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CASE REPORT

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A review of pathologies associated with high T1W signal intensity in the basal ganglia on Magnetic Resonance Imaging

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

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Summary

With several functions and a fundamental influence over cognition and motor functions, the basal ganglia are the cohesive centre of the brain. There are several conditions which affect the basal ganglia and these have various clinical and radiological manifestations. Nevertheless, on magnetic resonance imaging there is a limited differential diagnosis for those conditions presenting with T1 weighted spin echo hyperintensity within the central nervous system in general and the basal ganglia in particular. The aim of our review is to explore some of these basal ganglia pathologies and provide image illustrations.

Keywords: Basal Ganglia • MRI • T1W Signal Intensity

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Background

The basal ganglia include the striatum, the globus pallidi, the subthalamic nuclei and the substantiae nigrae [1]. In addition, the central complex of the thalamus and the pedunculopontine nuclei play an important role in the functions of the basal ganglia.

The caudate nuclei originate from the telencephalon. The putamina and the globus pallidi originate from the junction of the telencephalon and diencephalon. The subthalamic nuclei (STN) and the substantiae nigrae originate from the diencephalon and mesencephalon respectively [2]. In this review we focus on the telencephalic basal ganglia (globus pallidi, putamina and caudate nuclei).

The putamen controls movement and regulates several types of learning. Along with the globus pallidus, it constitutes the lentiform nucleus. The role of the basal ganglia extends beyond constituting part of the extra-pyramidal system to memory, emotion and other cognitive functions [3].

The main causes of high T1 intensity were divided into three main categories: blood products, calcification and metals. For each of these sections a few examples are given with image illustrations.

Blood Products

In both the early (days 3–7) and late (1–2 weeks) sub-acute phases, methaemoglobin has high T1 weighted signal. Red cells are intact in the early subacute phase causing a low T2 weighted signal, whereas haemolysis in the late subacute phase leads to the release of methaemoglobin into the haematoma cavity and high signal on T2 weighted images. Below are a few examples of high T1W intensity which are attributed to blood products.

Haemorrhagic infarct (Figure 1) and hypoxic ischemic encephalopathy (Figure 2)

HIE may result from an insult related to birth, cardiac arrest, drowning or asphyxiation. Profound asphyxia most commonly affects the basal ganglia, the thalamus, the

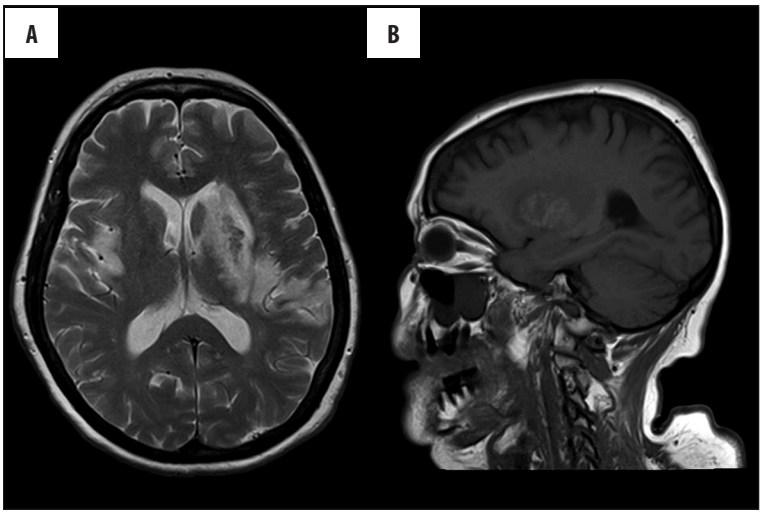


Figure 1. (**A**) Axial T2W and sagittal T1W images respectively of a patient with bilateral middle cerebral arteries infarcts. (**B**) Haemorrhagic transformation on the left lentiform nucleus seen as a high T1W signal.

