Brain Magnetic Resonance Imaging Findings and Distribution of the Findings According to the Age Groups in Childhood Epilepsies

SK Balık¹, M Öztürk², C Göya³, F Ekici³

ABSTRACT

Objective: To retrospectively analyse the magnetic resonance imaging (MRI) observations and the distribution of etiological factors according to age groups in cases with MRI examinations for an epilepsy diagnosis.

Methods: The brain MRI of 606 cases from the 0–17 years age group were analysed retrospectively, and the findings were evaluated according to the age group.

Results: In 274 cases (45.2%) at least one lesion from different pathology groups was observed. The most frequently observed pathologies were parenchymal damage, hippocampal sclerosis and cortical developmental anomalies.

Conclusion: An MRI is a non-invasive, irradiation-free imaging method that can be used in the follow-up of epileptic patients to detect underlying pathologies and treatable causes of epilepsy.

Keywords: Children, epilepsy, magnetic resonance imaging.

INTRODUCTION

A seizure is a paroxysmal attack caused by a cerebral neuron, abnormal and excessive release of neurotransmitters and is symbolized by a sudden modification in sensory-motor functions, behaviour, memory or consciousness. Epilepsy may be defined as the tendency of the brain to create epileptic seizures, and this situation may have conscious, psychological and social results and may be defined as a clinical pattern (1). Seizures may be observed at a rate of 3%-5% during childhood. While epilepsy occurs at 0.5%–1% in the general population, it starts during childhood in 60% of patients (2). Epilepsy is considered a genetic or acquired disease and is believed to be multifactorial (3). In various studies and in 30%-35% of cases, the original aetiology has been determined (4). The determination of the aetiology is important in the planning of the treatment.

Imaging methods are required in patients believed to be epileptic as a result of physical and neurological examinations and laboratory analyses for epileptic patient evaluations (5). Magnetic resonance imagings are performed more than other methods for the evaluation of epileptic patients in which the diseases are associated with cerebral structural impairments. Magnetic resonance imagings hold the most important place among radiological imaging methods for the evaluation of epileptic patients due to their high resolution of soft tissue and multi-planar imaging capacity. There are few extended MRI studies concerning imaging method epilepsies and the distribution of etiological factors according to age.

The aim of our study was to diagnose early MRI observations and treatable causes of epilepsy and to determine the etiological factors according to age group.

SUBJECTS AND METHODS

This retrospective study was carried out following the approval of the local institutional review board. Adherence to principles announced in the Declaration of Helsinki was observed. The study included 606 cases

From: ¹Department of Radiology, Diyarbakır Children's Hospital, 21100 Diyarbakır, Turkey, ²Department of Radiology, Medical Faculty, Selcuk University, 42200 Konya, Turkey and ³Department of Radiology, Dicle University School of Medicine, 21280 Diyarbakır, Turkey.

Correspondence: Dr M Öztürk, Department of Radiology, Medical Faculty, Selcuk University, 42200 Konya, Turkey.

DOI: 10.7727/wimj.2017.175

Email: drmehmet2121@gmail.com

between 0 and 17 years of age who were submitted to brain MRI examinations and diagnosed as having epilepsy in the Paediatric Neurology Department of our hospital between June 2008 and August 2012. The patients who underwent operations and were non-conforming for imaging procedures due to movement artefacts were not included in the study.

