Characteristic MR findings in seizures associated with nonketotic hyperglycemia (NKH): diagnostic value of contrast enhanced FLAIR imaging (CE-FLAIR)

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Target audience

For anyone not familiar with contrast enhanced FLAIR imaging.

Purpose

Nonketotic hyperglycemia (NKH) have been reported to induce seizures, and may cause transient signal changes on magnetic resonance imaging (MRI)[1,2,3]. The aim of our study was to describe the characteristic MRI abnormalities in patients with NKH complicated with seizures and to evaluate the diagnostic value of contrast enhanced FLAIR imaging (CE-FLAIR). We also attempt to characterize the pathogenesis of this unusual finding.

Methods

We retrospectively studied MRI abnormalities in 10 patients with seizures associated with NKH. All patients underwent brain MRI within 2 days of seizure onset. The clinical manifestations, laboratory findings, MR findings, and clinical outcome in each patient were analyzed.

Results

All patients except one patient presented with focal seizures, either simple or complex partial seizures or negative motor seizures. All patients had long-standing uncontrolled diabetes mellitus. The MRI abnormalities observed acutely were focal subcortical hypointensities on T2WI and FLAIR imaging in all patients with overlying cortical gyral hyperintensities in 2 patients. Focal overlying cortical or leptomeningeal enhancement on CE-T1WI or CE-FLAIR was observed in all patients. And CE-FLAIR was superior to CE-T1WI for detecting characteristic leptomeningeal enhancement. Diffusion-weighted imaging showed mild restricted diffusion in 2 patients with cortical gyral T2 hyperintensities. The parietal and occipital lobes were most commonly involved. On clinical recovery, the subcortical T2 hypointensities, leptomeningeal enhancement and overlying cortical hyperintensities reversed.

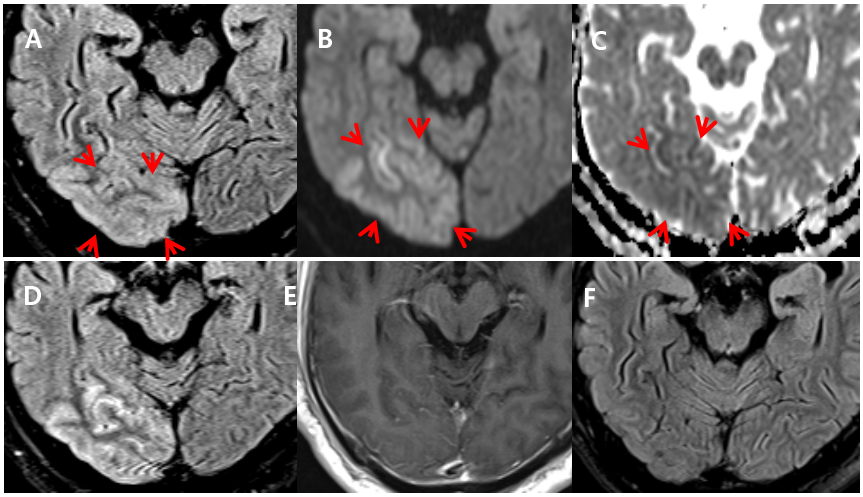

Fig 1. A 58 year old man with seizures associated with NKH. FLAIR image (A) show focal subcortical hypointensity in right occipitotemporal region with hyperintense cortical edema. DWI (B) and ADC maps (C) show restricted diffusion in the corresponding area. CE-FLAIR (D) images show diffuse cortical and leptomengeal enhancement while CE-T1 (E) image shows no significant enhancement. Follow up image (F) 252 days later shows remarkable resolution of the subcortial hypointense lesion and cortical hyperintensity.

Discussion

The possible mechanisms of focal subcortical T2 hypointensity is due to an accumulation of free radicals and iron deposition due to excitotoxic axonal damage during seizures. Cortical T2 hyperintense lesions reveal seizure-induced transient cytotoxic and vasogenic edema. Enhancement of the leptomeninges is supposed to occur due to seizure-induced dilatation of leptomeningeal vasculatures and cortical enhancement is believed to be the result of seizure-induced hypoxia and acidosis with alteration of vascular permeability and breakdown of the blood brain barrier, leading to contrast extravasation.

Conclusion

Patients with seizures in NKH may have transient MRI abnormalities that are characterized by subcortical T2 hypointensity with overlying cortical or leptomeningeal enhancement in additional to cortical T2 hyperintensity. CE-FLAIR is superior to CE-T1WI for detecting the breakdown of the blood-brain barrier (BBB) in these diseases. Although these MRI abnormalities pose a broad differential diagnosis, recognition of these radiologic abnormalities in NKH is important in restricting unwarranted investigations and to institute early therapy.

References

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