Pictorial Essay

Acute and critical paediatric neurological conditions: The role of the neuroradiologist

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Abstract

Acute paediatric neurological diseases are common conditions that can result in significant morbidity and mortality, particularly if a timely diagnosis is not made. Unfortunately, clinical presentations can be non-specific, with seizures and headaches being the most frequent complaints. It is therefore important for radiologists to be familiar with these conditions and their respective imaging features to allow prompt diagnosis and management.

The aim of this article is to briefly review the clinical presentation and imaging features across six common categories: intracranial haemorrhage, toxin-related injury, infections, autoimmune disorders, stroke, and seizures.

Keywords: Acute toxic leucoencephalopathy, ADEM, Neonatal CNS infection, Neuroradiologist, Paediatric, Paediatric stroke, PRES.

Background

In a multi-institutional study of 960,020 paediatric admissions across 11 states in the US, 10.7% of admissions included a neurologic diagnosis [1]. Children admitted with a neurologic diagnosis had a significantly longer median length of hospital stay, were nearly three times more likely than other hospitalized children to require intensive care, and had a threefold higher mortality rate [1].

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Imaging Modality

Non-contrast head CT is the study of choice in patients with suspected acute intracranial haemorrhage. It is also indicated for patients requiring rapid imaging due to acute changes in neurologic status, particularly if brain MRI is not readily available. As paediatric patients are more susceptible to the adverse effects of ionizing radiation exposure than adults, CT protocols must be optimized for paediatric dosing following the As Low As Reasonably Achievable (ALARA) principle. Contrast may be necessary when infection is suspected.

MRI is sometimes handicapped by its long scan time and the need for anaesthesia for younger children. However, it has higher sensitivity and specificity than CT and ultrasound for detecting many pathologies and hence remains a crucial tool in the diagnosis and monitoring of many paediatric neurological diseases. The use of non-pharmacological methods including "feed and wrap" in infants or MRI video displays in children should be employed whenever possible. Fast MRI protocols such as HASTE and SSFSE allow rapid image acquisition, which can minimize or sometimes eliminate the need for sedation. If sedation is deemed necessary, patients should be monitored closely by the anaesthesia or paediatric care team.

Intracranial Haemorrhage

A range of injuries to the scalp, skull, or brain may be seen in term babies following normal or assisted vaginal delivery. There are various maternal and fetal factors which increase the risk of cranial trauma.

In birth-related head trauma, the most common cranial injuries are subdural haematoma (SDH), cephalohaematoma, subarachnoid haemorrhage, and intraparenchymal haemorrhage [2] (Figure 1). Common locations of SDH include the tentorial, interhemispheric, cerebral convexity, and parafalcine regions [2]. Vacuum- and forceps- assisted delivery are by far the most common causes of birth-related head injuries [2] (Figure 2). Fractures caused by birth trauma are more frequently depressed than linear, and the parietal and frontal bones are most often involved [3].

The dating of extra-axial haematomas based on CT attenuation must be interpreted with caution. A study was conducted to analyse the first appearance of a hypodense component within an SDH in 11 infants who had a history of abusive head injury. Uniformly hyperdense SDH was seen on the initial CT scan. For each case, there is a

range of times over which the hypodense component could have appeared, and these ranged from a minimum of 0.3 days to a maximum of 16 days [4]. Multiple factors are known to affect the neuroimaging appearances of SDH, such as the concentration of red blood cells, the integrity of cell membranes, the protein content of the blood clot, and the amount of cerebrospinal fluid within the collection.

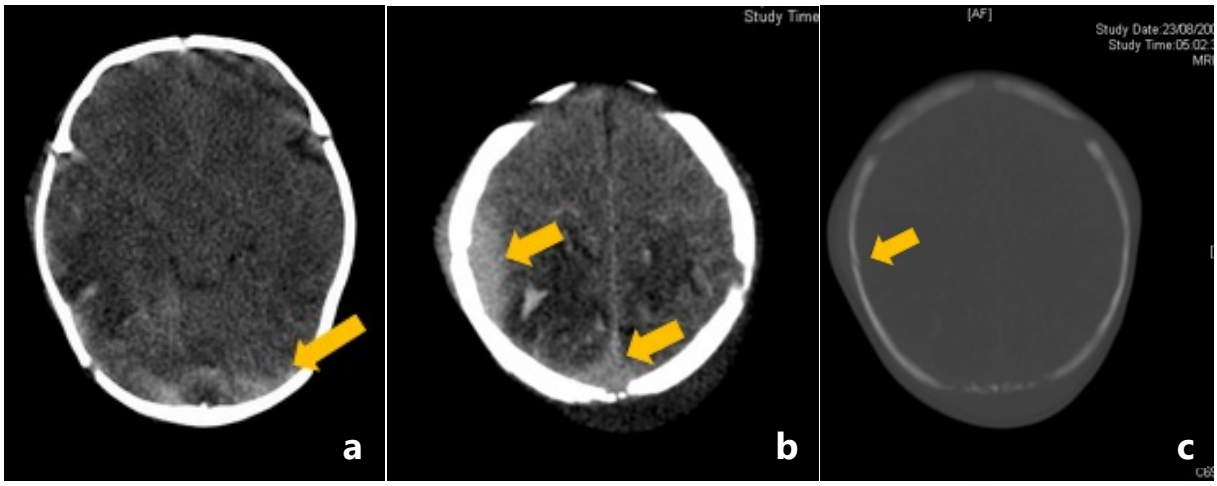

Figure 1. *Non-enhanced CT brain of a full-term infant born by caesarean delivery who presented with seizure and apnoea. (a) There is evidence of subdural haematoma along the tentorium, (b) epidural haematoma, and (c) a right parietal skull fracture.*

