Bratisl Lek Listy 2010; 111 (8)
467 – 468

CASE REPORT

A case with an asymptomatic malformation of cortical development diagnosed in eighth decade of life

Irsel Tezer-Filik F[1], Kader K Oguz[2], Saygi S[3]

Institue of Neurological Sciences and Psychiatry, Hacettepe University School of Medicine, Ankara, Turkey. **irseltezer@yahoo.com.tr**

Abstract: We report a patient newly diagnosed with cortical dysplasia upon magnetic resonance imaging (MRI) in his eighth decade of life after a recent syncopal attack. The neurological examination and laboratory findings were normal. His cranial MRI revealed a lesion giving a low signal on T1-weighted images, which was evaluated as focal cortical dysplasia. No treatment was given, and he did not have any further syncopal or epileptic attacks during the subsequent two-year follow up. No previous study has described such a case of malformations of cortical development (MCD) in patients older than 70. Especially in asymptomatic or clinically less severe patients, the underdiagnosis of MCD may result in a clinical spectrum that is too narrow to reflect the reality *(Fig. 1, Ref. 6)*.Full Text in free PDF *www.bmj.sk.*
Key words: cortical dysplasia, adult onset, MRI, asymptomatic, neoplasm.

Malformations of cortical development (MCD) are frequent and important causes of epilepsy in childhood and adulthood (1) but their clinical manifestations vary depending on type, location, and extent of MCD.

Until the introduction of new imaging techniques, in particular the magnetic resonance imaging (MRI), these disorders were almost exclusively the domain of pathologists. MRI has provided tremendous insights into abnormal cortical lesions and facilitated the diagnosis of MCD (1, 2). Herein, we report an elderly patient newly diagnosed with MCD upon MRI.

Case

A 74-year-old male presented with a syncopal attack. His past medical and family histories were unremarkable. The neurological examination and laboratory findings were normal. His electroencephalography was normal. His electrocardiography and echocardiography had no abnormal findings potentially leading to syncopal attack. In addition, his magnetic resonance angiography did not show any abnormality either. His cranial MRI revealed a tumoral mass lesion in the left frontal cortex but magnetic resonance spectroscopy did not show any such lesion. Therefore, the lesion giving a low signal on T1-weighted images was evaluated as focal cortical dysplasia (Fig. 1).We had no pathological diagnosis because the patient did not accept biopsy. No treatment was given, and he did not have any new syncopal or epileptic attacks during the subsequent two years of follow up.

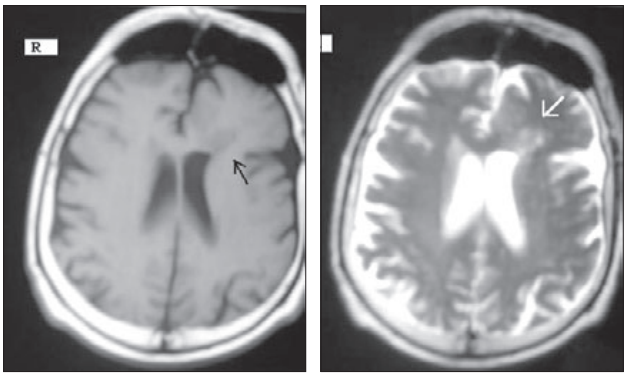

Fig. 1. Axial T1(a) and T2(b)-weighted MRI of patient with focal cortical dysplasia in the left frontal cortex.

Discussion

To the best of our knowledge, no cases of newly diagnosed asymptomatic MCD in the eighth decade of life have been reported. Patients with epilepsy, which is a common complication of MCD, were reported in their fifth and sixth decades (3-5). Our patient had only one syncopal attack before the diagnosis of MCD based on his cranial MRI. This attack was probably not related to the found cortical lesion. Some patients with cortical dysplasia may be apparently healthy individuals with no history of epilepsy. Histological postmortem studies have similarly de-

[1]Institue of Neurological Sciences and Psychiatry, Hacettepe University School of Medicine, Ankara, Turkey, [2]Department of Radiology, Hacettepe University School of Medicine, Ankara, Turkey, and [3]Department of Neurology, Hacettepe University School of Medicine, Ankara, Turkey

Address for correspondence: F. Irsel Tezer-Filik, MD, Ziya Gokalp Cad. No: 72-14, Ankara, Turkey.
Phone: +90.312.30511 82, Fax: +90.312.3093451

monstrated gross cortical dysgenesis in approximately 2 % of population (6). Periventricular nodular heterotopia (PNH) was also identified upon MRI in healthy individuals before the onset of epilepsy or other manifestations (4). Our patient had no PNH on MRI. He had a localized area of cortical dysgenesis that was mostly responsible for intractable epilepsy (4). However, we were unable to show that there might really be a PNH with pathological involvement of focal cortical dysplasia in later years.

These developmental cortical abnormalities, particularly the neuronal migration abnormalities most commonly follow the normal MR signal intensity characteristics of gray matter on all pulse sequences, (2). Whenever a signal of such a parenchymal lesion varies from that of normal gray and white matter, the primary differential considerations should be focused on glial neoplasm or hamartomatous lesion. The appearance of most neoplasms such as low-grade neoplasms and infiltrative astrocytomas can be quite similar to that of MCD. They occur most often in children and young adults. Patients usually present with partial seizures that are difficult to control with anticonvulsant drugs (1). The combinations of different MR pulse sequences such as FLAIR images, T2-weighted spin echo and gradient echo volume acquisitions, and rarely T1-weighted spin echo studies in addition to MR spectroscopy (2) make it easy to distinguish the heterotopic tissue from mass lesions as in our patient. Furthermore, in contrast to patients with mass lesions, our patient had mild clinical symptoms with no progression.

Advanced neuroimaging techniques with involvement of special sequences on MR imaging have increased the diagnosis of MCD. Especially in clinically less severe patients and asymptomatic individuals as was the presented case, the underdiagosis of MCD may result in a clinical spectrum that is too narrow to reflect the reality.

References

1. Fish DR. Localized cortical dysgenesis and epilepsy. In: Kotagal P, Lüders HO (Eds). The epilepsies Etiologies and Prevention. London, Academic Press, 1999: 45–62.

2. Ruggieri PM. MRI Techniques for the identification of neuronal migration disorders: Practical considerations. In: Kotagal P, Lüders HO (Eds). The Epilepsies: Etiologies and Prevention. London, Academic Pres, 1999: 19–27.

3. Rademacher J, Aulich A, Reifenberger G et al. Focal cortical dysplasia of the temporal lobe with late onset partial epilepsy; serial quantitative MRI. Neuroradiology 2000; 42: 430–435.

4. Tinuper P, d'Orsi G, Bisulli F et al. Malformation of cortical development in adult patients. Epileptic Disord 2003; 5 (Suppl 2): 85–90.

5. Imamura S, Nagado T, Toyomitsu Y et al. Late onset epilepsy associated with migration disorder:a case report. No To Shinkei 2006; 58 (2): 151–154.

6. Meencke HJ, Veith G. Migration disturbances in epilepsy. Epilepsy Res 1992; Suppl 9: 31–40.

Received November 3, 2008.
Accepted May 3, 2010.