MR imaging – an unreliable and potentially misleading diagnostic modality in patients with intracerebral calcium depositions

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Abstract

BACKGROUND: Since magnetic resonance imaging (MRI) is a method of choice for establishing the correct diagnosis in a great majority of neurologic disorders, especially in detection the causes of seizures and both acute and slowly progressive neurologic disturbances, computerized tomography is becoming more frequently excluded from obligate spectrum of diagnostic protocol.

METHODS: MRI was performed on 1.5 T MR scanner in two patients, in one suffering from pseudohypoparathyroidism, and in another with Fahr disease, while CT was initially excluded from the diagnostic protocol. In third patient, 11-yaer-old boy with hypercalcemia, both CT and MRI were indicated because of seizure attack.

RESULTS: Completely normal appearing brain parenchyma was seen on T2W images in a patient with clinical diagnosis of pseudohypoparathyroidism while extensive intracerebral calcifications were noted after additionally performed computerized tomography of the brain. In another patient with Fahr disease and neurologic symptoms, extensive calcifications were evident on CT, while MR examination had revealed bilateral symmetric lesions of prolonged T2W signal in the basal ganglia, supratentorial white matter and cerebellum, most compatible toxic/metabolic demyelination. In the third patient, a boy with hypercalcemia, marked left parietal cortical calcification was noted on CT, while MRI, including T2 gradient-echo sequence was inconclusive.

CONCLUSIONS: MRI, without CT, can be not only confusing, but even misleading diagnostic modality for detection of not only subtle, but also extensive cerebral calcifications. The benefit of gradient-echo T2 sequence, that is usually included in MR protocol when intrcranial calcifications are suspected, is also rather limited.

CASE REPORT

INTRODUCTION

Since magnetic resonance imaging (MRI) is method of choice in establishing the correct diagnosis in a great majority of neurologic disorders, especially in detection the causes of seizures and slowly progressive neurologic disturbances, computerized tomography (CT) is becoming more frequently excluded from obligate spectrum of diagnostic protocol. The purpose of this report is to show potential misinterpretation of brain calcifications on MRI if CT has not been performed previously since advantages of CT in detection of calcium deposits are significant.

PATIENTS

First patient

The upper and lower extremities spasms were identified for the first time in the girl at the age of 12, and have been repeating several times annually, mainly after exhausting physical activity. At the age of 24, the patient was hospitalized for muscle spasms associated with hypocalcemia and hyperphosphatemia. MR examination of the brain was indicated. No abnormalities were seen on T2-weighted images (Figure 1A), while slight bilateral symmetric T1W shortening was obvious within the basal ganglia (Figure 1B). CT