Brain MRI observations were performed using 1.5 and 3.0-T (Achieva; Philips Medical Systems, Best, the Netherlands) devices and head coils. All observations were performed according to the epilepsy protocol. In Dicle University, Medical Faculty Hospital, Diyarbakir, Turkey, the MRI protocol used as a routine in 1.5-T and 3.0-T MRI devices presents the T2-weighted fast spin-echo sequence (FSE) in the axial and coronal planes, sequence fluid-attenuated inversion recovery (FLAIR), and non-contrast T1-weighted 3D turbo field echo sequences. In examinations performed in the 1.0-T MRI device, T1-weighted spin echo (SE), T2-weighted FSE, proton-weighted FSE in the coronal plane, and T1-weighted SE sequences in the sagittal plane were used. According to the pathology determined during the examination, 0.1 mmol/kg of an intravenous paramagnetic contrast substance (gadodiamide [Omniscan; Amersham Health, Cork, Ireland], gadopentetate dimeglumine [Magnevist; Schering AG, Berlin, Germany] and gadobutrol [Gadovist; Schering AG, Berlin, Germany]) was administered at 2 ml/sec via the antecubital vein and the SE sequence T1-weighted axial, coronal and sagittal plane images were obtained. To reduce movement artefacts in children, chloral hydrate (50 mg/kg) was given orally to help the patient sleep. No sedatives were used in older children who cooperated. Two radiologists with 10 and 3 years of experience of neuroradiology reviewed the MRI images in consensus and blind to diagnosis.

Statistical analyses

The computer program SPSS 22.0 for Windows (IBM Corporation, Armonk, New York, NY, USA) was used to perform the statistical analysis (descriptive study).

RESULTS

A total of 606 cases including 369 boys (60.9%) and 237 girls (39.1%) from the 0–17 years age group (mean age: 7.4 years) were included in this study. In our study, there were 79 cases in the infant age group (13.2%), 358 cases in the children age group (59%), and 169 cases in the adolescent age group (27.8%). No lesions were detected in the brain MRIs of 332 cases (54.8%). In 274 cases (45.2%), at least one lesion from different pathology groups was observed. The distributions of the lesions detected in the MRIs according to age and the pathology groups are given in Table 1.

When we classified the cases of our study according to age groups (Table 2), the most important proportion of pathologic findings was for the infant group and the most frequent pathology encountered in all groups was parenchymal damage with 118 patients (43%) (Fig. 1). The MRI observations detected in these cases were encephalomalacia and gliosis development in the prenatal-perinatal-post-natal periods due to ischemic, toxic, infectious, inflammatory and traumatic causes.

The second largest group was the cortical developmental anomalies group with 34 (12.5%) cases in our study (Fig. 3). The pathologies we determined were cortical dysplasia (n = 10), heterotopia (n = 8), polimicrogiria (n = 7), corpus callosum agenesis/dysgenesis (n = 4), holoprosencephaly (n = 2), hemimegalencephaly (n = 1), schizencephaly (n = 1) and Aicardi syndrome (n = 1). The most frequently observed pathologies in this group were cortical dysplasia, heterotopia, and

| Table 1: Distribution of lesions detected in MRI | according to the pathology groups |
|--|-----------------------------------|
|--|-----------------------------------|

| Pathology Groups detected in MRI | Infants (n = 42) | Children (n = 160) | Adolescents (n = 72) | Number of cases | Percentage of cases |
|----------------------------------|---------------------|-----------------------|-------------------------|-----------------|---------------------|
| Parenchymal damage | 22 | 64 | 32 | 118 | 43 |
| Hippocampal sclerosis | 2 | 18 | 12 | 32 | 11.7 |
| Cortical developmental anomalies | 5 | 22 | 7 | 34 | 12.5 |
| Cerebral atrophy | 4 | 16 | 6 | 26 | 9.5 |
| Tumour/cyst | 1 | 17 | 10 | 28 | 10.3 |
| Neurocutaneous syndromes | 1 | 8 | 1 | 10 | 2.7 |
| Myelinization disorder | 4 | 4 | 1 | 9 | 3.3 |
| Vascular anomalies | 2 | 5 | 1 | 8 | 2.9 |
| Metabolic-degenerative diseases | 1 | 6 | 2 | 9 | 3.3 |

MRI = magnetic resonance imaging.

