



Reversible periictal MRI abnormalities: Clinical correlates and long-term outcome in 12 patients

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Summary Although a wide spectrum of reversible periictal magnetic resonance imaging (MRI) abnormalities (RPMA) are being increasingly identified, the clinicians are often in a dilemma about their localization significance. This prompted us to analyze the clinical, MRI, electroencephalographic (EEG) and follow-up data of 12 patients with RPMA seen in a tertiary referral epilepsy center. RPMA occurred after a single or a cluster of focal seizures with or without secondary generalization. The interictal and ictal EEG abnormalities were localized to the site of RPMA in nine patients. RPMA involved areas remote from the site of EEG abnormalities in four patients. We have developed a comprehensive classification to account for the wide spectrum of RPMA involving gray matter, white matter and leptomeninges with or without contrast enhancement or mass effect. Follow-up MRIs showed complete resolution of RPMA in all, except in four patients, who developed residual focal atrophy. During median follow-up period of 3 years, recurrence of RPMA was observed in two patients. Diffusion weighted MRI in two patients and histopathological finding in one patient favored causal role of hypoxia in the pathogenesis of RPMA. Our observations help to understand the electroclinical profile, radiological spectrum, localization significance and natural history of RPMA better.

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Introduction

With recent advances in magnetic resonance imaging (MRI) technology, a wide spectrum of focal brain abnormalities are

being increasingly identified immediately following a single or a cluster of seizures that resolve within the next few days or weeks of the ictus (Cole, 2004; Briellmann et al., 2005). These reversible periictal MRI abnormalities (RPMA) are believed to be the consequence of the epileptic activity rather than its cause. A majority of reports on RPMA are based on single (Riela et al., 1991; Stübgen, 1995; Senn et al., 2003) or small series (Yaffe et al., 1995; Meierkord et al., 1997; Silverstein and Alexander, 1998; Lansberg et al.,

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Table 1 Classification of periictal MRI abnormalities and their distribution among 12 patients

Classification	Sites of involvement	Patient no.
Type I	Superficial gray matter	2, 9, 10, 12
Type IIa	Superficial gray matter and subcortical white matter	3, 5, 6, 8
Type IIb	Superficial gray matter and white matter involvement extending up to the adjacent periventricular surface	1, 6
Type III	Involvement of the deep gray matter nuclei (thalamus) with any degree of involvement of the cerebral cortex and the white matter	4, 6
Type IV	Involvement of white matter alone	7, 11
Type V	Leptomeningeal enhancement and/or gyriform cortical enhancement	1, 2, 3, 5, 6, 8, 12

1999; Kim et al., 2001; Polster et al., 2001) of patients, and lack uniform diagnostic criteria, clinical correlation and adequate follow-up information. The clinicians are often in a dilemma about the significance of RPMA and their localization value in relation to epileptogenesis. This prompted us to carefully analyze and report the clinical, radiological, electroencephalographic (EEG) and follow-up data of 12 patients with RPMA. Our objectives are four-fold: to describe the radiological spectrum of RPMA, to characterize their evolution and resolution, to delineate their clinical and EEG correlates and to assess their localization value in relation to epileptogenesis.

Materials and methods

Study site and subjects

This study was carried out in the Departments of Neurology and Imaging Sciences at the Sree Chitra Tirunal Institute for Medical Sciences and Technology, a tertiary referral center at Trivandrum, Kerala, South India. Between January 1999 and December 2005, 1700 patients were hospitalized for evaluation of epileptic seizure disorders. Among them, we identified 12 patients, who had focal MRI abnormalities following a single or a cluster of seizures that resolved completely, except for occasional residual focal brain atrophy, on follow-up MRI. We excluded patients with preceding history of classical migraine with or without seizures, and those with symptomatic seizures due to an underlying specific etiology.

Clinical data collection

Utilizing a structured proforma, we abstracted the clinical and investigative data from the patients' medical records. We contacted all 12 patients for a detailed re-evaluation of their seizure history. Epileptic seizures were classified according to International League Against Epilepsy (ILAE) classification (Commission, 1981). The seizure(s) for which imaging was carried out was designated as 'index seizure'. The EEGs were performed based on a standard protocol using a 16-channel digital EEG acquisition system with the electrodes placed according to the international 10–20 systems (Radhakrishnan et al., 1999). To classify EEG findings, we used the definitions of the Committee on Terminology of the International Federation of Societies for

EEG and Clinical Neurophysiology (IFSECN) (Chatrian et al., 1974).

Review of imaging findings and classification

The MRI studies, performed on a 1.5T MRI scanner (Signa GE, Milwaukee, USA), were independently reviewed by both the radiologists (TK and CK) involved in this study. The T1 weighted (T1W), T2 weighted (T2W), proton density (PD), fluid attenuation recovery (FLAIR) sequences and intravenous gadolinium (0.1 mmol/kg) enhanced studies were available in all cases. The imaging abnormalities were referred to as 'local', when it was confined to the region of the epileptiform discharges on EEG or 'remote', when the changes were located in distant areas. We classified the RPMA into five types based on the extent of anatomic involvement and the presence/absence of contrast enhancement (Table 1).

Results

The demographic and clinical features, and the EEG and MRI data are summarized in Tables 2 and 3, respectively.

Demographic characteristics

Twelve patients (nine males, three females) with RPMA comprised 0.007% of the 1700 patients hospitalized during the study period for evaluation of seizure disorders. Their age at presentation ranged from 1.5 to 47 years (mean 20 ± 15 years, median 21 years). The age at seizure onset ranged from 1.5 to 47 years (mean 16.7 ± 12.8 years, median 17 years).

Seizure characteristics

Eight patients had past history of seizures for a median duration of 13 months. The remaining four patients presented with the index seizure, and had recurrent seizures on follow-up, except Patient 8, who had only a single seizure (Table 2). The index seizures were partial in all patients (simple partial in nine and complex partial in three), with secondary generalization in eight (66.6%) of them. The index seizure frequency ranged from a single seizure to status epilepticus. Clustering of seizures was noted in 10 out of 12 (83.5%) patients. The individual seizures were either brief

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