diagnosis and cure of cerebral venous sinus thrombosis, the prognosis of this condition is favourable for most patients. **BJHM**

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Conflict of interest: none.

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