Balik et al 65

Table 2: Number of cases and proportion of pathologic findings according to the age group

| | Total number of cases | Cases with no pathology | Cases with pathology | Parenchymal damage |
|--------------------------|-----------------------|-------------------------|----------------------|--------------------|
| Infant (1–12 months) | 79 (13.2%) | 37 (47%) | 42 (53%) | 22 (52.3%) |
| Children (1–10 year) | 358 (59%) | 198 (55.5%) | 160 (44.5%) | 64 (40%) |
| Adolescent (10–17 years) | 169 (27.8%) | 97 (57.4%) | 72 (42.6%) | 32 (44.4%) |

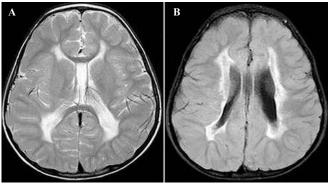


Fig. 1: Leukomalacia/gliosis with ventricular enlargement and volume loss at transverse T2-weighted fast spin-echo MR (A) and transverse fast fluid-attenuated inversion recovery (FLAIR) MR images (B). MR = magnetic resonance.

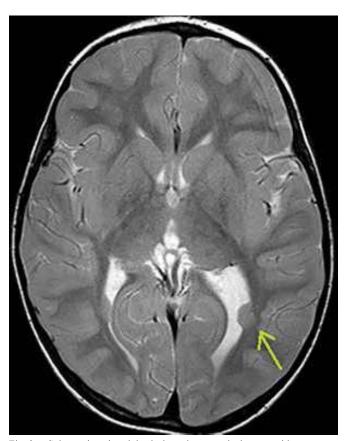


Fig. 3: Subependymal nodular lesions that were isointense with grey matter extending into the lateral ventricles (arrow) at a transverse T2-weighted fast spin-echo MR image (subependymal heterotopia). MR = magnetic resonance.

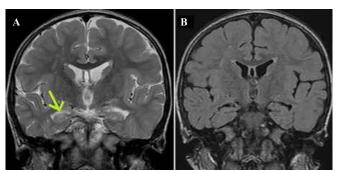


Fig. 2: Right hippocampal signal abnormality and volume loss in a 9-yearold boy. Increased signal with a volume loss in the right hippocampus at coronal T2-weighted fast spin-echo MR (A) and a volume loss at coronal fast fluid-attenuated inversion recovery (FLAIR) (arrow) MR images (B). MR = magnetic resonance.

polimicrogiria. Congenital bilateral perisylvian syndrome was associated with five cases of polimicrogiria.

The other largest group was the hippocampal sclerosis group with 32 cases (11.7%) (Fig. 2). Of these cases, we determined bilateral hippocampal sclerosis in 10 cases, left hippocampal sclerosis in 12 cases and right hippocampal sclerosis in 17 cases.

In 26 of our patients (9.5%), we observed increases in the depth and width of the cerebral fissure and sulcus. These were associated with diffuse cerebral atrophy and enlargements at various levels of the ventricular system.

In our study, arachnoid cysts were detected in 16 cases. Arachnoid cysts were present in the left temporal fossa (n = 6), retrocerebellar region (n = 5) and in the right temporal fossa (n = 2). Moreover, there were choroid fissure cysts in three patients. In 12 cases in which we detected tumour lesions (4.01%), hamartom (n = 5), low-grade glial tumour (n = 3) and dysembryoplastic neuroepithelial tumour (DNET) (n = 4) were present. In the cases with low-grade glial tumour, masses were present in the left temporal lobe in one patient, and in the left globus pallidus in the other. In two of the four DNET cases detected, the lesions were localized in the left temporal gyrus and in the left median temporal lobe. In a case believed to be DNET, masses were present in the right temporal lobe, and in another case, cortical masses were present in the right parietal parafalcine.

We determined there were eight patients in the neurocutaneous syndrome group (2.7%). Tuberosclerosis

(n = 5), neurofibromatosis (n = 3) and Sturge-Weber syndrome (n = 2) were observed in the neurocutaneous group.

In our study, myelination defects were observed in eight patients (3.3%). Furthermore, regions with modifications in hyper-intense signals in the white matter in T2 imaging, considered as dysmyelination, were observed.

There were nine cases (3.3%) in the metabolic-degenerative group. Three of these patients presented with Van der Knapp disease, while the other two presented with Leigh disease, four cases glutaric aciduria type 2. We determined that there were eight patients (2.9%) in the vascular anomaly group. Sinus vein thrombosis (n = 2), venous angioma (n = 2), cavernous angioma (n = 2) and arteriovenous malformation (AVM) (n = 2) were observed in this group.