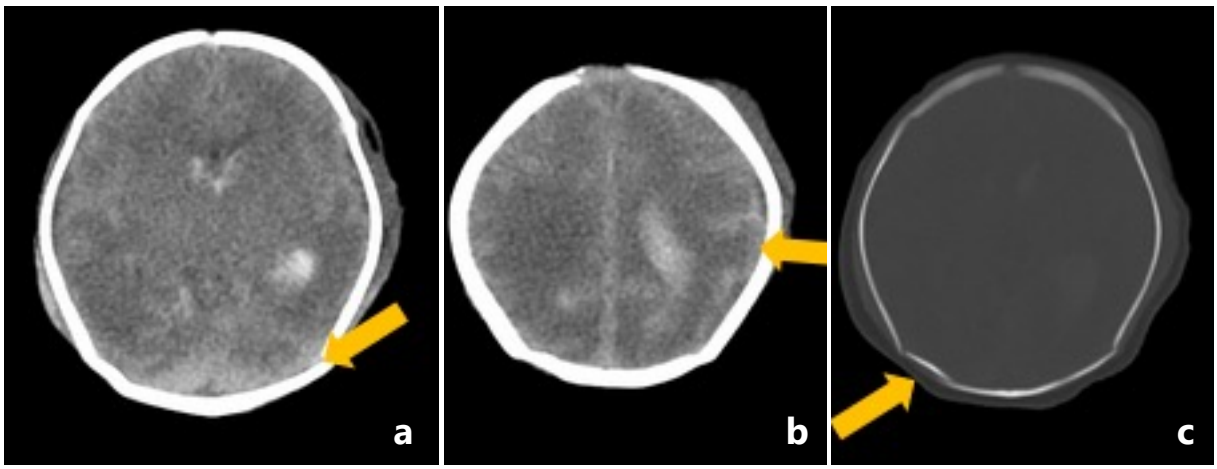

Figure 2. *Non-enhanced CT brain of a premature infant born at 32 weeks of gestation born via forceps-assisted delivery. (a) Subdural haematoma is seen along the tentorium (arrow), (b, c) demonstrate grade III intraventricular haemorrhage and subarachnoid haemorrhage (arrow), with marked cerebral oedema and sulcal effacement. (c) On the bone window, there is a mildly depressed fracture involving the occipital bone (arrow).*

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Toxin-Related Insults

There are multiple causes of neurotoxicity in children. These include intrinsic causes which result from the build-up of toxic metabolites due to genetic or acquired disorders, and metabolic abnormalities, as well as extrinsic causes such as medications, toxins, and other insults (Table 1).

Early identification of imaging findings can facilitate early diagnosis and the institution of appropriate treatment to reverse or limit injury to the developing brain.

Table 1. *Intrinsic and external causes of neurotoxicity in children.*

Intrinsic causes	Extrinsic causes
Inborn errors of metabolism	Chemotherapy and radiotherapy
Progressive organ failure causing toxic metabolites to build up	Medications, illicit drugs, and poisons

Two common imaging patterns of neurotoxicity of extrinsic nature are posterior reversible encephalopathy syndrome (PRES) and acute toxic leukoencephalopathy [5].

The pathogenesis of PRES is not well understood but is generally believed to be secondary to endothelial dysfunction due to exposure to exogenous toxins. Common causes include medications, including chemotherapeutic agents and steroids, infections, and transplantation (including bone marrow, stem cell, and solid organ transplantation). Common symptoms include headache, seizures, and encephalopathic features. Typically, there is bilateral vasogenic oedema of the parieto-occipital lobes, with sparing of the central white matter. Despite its name, frontal, temporal, and cerebellar regions can also be involved (frontal involvement is demonstrated in Figure 3). Atypical imaging patterns include purely unilateral involvement, brainstem or basal ganglia involvement without cortical white matter involvement (also known as central PRES), and spinal cord involvement. Progression to infarction and haemorrhage can also be seen. The various manifestations of PRES can sometimes make diagnosis challenging; therefore, correlation with clinical features as well as predisposing factors is important. Supportive treatment and removal of the offending agent are required.