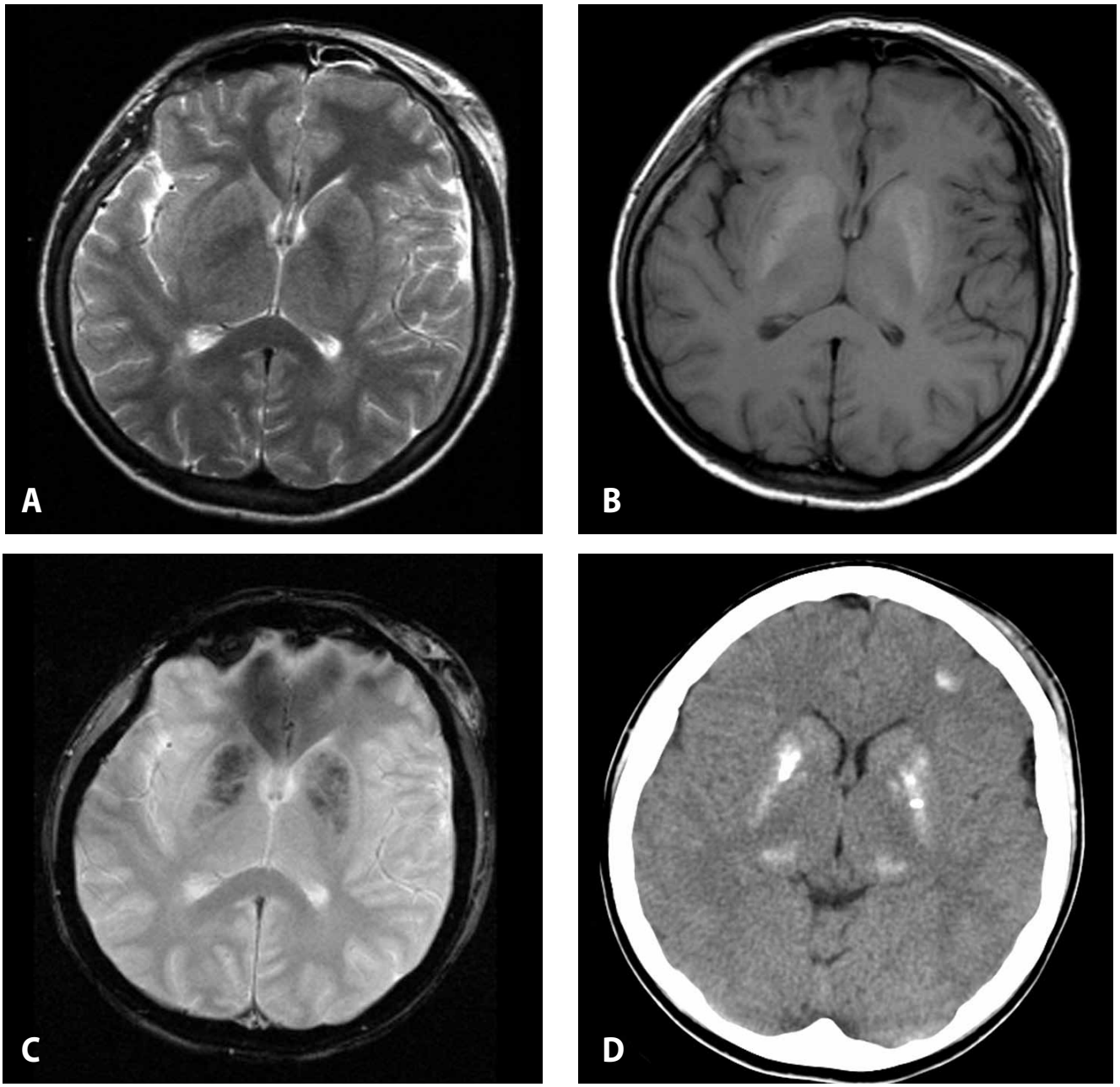

Fig. 1. Normal appearing brain parenchyma on T2W axial image in a 24-year-old patient with pseudohypoparathyroidism (**A**) associated with hyperintense signal in the basal ganglia on T1W sequence (**B**) and hypointense on GRE T2W sequence (**C**) while calcium depositions in basal ganglia, left subcortical region and thalami on corresponding CT image are evident (**D**). Note that thalamic calcifications are noted only on CT while all three MR sequences are completely inconclusive.

examination revealed extensive, relatively symmetric calcifications in the basal ganglia and subcortical white matter (Figure 1D). Thalamic calcifications were undetectable on all performed MR sequences, including gradient-echo T2-weighted images (the sequence sensitive for revealing the lesions with ferromagnetic properties, like calcifications and hemosiderin depositions) (Figure 1C). The diagnosis of pseudohypoparathyroidism has been established after extensive further endocrinologic evaluation.

Second patient

MR examination revealed bilateral symmetric lesions of prolonged T2W signal in the basal ganglia, supratentorial white matter and cerebellum in a 64-year-old patient with clinical signs of progressive dysarthria and