DISCUSSION

In 45.2% of the epilepsy cases during childhood, we detected pathologies using MRIs. The most frequently determined pathologies were parenchymal damage, hippocampal sclerosis and cortical developmental anomalies. Cerebral atrophy, tumours/cysts, myelinization defects, vascular anomalies, neurocutaneous syndromes and metabolic-degenerative disorders were also detected. When considered according to age groups, the most important proportion of pathologic findings was observed in the infant group. The most frequent pathology encountered in all groups was parenchymal damage.

Early diagnosis and treatment are very important in the control of seizures during childhood and also for the protection of neuronal damage due to repetitive seizures, normal development, reduction of drug side effects and increasing the quality of life (6). Imaging methods are not needed in the idiopathic epilepsy group. The diagnosis is established according to the seizure type and the electroencephalography observations. There is no structural damage in the brain of such patients (7, 8). In the symptomatic epilepsy group, there is structural damage that leads to frequent seizures. In patients with symptomatic seizures, imaging methods, especially MRIs, are required (5).

One of the important causes of neurological damage developed during the pre-perinatal period is hypoxic-ischemic events (9). The maturation state of the brain during the period of brain damage development is related to the intensity and duration of the event. Hypoxic-ischemic encephalopathy developing in early pregnancy before the 20th week leads to periventricular white matter damage [periventricular leukomalacia]

(10). Hypoxic-ischemic event rates during childhood epilepsy vary between 8% and 23% (11–13). In our study, this rate was 19.7%, which is consistent with the results reported by previous studies.

Hippocampal sclerosis is the most frequently observed pathologic lesion in patients with temporal lobe epilepsy and is the cause of 60%–80% of complex partial seizures (14). The surgical success rate in patients with hippocampal sclerosis is relatively high; thus, a pre-operative diagnosis is very important. The most reliable qualitative MRI observations in a FLAIR sequence in hippocampal sclerosis are atrophy and signal changes in the hippocampus (15, 16). The hippocampal sclerosis rate in epilepsy cases in the children's group varies between 50% and 70% (17–20). In a similar study by Kalnin *et al* (11), this rate was 14.9%. In our study, this rate was 11.7%. The differences in the rates may be associated with differences in age groups and patient populations.

Cortical developmental anomalies may be defined as the most frequent epileptogenic lesion in chronic extratemporal epilepsies. An MRI is a valuable method in the diagnosis of cortical developmental malformations and allows for the determination of 50%-70% of the anomalies (21). Kalnin et al (11) studied childhood epilepsy and reported a cortical development anomaly rate (12%) similar to our results. Cerebral/cerebellar atrophy is characterized by non-recoverable brain tissue loss, is associated with enlargements in regions with intra- and extracerebral cerebrospinal fluid (CSF) and has many causes including metabolic, demyelinating, degenerative and cerebrovascular diseases (22). The cerebral/cerebellar atrophy rate for childhood epilepsies is reported to be between 10% and 19% (11, 13). In our study, this rate was similar at 9.5%.

The brain tumour rate in the general epilepsy population is 2%–4%, and the sensitivity of MRI use for their detection is about 100%. The tumour lesions leading to epilepsy are usually close to the cortex and temporal lobe. Low-grade lesions such as low-grade astrocytoma, oligodendroglioma, ganglioglioma, ganglioneuroma, DNET and mixed gliomas have been detected in one-third of the children submitted to temporal lobectomy due to epilepsy (23, 24). The brain tumour rate in the children's epilepsy group is 3.2%–4.6% (25, 26). In our study, this rate was 4%, which confirms the literature data. Arachnoid cysts are arachnoid membrane congenital lesions with CSF secretion. They constitute 1% of all intracranial masses (27).