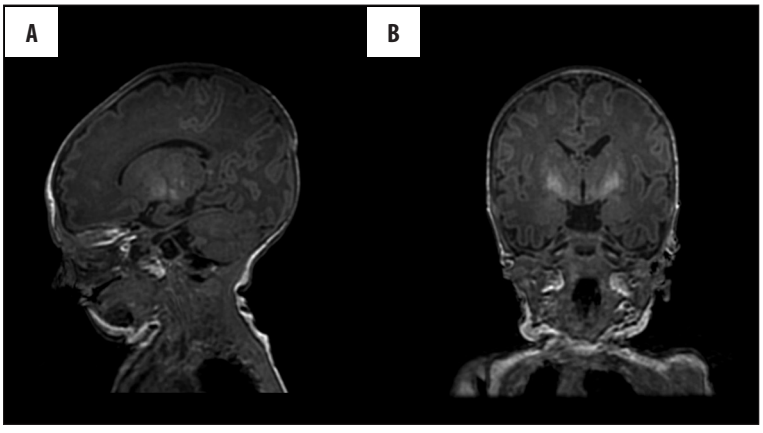


Figure 2. (**A, B**) A sagittal and coronal T1W images respectively of an infant with hypoxic ischemic encephalopathy demonstrating high T1 weighted signal in the globus pallidi.

peri-rolandic cortex, the sub-cortical white matter and the hippocampus [4]. The reason for the hyperintensity is postulated to be related to minute haemorrhages due to leakage of the red cells through the damaged endothelium after reperfusion in the subacute phase.

Carbon monoxide poisoning

Carbon monoxide poisoning predominantly affects the globus pallidi [3]. Other areas that may be involved in the brain include the deep white matter, occasionally the putamina, the caudate nuclei, thalami and hippocampus [5,6]. The reason for this predilection is not fully understood. Necrosis in the globus pallidi is a common pathological finding and manifests as high T2 and low T1 weighted signals. However, haemorrhage can occur and produce high T1 weighted signal [7]. Contrast enhanced T1 weighted images may show patchy or peripheral enhancement and diffusion weighted imaging demonstrates restricted diffusion as a result of cytotoxic oedema associated with acute tissue necrosis [8].

Methanol poisoning

Methanol is converted into its toxic substrate formate. Optic neuritis is usually the initial manifestation. Haemorragic necrosis of the putamina may occur following methanol poisoning [3].

Calcification

With basal ganglia calcification the commonest finding on MRI is hypo-intensity on T1 and T2 weighted images due to insufficiency of mobile protons. The occasional T1 weighted hyperintensity with calcification is thought to be due to the crystalline structure of the calcium. The larger the calcium particle the shorter the T1 weighted relaxation time [9]. This is known as the surface relaxation mechanism [9,10]. The motion of the liquid molecules at the interface with a solid particle is restricted. Their rotational and translational frequencies approach the Larmor frequency, hence their shorter relaxation time. The causes of calcifications were divided into acquired and congenital and below are few examples given for each.

Acquired calcification

Physiological calcification

Physiological calcification is usually punctate. It is almost always found in the globus pallidi but can also occur in the head of the caudate nuclei and the putamina. Physiological calcification becomes more prevalent with age. It is unusual to find physiological calcification in individuals under the age of thirty. Alternative diagnoses such as Cockayne syndrome, metabolic causes or infections should be considered in younger patients.