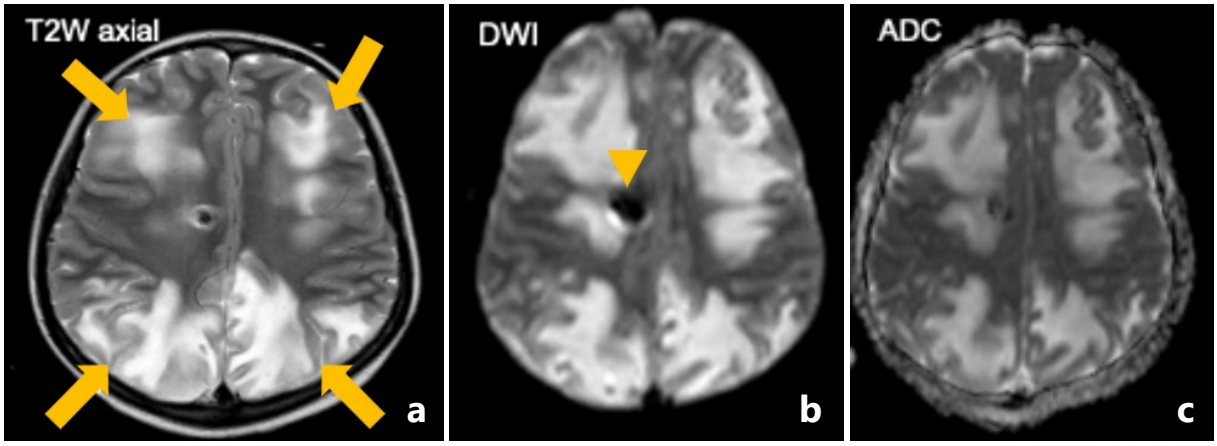

Figure 3. *Non-enhanced MRI brain of a 13-year-old patient on induction chemotherapy for acute lymphoblastic leukaemia, presenting with seizures and labile hypertension [6]. (a) The T2 sequence shows bilateral symmetrical parenchymal abnormalities (arrows) with DWI (b) and ADC sequences (c) showing vasogenic oedema. These findings are consistent with PRES. An extra-ventricular drainage catheter was inserted for monitoring of intracranial pressure (arrowhead).*

Acute toxic leukoencephalopathy is a neurotoxic condition that predominantly damages the white matter. It is most commonly secondary to medications, namely methotrexate and other chemotherapeutic agents such as fluorouracil. Methotrexate use is the most common cause among the paediatric oncological population, and the risk increases with high-dose regimens and intrathecal administration. These patients typically present 2-4 weeks after methotrexate administration. Sepsis and metabolic abnormalities such as uraemia are less common causes. Clinical presentations vary and can range from a decreased level of consciousness to more severe manifestations including hemiplegia and aphasia. On imaging, these typically manifest as restricted diffusion in the unilateral or bilateral centrum semiovale, which crosses vascular territories. In contrast, restricted diffusion is only present in 25% of PRES cases. Treatment with aminophylline and leucovorin may aid in reversing the acute parenchymal changes.

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Infections

Neonatal infections of the brain are often non-specific. Early imaging, particularly MRI, aids in early diagnosis, which can avoid devastating outcomes including neurodevelopmental delay.

Neonatal leptomeningitis occurs in 0.4% of births. The commonest pathogens are group B streptococcus and Escherichia coli [7]. Imaging findings include enlargement of the subarachnoid spaces, subdural effusions (Figure 4) as well as leptomeningeal thickening and enhancement (Figure 5). Parenchymal involvement could lead to cerebritis and cerebral liquefaction. Inflammation of meninges can also affect the perivascular spaces, leading to infarction. This occurs in approximately 10% of neonates with meningitis [8]. Other complications include cerebral abscesses and empyema formation.

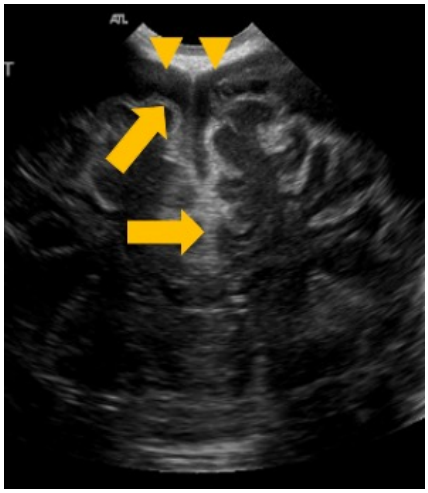

Figure 4. *USG brain of a 5-month-old infant presenting with late-onset GBS meningitis. There is marked leptomeningeal thickening (arrow) and bilateral subdural effusions (arrowhead), causing mild mass effect on the underlying brain.*

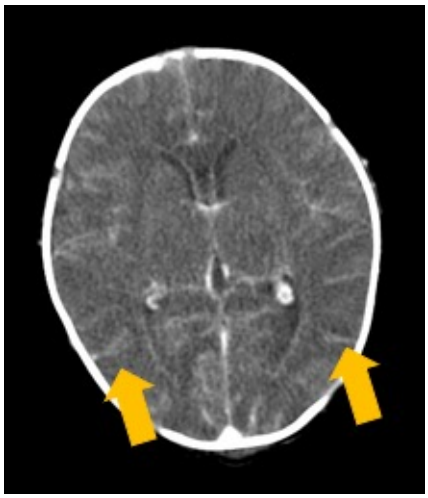

Figure 5. *Contrast-enhanced CT brain of a term infant presenting on the day of birth with septic shock, respiratory failure, and leptomeningitis. There is marked cerebral oedema with effacement of the sulcal spaces, while the lateral ventricles demonstrate increased ependymal enhancement and mild dilatation. Increased leptomeningeal enhancement is also noted (arrows).*

Viral encephalitis is commonly caused by herpes simplex virus (HSV) types 1 and 2, cytomegavirus (CMV) and human herpesvirus-6 (HHV) HHV-6. Patients in the early post-transplant period following haematopoietic stem cell transplantation are particularly at risk. Imaging features are non-specific, though abnormal T2 hyperintensities involving both grey and white matter are often seen. Associated restricted diffusion caused by cytotoxic oedema is also a common feature (Figure 6). Classic imaging patterns associated with different viruses have also been described. In CMV infections, there is periventricular white matter involvement with ependymal enhancement. In HSV infections, the limbic system and periventricular white matter are often involved, and the basal ganglia is typically spared. HHV-6 affects the limbic system, with involvement of the hippocampus and amygdala. Of note, these classical findings are sometimes absent due to the patients' impaired immune response.