Balik et al 67

In a similar study by Amirsalari *et al* (13), the cyst determination rate (5%) was similar to our value. The development of normal myelin may be monitored by conventional MRI. Ischemia, infection, myelin damage associated with toxins (demyelination) and modifications related to the construction of damaged myelin (dysmyelination) may be easily detected by MRI, but these two situations may not be differentiated. Cerebral MRI examinations present a high sensitivity for complex neuronal development disorders and neurodegenerative diseases; unfortunately, they also have low specificity (28). In our study, myelination disorders were present in 3.3% of cases and there is no data about the rate of myelinization disorders in the literature.

One of the etiological groups causing epilepsy is vascular anomalies. Presentation types include seizures, progressive neurological deficits, restless headaches, intracranial haemorrhages and hydrocephaly (29). Extensive studies concerning the imaging of epileptic diseases (26, 30) have reported that they have roles in 2%–5% of cases, but this rate is expected to be lower during childhood. In our study, this rate was 2.9%.

Metabolic and neurodegenerative diseases may affect grey and white matter, or both. Magnetic resonance imaging observations are mostly non-specific. A primary cortical block leads to epileptic seizures and mental destruction. In our study, the rate of metabolic-degenerative diseases was 3.3%, and there is no data concerning the rate of metabolic-degenerative diseases in the literature.

Dura'-Trave' et al (12) reported that the pathology detection rate with MRIs is higher in the infant group than in both the children and adolescent groups. In our study, the rate of MRI pathology detection was higher in the infant group. The most frequent pathology determined in all age groups was parenchymal damage, similar to the study by Dura'-Trave' et al (12).

CONCLUSION

Magnetic resonance imaging is a non-invasive imaging modality that does not contain any ionizing radiation and may be used to determine pathologies leading to epileptic attacks during childhood. The most frequent pathologies in all age groups were signs of parenchymal damage. Other common pathologies were hippocampal sclerosis and cortical developmental anomalies.

AUTHORS' NOTE

SKB Ferguson conceived the paper, oversaw data collection, conducted data analysis, wrote the manuscript and approved the final version. MÖ Tulloch-Reid participated in study design, data analysis and interpretation, critically revised manuscript and approved the final version. CG Younger participated in study design, data analysis and interpretation of data and revision of the manuscript and approved the final version. FE Wright-Pascoe participated in study design, interpretation of data and revision of the manuscript and approved the final version. MÖ Boyne participated in study design and interpretation of data, critically revised the manuscript and approved the final version. SKB Soyibo participated in study design and interpretation of data, critically revised the manuscript and approved the final version. CG Wilks provided oversight to the study, participated in data interpretation and revision of the manuscript, and approved the final version. The authors declare that they have no conflicts of interest.

REFERENCES

- Fisher RS, van Emde Boas W, Blume W, Elger C, Genton P, Lee P et al. Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). Epilepsia 2005; 46: 470–2.
- Behrman RE, Kliegman RM, Jenson HB. Nelson textbook of pediatrics. In: Haslem RHA, ed. The nervous system: seizures in childhood. 16th ed. Philadelphia, PA: W.B. Saunders Company; 2000; 1813–4.
- Engel J. Seizures and epilepsy. Philadelphia, PA: F.A. Davis company; 1989.
- Holmes GL. Epilepsy and other seizure disorders. In Berg BO, ed. Principles of child neurology. New York, NY: McGraw-Hill; 1996; 223–84.
- Jack Jr CR. Epilepsy: surgery and imaging. Radiology 1993; 189: 635–46
- van Empelen R, Jennekens-Schinkel A, van Rijen PC, Helders PJ, van Nieuwenhuizen O. Health-related quality of life and self-perceived competence of children assessed before and up to two years after epilepsy surgery. Epilepsia 2005; 46: 258–71.
- Buchalter JR. Inherited epilepsies of childhood. J Child Neurol 1994; 9: 12–9.
- Feinstein A, Ron M, Wessely S: Disappering brain lesions, psychosis and epilepsy: a report of two cases. J Neurol Neurosurg Psychiatr 1990; 53: 244–6.
- Volpe JJ. 4th ed. Neurology of the newborn. Philadelphia, PA: W.B. Saunders; 2001.
- Barkovich AJ, Truwit CL. Brain damage from perinatal asphyxia: correlation of MR findings with gestational age. Am J Neuroradiol 1990; 11: 1087–96.
- 11. Kalnin AJ, Fastenau PS, deGrauw TJ, Musick BS, Perkins SM, Johnson CS et al. Magnetic resonance imaging findings in children with a first recognized seizure. Pediatr Neurol 2008; **39:** 404–14.
- Durá-Travé T, Yoldi-Petri ME, Esparza-Estaún J, Gallinas-Victoriano F, Aguilera-Albesa S, Sagastibelza-Zabaleta A. Magnetic resonance imaging abnormalities in children with epilepsy. Euro J Neurol 2012; 19: 1053-9
- 13. Amirsalari S, Saburi A, Hadi R, Torkaman M, Beiraghdar F, Afsharpayman S et al. Magnetic resonance imaging findings in epileptic children and its relation to clinical and demographic findings. Acta Medica Iranica 2012; **50:** 37–42.
- Pıtkanen A, Tuunanen J, Kalviainen R, Partanen K, Salmenpera T. Amygdala damage in experimental and human hippocampal epilepsy. Epilepsy research 2000; 39: 121–5.