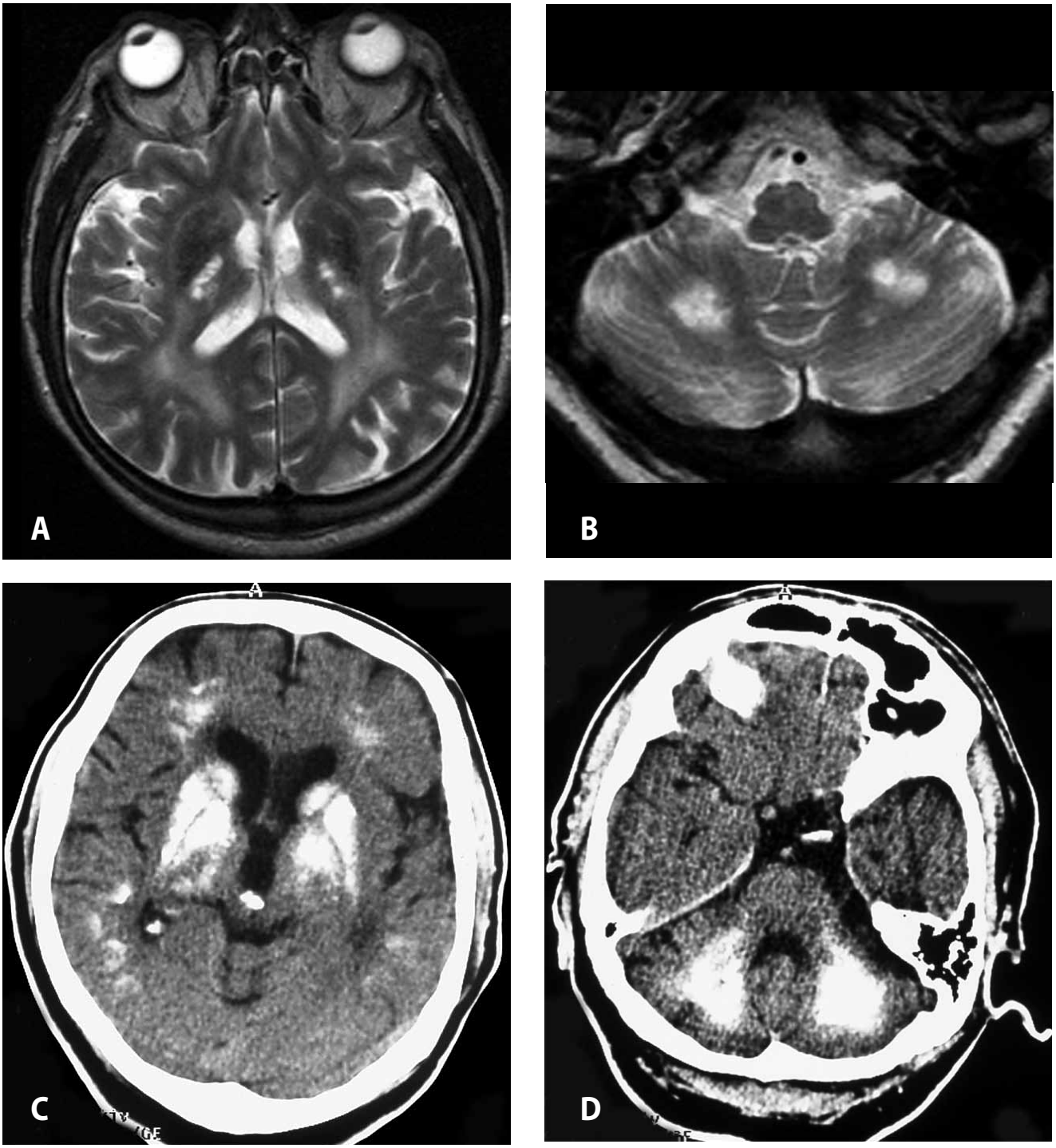

Fig. 2. Symmetric T2W hyperintense lesions in posterior crus of internal capsule (**A**) and cerebellum (**B**) in a 64-year-old male patient with Fahr disease presented with dysarthria and ataxia associated with much more extensive areas of calcifications on corresponding CT images (**C** and **D**). Underlying abnormalities on MR images appear to be responsible for neurological symptoms.

ataxia (Figure 2A, 2B). No marked abnormalities have been noted on routine blood study. The radiological interpretation of the lesions suggested toxic/metabolic demyelination. CT examination that was additionally performed six weeks later found extensive supratentorial and cerebellar calcifications (Figure 2C, 2D).

Third patient

Both MR and CT examination of the brain were performed in an 11-year-old boy after first seizure attack. Marked left parietal calcification was evident on CT, while MRI, including T2 gradient-echo sequence was inconclusive. Laboratory analyses showed elevation of serum ionized calcium – 2.2 mmol/L (normal reference range 1.03–1.23 mmol/L) with no other blood or biochemical abnormalities.

DISCUSSION

Intracranial calcifications, that are interpreted as "physiological" in the routine radiological and clinical practice are most commonly identified on CT examination in the pineal gland, habenular commissure, chorioid plexus, falx, tentorium and dentate nuclei. On high resolution CT scanners physiologic, usually symmetric calcifications are noted in the globi pallidi in more than 1% of asymptomatic adults, while dentate nuclei calcifications are much less frequently seen (Grossman, 1996; Burke *et al.*, 1988). Dystrophic calcifications in neoplastic disorders, infection (TORCH, cysticercosis, various parasitic and granulomatous infections, neurolues), vascular diseases (aneurismal, artherosclerotic, vascular malformations, infarcts), congenital disorders (Sturge-Weber disease, tuberous sclerosis, neurofibromatosis I) and endocrine diseases (hypoparathyroidism, pseudohypoparathyroidism, idiopathic, hypothyroidism) are clearly seen on CT examination. General opinion exists that intracerebral calcifications could be easily missed on MR examination, if T2 gradient-echo sequence (that is commonly not a part of routine protocol, except in patients with posttraumatic conditions) is not performed.