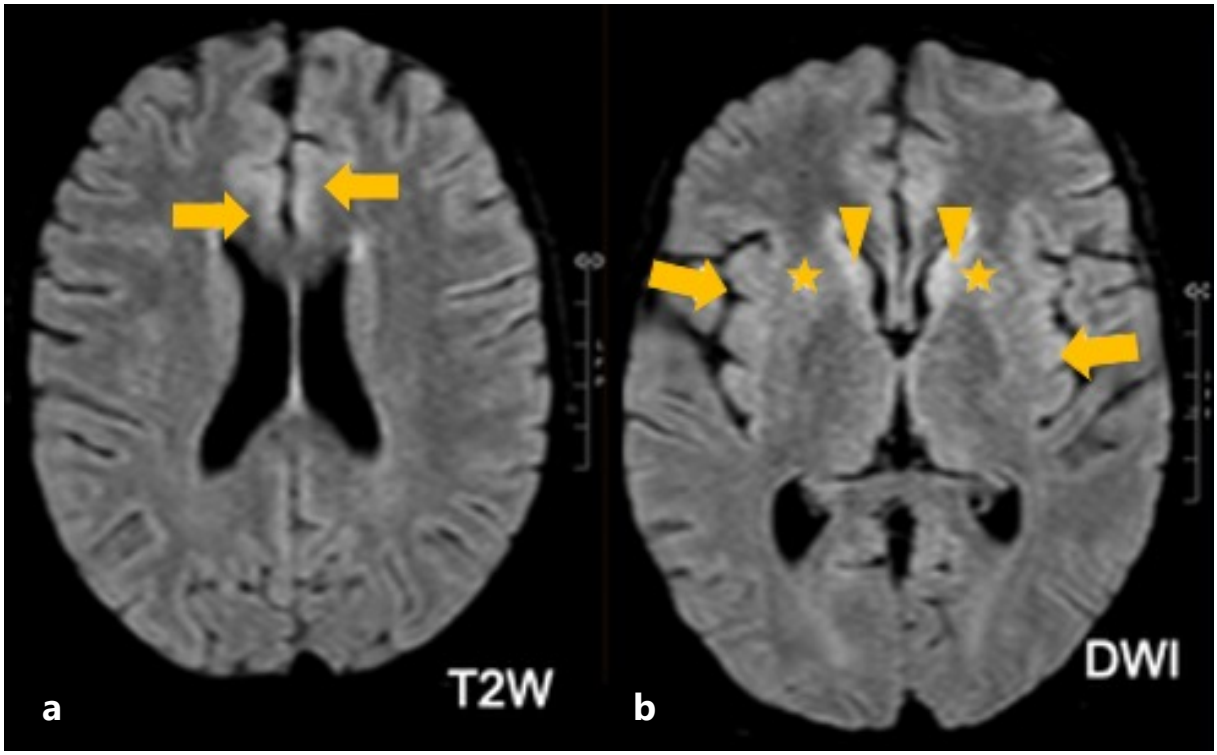

Figure 6. *MRI brain of a patient with acute myeloblastic leukaemia, following peripheral blood stem cell transplantation [6]. There are abnormally high T2 signals in the bilateral medial frontal lobes (a), anterior putamina (b, asterisks), caudate heads (b, arrowheads) and insula (b, arrows). Findings may suggest, but not limited to, viral encephalitis.*

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Among immunosuppressed patients, invasive fungal disease is a devastating complication with a high rate of mortality and morbidity. Unfortunately, clinical signs and symptoms are often non-specific, posing difficulties for early diagnosis. More common CNS-related symptoms include headache, seizures, and impaired vision. Radiological findings are also often non-specific. CT is commonly the initial imaging modality as it allows rapid assessment of potential complications including haemorrhage, hydrocephalus, and brain abscess. MRI, on the other hand, is more sensitive in the diagnosis of meningitis (Figure 7), cerebritis, and ventriculitis.