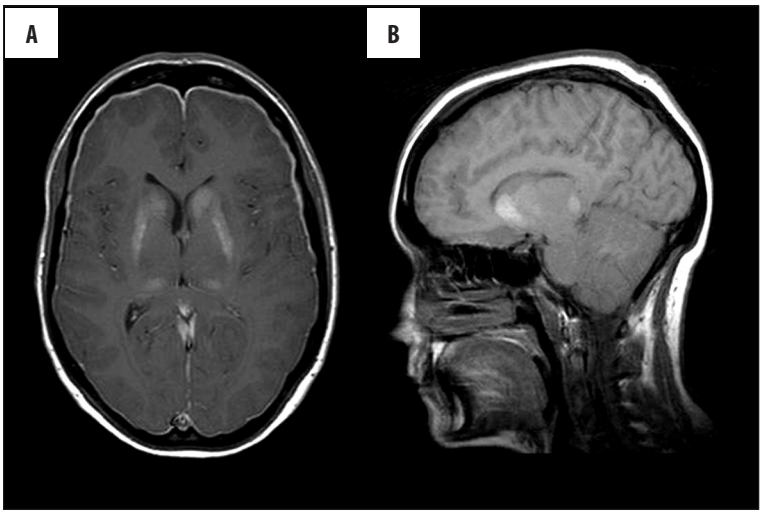


Figure 3. (**A**) axial T1WI post gadolinium and (**B**) sagittal non contrast T1WI of a patient with pseudohypoparathyroidism and spontaneous intra-cranial hypotension (SIH) demonstrating high signal within the putamina, heads of the caudate nuclei and pulvinar as well as dural enhancement due to SIH.

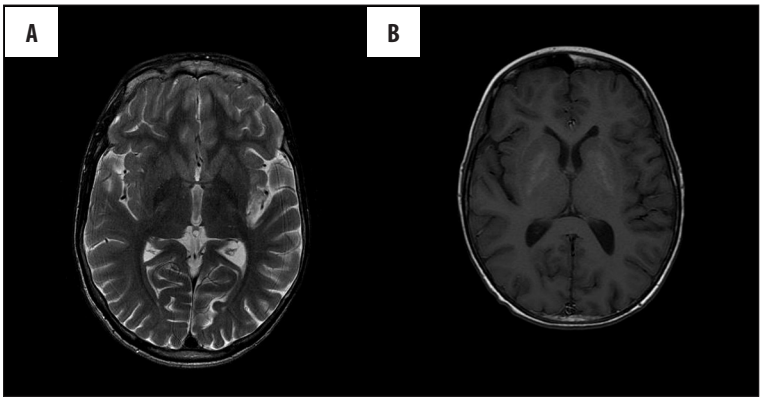


Figure 4. (**A**) Axial T2WI in a patient with left insular astrocytoma demonstrating residual high T2W signal change following chemoradiation. (**B**) Axial T1WI for the same patient post-chemoradiation showing high signal in the globus pallid.

Metabolic causes (Figure 3)

Endocrine abnormalities may produce basal ganglia calcification. These include hyperparathyroidism or hypoparathyroidism and pseudohypoparathyroidism.

Chemotherapy and radiation induced dystrophic calcification (Figure 4)

Mineralising microangiopathy is a rare histopathological complication following radio-chemotherapy which has been more commonly reported in patients who received radiation treatment before the age of ten. It affects the microvasculature of the central nervous system by fibrinoid necrosis, hyalinization and calcium deposition [9]. The calcification may display paradoxical T1 weighted hyperintensity due to the surface relaxation mechanism.

Infections

Basal ganglia calcification associated with infection is usually asymmetrical and is just one feature in the spectrum of other findings. The most frequent infections that cause calcification include cytomegalovirus, toxoplasmosis, Epstein Barr virus and tuberculosis. In CMV infection, intracranial calcification is the most common finding on imaging. The calcifications tend to occur in the periventricular white matter and the basal ganglia. Calcification within the basal ganglia tends to be punctuate unlike the calcification in other regions, which tends to be more extensive.

In acquired toxoplasmosis affecting immune-compromised patients, the MR imaging may demonstrate several lesions in the basal ganglia and at the grey white matter junction. These lesions may be haemorrhagic and hyperintense on T1 weighted images. However, typically the lesions are hypo or isointense on T2 weighted images with significant mass effect and vasogenic oedema. These lesions may show calcification post treatment, which may be punctuate or more extensive.