- Bronen R. MR of mesial temporal sclerosis: how much is enough? Am J Neuroradiol 1998; 19: 15–8.
- De Coene B, Hajnal JV, Gatehouse P, Longmore DB, White SJ, Oatridge A et al. MR of the brain using FLAIR pulse sequence. Am J Neuroradiol 1992; 13: 1555–64.
- 17. Bronen RA. Epilepsy: The rol of MR Imaging. AJR 1992; 159: 1165-74.
- Brooks BS, King DW, el Gammal T, Meador K, Yaghmai F, Gay JN et al. MRI in patients with intractable complex partial epileptic seizures. AJNR 1990: 154: 577–83.
- Boon PA, Williamson PD, Fried I, Spencer DD, Novelly RA, Spencer SS et al. Inracranial, intraaxial, space occupying lesions in patient with intractable partial seizures: an anatomoclinical, neuropsychological and surgical correlation. Epilepsia 1991; 32: 467–76.
- Duncan JS, Sagar HJ. Seizure characteristics, pathology and outcome after lobectomy, Neurology 1987; 37: 405–9.
- Whiting S, Duchowny M. Clinical spectrum of cortical dysplasia in childhood: diagnosis and treatment issues. J Child Neurol 1999; 14: 759–71.
- Dahnert W. Radiology Review Manual. 3rd ed. Baltimore, MD: Williams & Wilkins; 1993.
- Spencer DD, Spencer SS, Mattson RH, Williamson PD. Intracerebral masses in patients with intractable partial epilepsy. Neurology 1984; 34: 432–6.
- Otsubo H, Chuang SH, Hwang PA, Gilday D, Hoffman HJ. Neuroimaging for investigation of seizures in children. Pediatr Neurosurg 1992; 18: 105–16.

- Berg AT, Testa FM, Levy SR, Shinnar S. Neuroimaging in children with newly diagnosed epilepsy: a community-based study. Pediatrics 2000; 106: 527–32.
- Hauser WA. Seizure disorders: The changes with age. Epilepsia 1992;
 33: 6–14.
- Caruso PA, Robertson R, Setty B, Grant E. Disorders of brain development. In: Atlas SW ed. Magnetic resonance imaging of the brain and spine. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009; 224–5.
- Osborn AG, Tong KA. Handbook of neuroradiology: Brain and Skull. 2nd ed. St Louis, MO: Mosby, 1996.
- Wojak JC. Intacranial arteiovenous malformations: general considerations. In: Connors JJ, Wojak JC, eds. Interventional neuroradiology. Philadelphia, PA: WB Saunders; 2000; 227–39.
- Wieshmann UC. Clinical application of neuroimaging in epilepsy. J Neurol Neurosurg Psychiatry 2003; 74: 466–70.

© West Indian Medical Journal 2024.

This is an article published in open access under a Creative Commons Attribution International licence (CC BY). For more information, please visit https://creativecommons.org/licenses/by/4.0/deed.en_US.