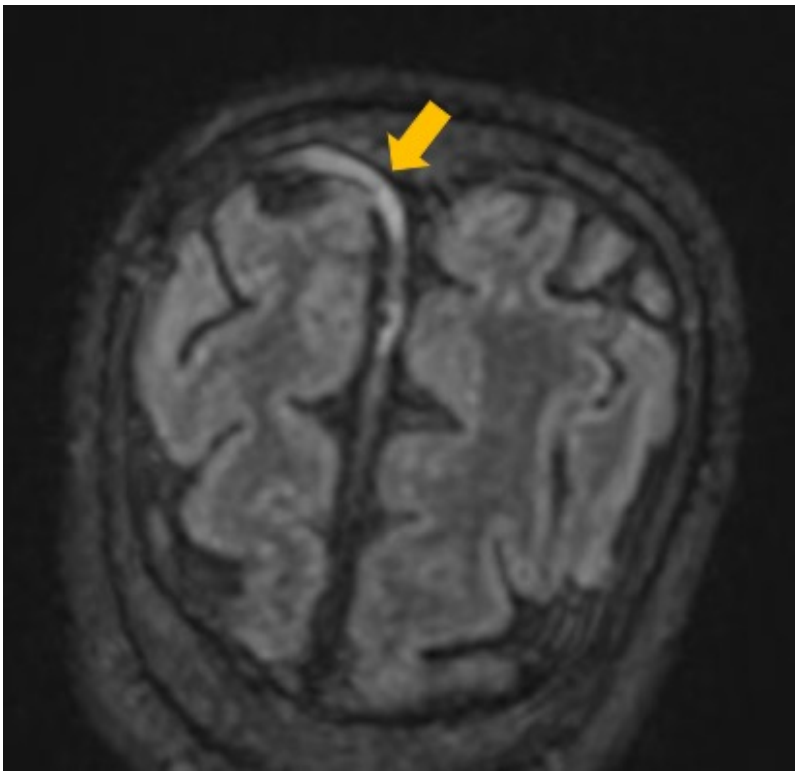

Figure 7. *Contrast-enhanced MRI brain of a 14-year-old boy with acute myeloblastic leukaemia [6]. He had invasive fungal infection involving the right nasal cavity and maxillary sinus. Contrast-enhanced MRI shows increased dural enhancement along the right frontal anterior falx (arrow), suggestive of intracranial extension of infection.*

Autoimmune Disorders

Associations and risk factors for central nervous system (CNS) autoimmunity are heterogeneous. Genetic defects can lead to immune disorders, resulting in autoimmune and lymphoproliferative diseases and immunodeficiency. Environmental factors, including exposure to hormones, and vitamin D levels, also play important roles. Infection and neoplasm are common triggers for first presentations and flare-ups. Autoimmune CNS conditions can be divided into primary CNS diseases and systemic autoimmune conditions with CNS involvement (Table 2).

Table 2. *Examples of primary CNS conditions and systemic conditions with CNS involvement.*

Primary CNS conditions	Systemic conditions
Multiple sclerosis	SLE
Acute disseminated encephalomyelitis	Sarcoidosis
Neuromyelitis optica spectrum disorder	Behcet's
Autoimmune encephalitis	Granulomatosis with polyangiitis
Primary vasculitis	Scleroderma
	Sjogren's syndrome
	Thrombotic microangiopathies
	Aicardi Gouteire
	Hemophagocytic lymphohistiocytosis

An example of a primary CNS autoimmune disorder is acute disseminated encephalomyelitis (ADEM), which is a clinical diagnosis that describes an acute presentation of polyfocal demyelination, accompanied by encephalopathy. It is usually a monophasic disorder. Peak onset occurs between 5 and 8 years of age, and a history of recent infection or vaccination is common. Commonly involved regions include the grey matter, particularly the basal ganglia, subcortical white matter, thalami and brainstem (Figure 8). The spinal cord may also be involved to a lesser extent (Figure 9).

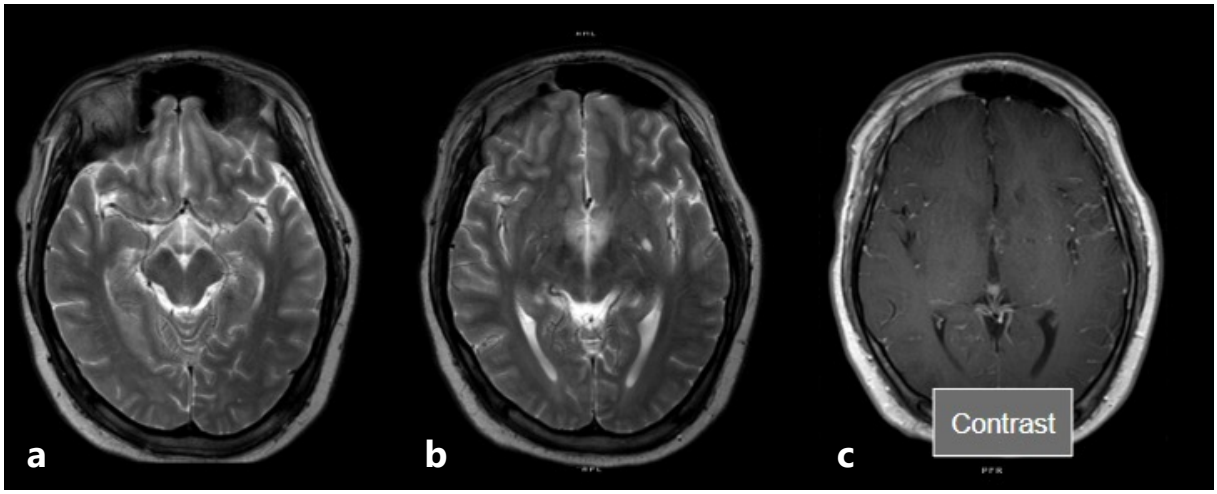

Figure 8. *Contrast-enhanced MRI brain of a 16-year-old patient with ADEM presenting with acute-onset bilateral lower limb weakness, acute retention of urine, and a sensory level at T4. On T2-weighted sequences, there are bilateral symmetrical T2 hyperintense signals in the bilateral paraterminal gyri (a, b), extending to the mamillary bodies. Post-contrast imaging shows mild leptomeningeal enhancement (c).*