Congenital calcification

Cockayne disease (Figure 5)

This autosomal recessive condition presents with dwarfism, microcephaly, progressive pigmentary retinopathy, cutaneous photosensitivity, ataxia, premature aging, growth and mental retardation [11]. The disease is caused by deficiency in the DNA repair mechanism [12].

The small vessels are involved by mural and extramural colloid deposits which contain iron and calcium and occur predominantly in the basal ganglia, the dentate nuclei and the cerebral white matter [7]. MRI shows atrophy and T2 prolongation in the periventricular white matter, basal ganglia and the cerebellar dentate nuclei. Cortical U fibers are commonly involved in the later stages of the disease. Calcifications predominantly affect the putamina of the basal ganglia and less often the cortex and dentate nuclei.

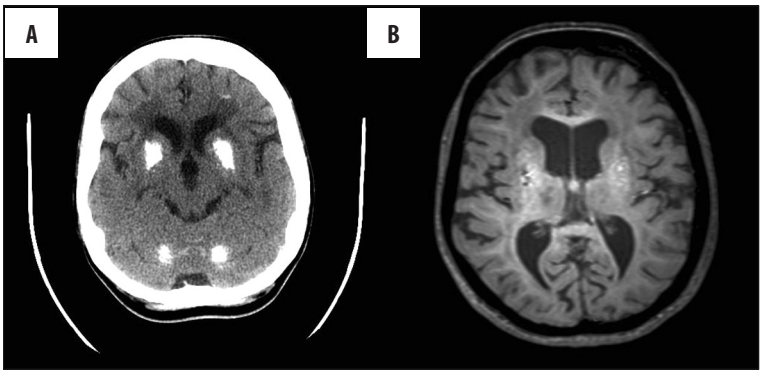


Figure 5. (**A**) AxiaL CT image of a patient with Cockayne Syndrome showing calcification in the globus pallidi and the dentate nuclei. (**B**) Axial T1 weighted image demonstrating areas of hyperintensities within the globus pallidi bilaterally containing small areas of low T1 weighted signal within the right globus pallidus.

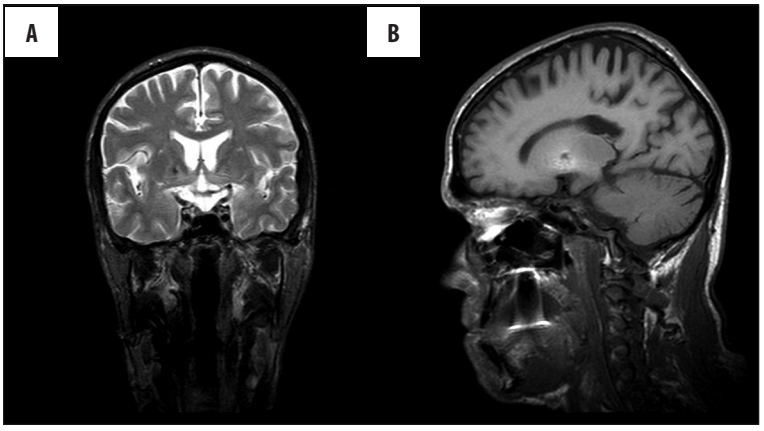
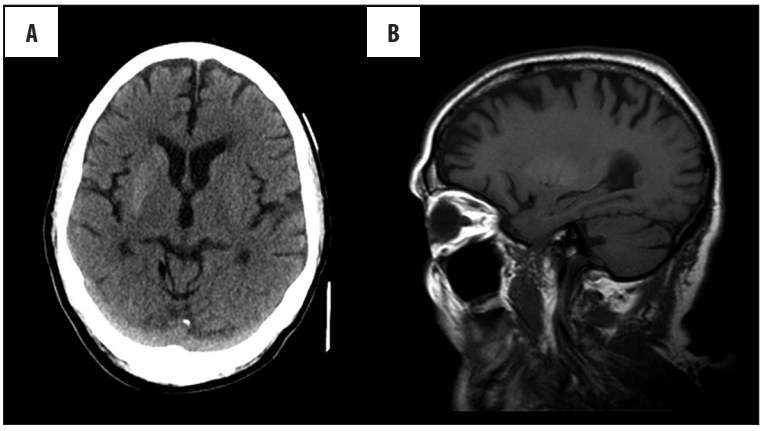


Figure 6. (**A**) coronal T2WI and (**B**) sagittal T1WI of a patient with alcoholic liver disease showing abnormal high intensity in the right globus pallidus with central focal low signal.