Even extensive calcifications could be completely invisible on conventional MR imaging, or may show increased or decreased T2W signal, depending on the chemical nature of the deposited calcium and potentially associated degenerative or demyelinating changes (Norenberg and Gregorios, 1985).

The presence of cerebral calcifications in pseudohypoparathyroidism has been more frequently seen than in idiopathic hypoparathyroidism and the exact mechanism of calcium depositions has not been completely explained (Mujdoubi and Zagermann, 2004; Fujita, 2004). In our first patient with pseudohypopyrythyroidism none of the extensive brain calcifications were identified on T2W images, while symmetric T2W prolongation was evident in the second patient with Fahr disease. On T1W images the presence of increased signal was identified in the basal ganglia. In several reports it has been suggested that in idiopathic basal ganglia calcifications, acid mucopolysaccharide usually accumulates and forms a non-calcified round body, associated with secondarily concentrated calcium and iron on the matrix of acid mucopolysacharide (Narita *et al.*, 2002; Adachi *et al.*, 1968). Fukunaga (1987) noted that patients with more prolonged hypocalcemia showed higher incidence of basal ganglia calcifications in idiopathic hypoparathyroidism. Symmetrical involvement of cortex, subcortical white matter, thalami and cerebellum may be additionally noted (Stelmasiak *et al.*, 2000; Friedman at al., 1987).

Observation that gradient-echo sequence was unable to detect calcium depositions, especially in the cortical/subcortical interface, but also in basal ganglia, detected in our patients, suggests that this sequence can not reliably substitute CT for his type of disorders. However, the benefit of MRI can be significant in patients with underlying T2W hyperintense signal since our two

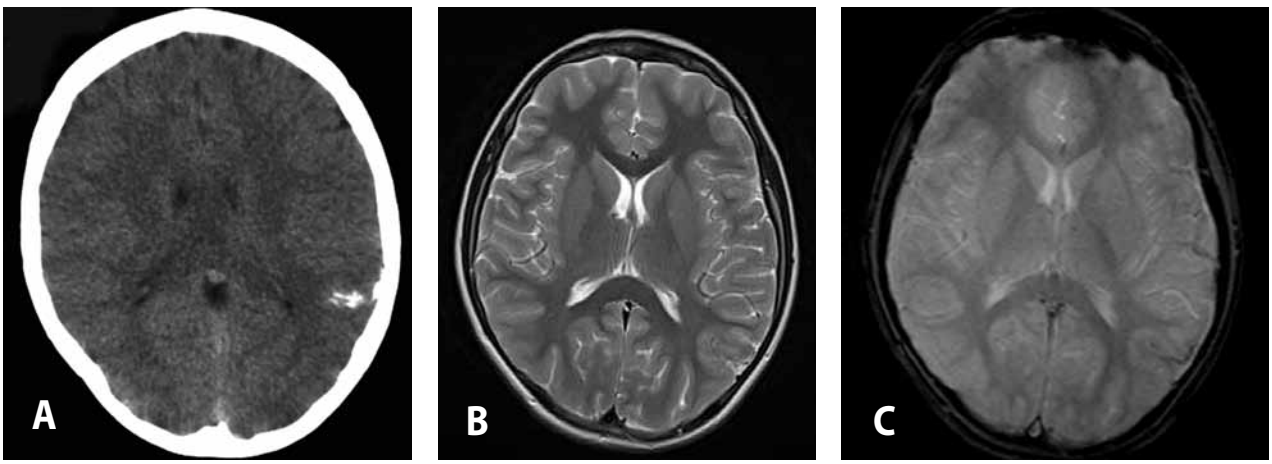

Fig. 3. Relatively extensive area of left parietal calcification on CT image in a 11-years-old boy with hypercalcemia and seizure attack (**A**) associated with completely inconclusive MR imaging – T2W sequence (**B**) and GRE T2W image (**C**)

patients (first and third) with intact brain parenchyma on T2W sequence showed no abnormalities on neurological examination while the patient with Fahr disease presented with severe dysarthria and ataxia.

CONCLUSION

MRI, without CT, can be not only useless, but also confusing and even misleading diagnostic modality for diagnosing intracranial "physiologic" and pathologic calcifications. The benefit of gradient-echo T2 sequence, that is usually included in MR protocol when intracranial calcifications are suspected, is also rather limited.

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