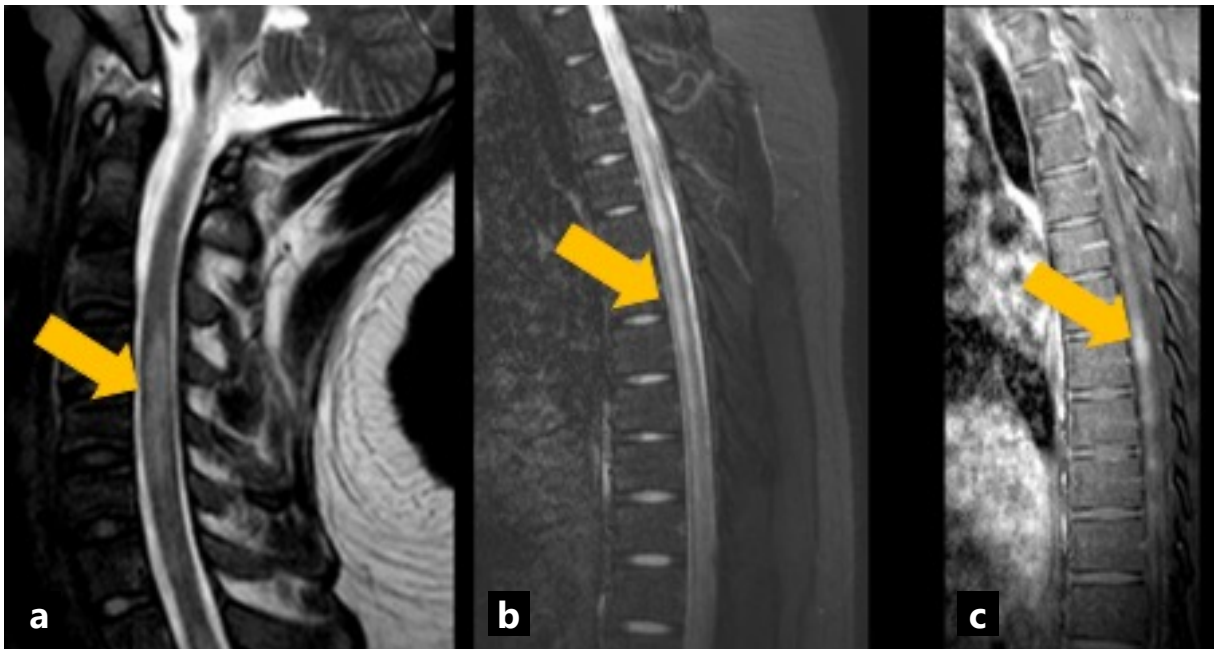

Figure 9. *CMRI spine of the same patient as Figure 8. Extensive T2 hyperintense signals are seen involving C4-7, T1-11 (a, b) with patchy enhancement on post-contrast sequences (c).*

A less common example of primary CNS autoimmune disorder is steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT), also known as Hashimoto's encephalopathy. This is a rare autoimmune encephalitis associated with autoimmune thyroid disease. Imaging findings are non-specific. Reported MRI findings include white matter T2W hyperintensity and dural enhancement (Figure 10).