Figures 7. (**A**) Axial CT image of an 82 year old male patient who presented with hemiballismus affecting the left arm and leg, showing high density within the right putamen. (**B**) Sagittal T1W image demonstrates high signal within the right putamen corresponding to the area of high density on the CT scan.

Fahr disease (bilateral striopallidodentate calcinosis)

Fahr disease is part of a spectrum of conditions characterised by idiopathic basal ganglia calcification. It can be transmitted as an autosomal recessive condition but autosomal dominant transmission has also been described. The calcifications are characteristically extensive and occur in the globus pallidi, putamina, the caudate nuclei, the thalami, dentate nuclei, the cerebellum and the centrum semiovale [3] but are particularly marked in the former [13]. Calcium accumulates within the walls of the capillaries and larger arteries and veins. Other elements such as zinc, phosphorus, magnesium, aluminium and potassium are also present. There are no recognised abnormalities of calcium or phosphate metabolism. The clinical presentation includes extra pyramidal symptoms, mental deterioration, speech disturbances. MRI signal is variable on T1 and T2 weighted sequences and may be both high and low on T1 and T2 weighted sequences.

Metals

The basal ganglia contain high concentrations of metals such as iron, copper, and manganese and constitute a frequent site for their deposition. It is the high concentrations of iron that account for the characteristic normal appearances of the basal ganglia on MRI. The globus pallidi are slightly hypointense whereas the caudate nuclei and the putamina are isointense to the cortical grey matter on T1 and T2 weighted sequences [3]. These metals are crucial cofactors required for normal metabolic processes, but they are also implicated in pathology. Iron for example catalyses the production of free oxygen radicals through the Haber Weiss reaction. These deleterious radicals interact with

several molecules (classically with lipids) to extract hydrogen through the process of peroxidation [14–16].

Hepatic failure and mangnanese toxicity (Figure 6)

Copper and manganese are excreted through the hepatobiliary system. High serum manganese can occur in patients with hepatic failure and in those on total parenteral nutrition [17,18]. This may subside when the liver function is corrected or the total parenteral nutrition is stopped. Manganese poisoning has also been described in welders exposed to welding fumes. The presenting symptoms overlap with Parkinson disease and include intention tremor and a staggering gait [19]. The deposition of these paramagnetic substances can lead to high T1 weighted signal on MRI in the basal ganglia. The globus pallidi are the most commonly affected areas, but it may also damage the substantia nigra [3,20] and hence the clinical overlap with Parkinson's disease.

Other Causes

Hyperglycemia associated with hemichorea and hemiballismus (Figure 7)

Hyperglycemia associated with hemichorea and hemiballismus (HC-HB) has been described as the presenting symptom of new onset diabetes, with type 2 and rarely with type 1 diabetes [20]. It could give high T1 and low T2 weighted signal in the contralateral putamen of the basal ganglia and high density on CT [20–22].

It has been postulated that the imaging appearances in HC-HB are related to a haemorrhagic process, which could account for the high T1W signal. However pathological studies from the high T1 weighted signal areas within the basal ganglia demonstrated astrocytosis without haemosiderin deposition [1].

Conclusions

The main entities causing high T1 weighted signal in the basal ganglia include calcifications, haemorrhage and metal deposition. However, some disease processes such as infection and malignancy could give high T1 weighted signal due to one or a combination of factors such as haemorrhage and calcification.

The commonest cause of basal ganglia calcification is physiological. However, there is a differential that should be considered in the appropriate clinical context.

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