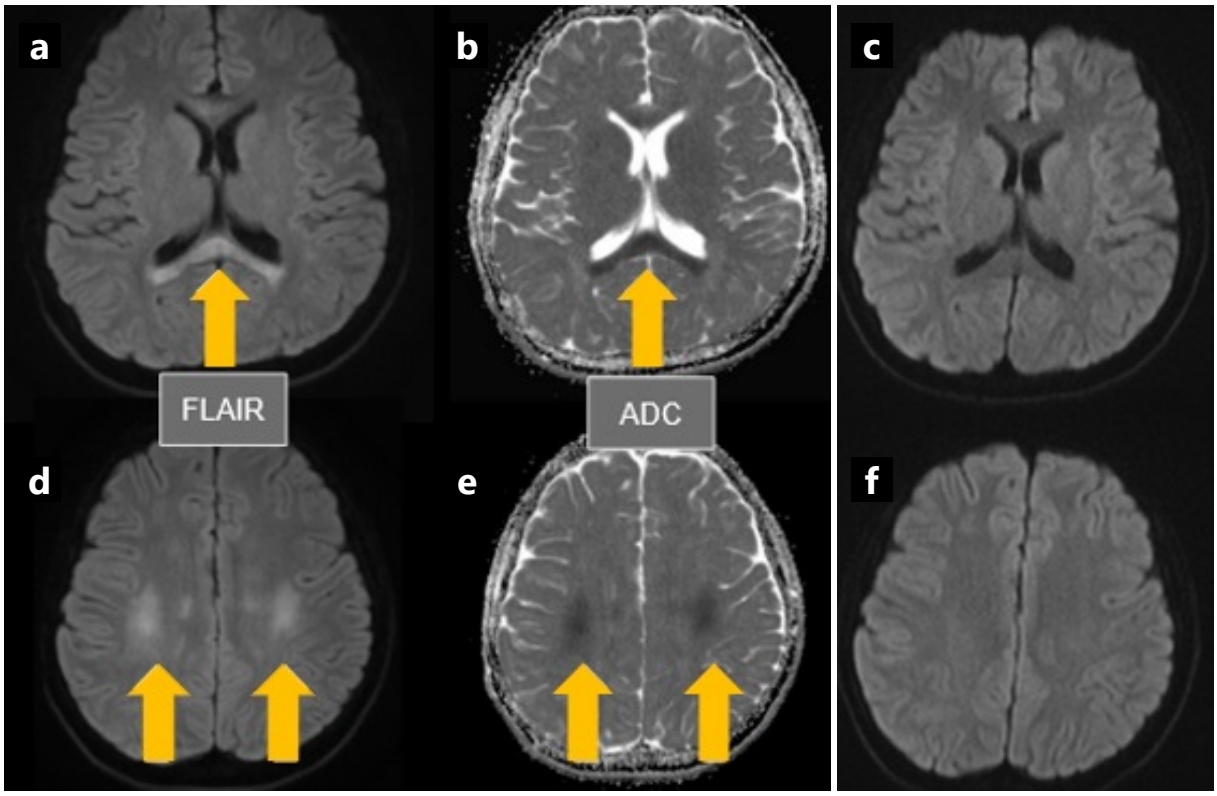

Figure 10. *Non-enhanced MRI brain of a 10-year-old patient with SREAT, who presented with thyroid storm and sudden onset of confusion and aphasia, with a GCS of 9/15. MRI FLAIR sequence shows T2 hyperintense and T1 hypointense signals in the splenium of corpus callosum (a, c, arrow) and bilateral centrum semiovale (b, d arrow). Non-enhanced MRI brain (e, f) FLAIR sequence of the same patient after 11 days of treatment (methylprednisolone and plasma exchange) demonstrates resolution of previously noted T2/FLAIR hyperintense signals.*

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Stroke

Stroke is rare in the paediatric population, with an incidence of 2.3-13 per 100,000 children. However, it is one of the commonest causes of childhood death [9]. Unlike the adult population, paediatric stroke is evenly distributed between ischaemic and haemorrhagic subtypes. Risk factors also differ, with arteriopathies, congenital heart disease, prothrombotic conditions, sickle cell disease, and vasculitis being common risk factors. Ischaemic stroke is further divided into arterial ischaemic stroke and cerebral sinovenous thrombosis (Table 3). While acute ischaemic stroke presents as focal neurological deficits, most patients with cerebral sinovenous thrombosis and haemorrhagic stroke present with non-specific symptoms, including headache and seizures.

Table 3. *Comparison between arterial ischaemic stroke and cerebral sinovenous thrombosis.*

Arterial ischaemic stroke	Cerebral sinovenous thrombosis
Focal deficit localising to an arterial distribution	Non-specific symptoms: headache, papilledema, nausea, seizures
Arteriopathies (moyamoya vasculopathy, focal cerebral arteriopathy, or dissection), congenital heart disease, prothrombotic conditions, sickle cell disease, systemic vasculitis	Head and neck disorders, acute and chronic systemic illnesses, prothrombotic states

One example of arterial ischaemic stroke is focal cerebral arteriopathy of childhood, which is associated with a high risk of recurrent ischaemic stroke. This condition is believed to be inflammatory, classically caused by Varicella zoster infections, and Herpes infections in some cases. MRI angiography can be used to visualise unilateral stenosis of the distal internal carotid artery, and proximal middle and/or anterior cerebral arteries. Vessel wall imaging could reveal wall thickening and concentric enhancement.

Intracranial venous thrombosis is associated with the use of L-asparaginase and high-dose corticosteroids (Figure 11). Key imaging findings include hyperattenuated and expanded veins on non-enhanced CT, filling defects and the “empty delta” sign on CT venography, and infarcts that are not confined to an arterial territory. Haemorrhagic infarcts can also be seen.

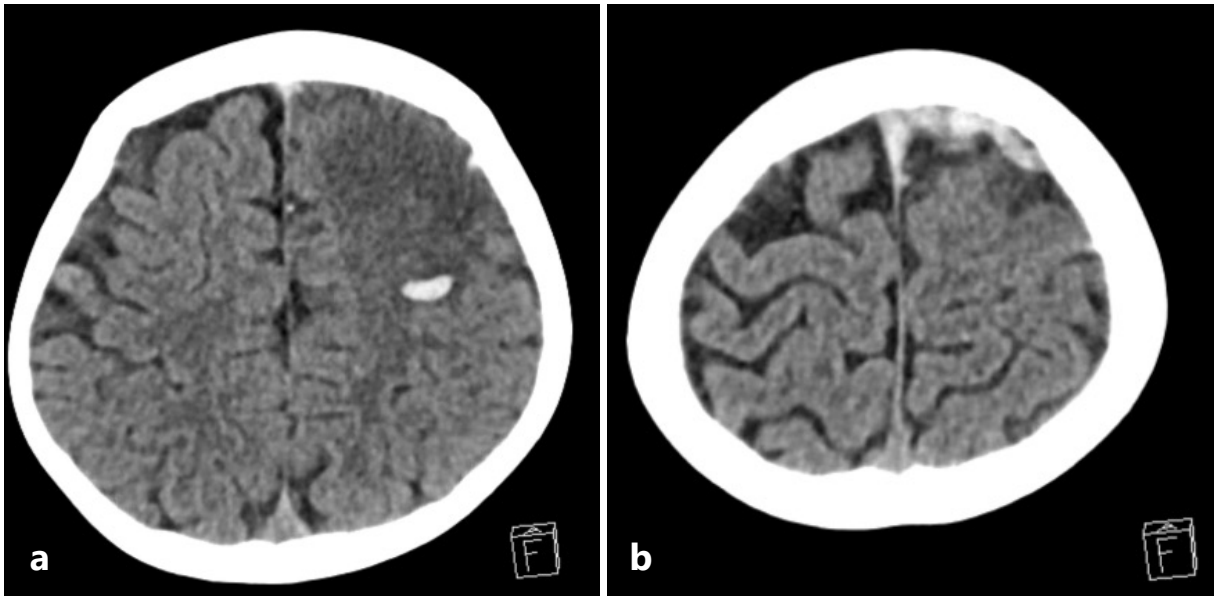

Figure 11. *Non-enhanced CT brain of a 6-year-old boy with acute lymphoblastic leukaemia who had received L-asparaginase and high-dose corticosteroids. (a) Hypodensity involving the left frontal lobe with loss of grey-white differentiation, suggestive of a recent infarct with an acute haematoma. (b) Hyperdensity within the superior sagittal sinus near the vertex with extra-axial curvilinear hyperdensities, suggestive of venous thrombosis and subdural haemorrhage.*

Seizures

Seizures are transient occurrences of signs and/or symptoms due to abnormally excessive or synchronous neuronal activity in the brain. Epilepsy refers to recurrent and unprovoked seizures. These range from the benign and self-limiting simple febrile seizures to life-threatening status epilepticus. The Centers for Disease Control and Prevention estimate that approximately 0.6% of children younger than 17 years of age suffer from epilepsy [10]. Status epilepticus is the commonest neurologic emergency in children. Underlying causes include genetic factors and intracranial and systemic abnormalities.

Children with neurometabolic disease can present with seizures. For example, urea cycle disorders are caused by a deficiency of enzymes that convert ammonia to urea. Hyperammonaemia results in diffuse generalised oedema involving the cerebral cortex and the subcortical white matter, which could be caused by increased levels of glutamine resulting from the metabolism of ammonia. In addition, the basal ganglia may show abnormal signal intensities, including T1 and T2 hyperintensities of the globus pallidi and T2 hyperintensity of the putamina and caudate nuclei. This pattern of basal ganglia involvement distinguishes urea cycle disorders from hypoxic-Ischemic encephalopathy. While T1 and T2 findings are relatively non-specific, a scalloped ribbon pattern of injury at the depth of sulci on diffusion-weighted imaging strongly supports the diagnosis (Figure 12).

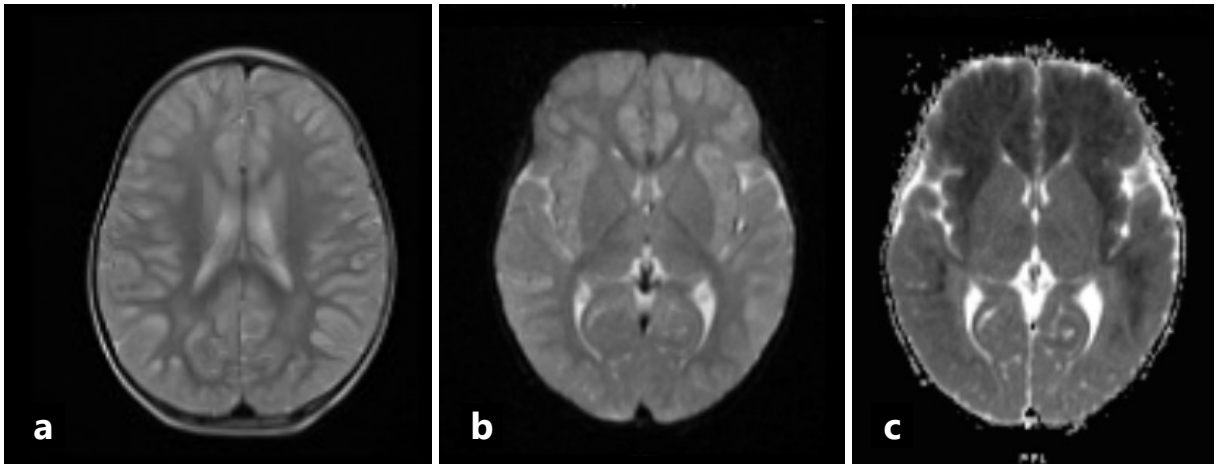

Figure 12. *Non-enhanced MRI brain of a 2-year-old boy with hyperammonaemia, presenting with seizures. (a) A T2 weighted sequence shows increased T2 signals in the bilateral subcortical white matter. (b) DWI and (c) ADC images show wavy bands of restricted diffusion at the cortical-subcortical junction of the insular regions (also known as the deep sulcus sign).*

Conclusion

Acute paediatric neurological diseases can result in morbidity and mortality, yet they commonly present with non-specific symptoms. In this article, we reviewed common acute critical neurological diseases in the paediatric population. Imaging can help identify specific entities when provided with a precise clinical history and unequivocal imaging findings, such as the diagnosis of empyema in a patient with known meningitis. In other cases, imaging can help localise abnormalities that correlate with specific clinical findings, such as anatomical localisation in autoimmune diseases or acute ischaemic stroke. Even when both clinical and radiological findings are inconclusive, imaging features can often help narrow the list of differential diagnoses. Thus, imaging plays a pivotal role in the accurate and timely diagnosis of critical neurological diseases.

Conflicts of interest

The authors declare no conflict of interest.

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