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CASE REPORT

Brain Imaging Abnormalities in Cerebral Palsy

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ABSTRACT

BACKGROUND

We have previously reported our extensive experience with cerebral palsy in a plethora of publications. However, brain imaging studies were not available for all of the patients, and the brain imaging abnormalities observed in children with cerebral palsy were not adequately highlighted. The aim of this retrospective study is to describe brain imaging abnormalities in children with cerebral palsy.

PATIENT AND METHODS

Brain imaging studies (CT-scan, MRI, Ultrasound) or medical reports including descriptions of brain imaging studies were available for twenty-nine patients (aged 10 months to 8 years) with cerebral palsy (20 males and 9 females) including 21 patients with spastic cerebral palsy, 3 patients with ataxic cerebral palsy, and 5 patients who didn't have significant spasticity that were considered to have an unclassified type rather than hypotonic cerebral palsy.

RESULTS

Of the twenty-one patients with spastic cerebral palsy, thirteen patients had evidence of brain atrophy with or without other abnormalities such as periventricular leukomalacia, and three of them had evidence of diffuse brain atrophy. Two patients had arachnoid cyst. One patient had isolated periventricular leukomalacia without obvious brain atrophy or other imaging abnormalities. Of the five patients with unclassified type, four had evidence of brain atrophy. Other patients with ataxic cerebral palsy had cerebellar atrophy/cerebellar hypoplasia.

CONCLUSION

Brain atrophy was the most important imaging abnormalities in children with cerebral palsy, occurring in 18 of the 29 patients in this study. Other brain imaging abnormalities include periventricular leukomalacia, arachnoid cyst, and cerebellar atrophy/cerebellar hypoplasia. The pattern of brain imaging abnormalities was rather different from the pattern reported from other countries such as USA, Spain, Japan, and United Arab Emirates.

KEYWORDS

Cerebral palsy; Abnormal development; Brain damage

INTRODUCTION

Cerebral palsy is a group of heterogeneous disorders caused by an abnormal development or brain damage affecting mostly regions controlling movements, balance, and posture. The brain damage and abnormalities in cerebral palsy are characteristically non-progressive, but lead to permanent abnormalities of movements, posture, and limitation of mobility [1-4].

We have previously reported our extensive experience with cerebral palsy in a plethora of publications. However, brain imaging studies were not available for all of the patients, and the brain imaging abnormalities observed in children with cerebral palsy were not adequately studied nor highlighted [1-15]. The aim of this retrospective study is to describe brain imaging abnormalities in children with cerebral palsy.

PATIENT AND METHODS

Brain imaging studies (CT-scan, MRI, Ultrasound) or medical reports including descriptions of brain imaging studies were available for twenty-nine patients (10 months- 8 years) with cerebral palsy (Twenty males and nine females) including 21 patients with spastic cerebral palsy, 3 patients with ataxic cerebral palsy, and 5 patients who didn't have significant spasticity that were considered to have an unclassified type rather than hypotonic cerebral palsy.

There were 21 patients (aged 10 months- 8 years) with spastic cerebral palsy (12 males and 9 females) including three patients with severe disease, and one of them had epilepsy. Two patients had hemiplegic cerebral palsy including one patient with history of seizure disorders. One female patient had significant squint causing torticollis.

In two patients, there was a clear history of birth asphyxia that represented an obvious contributory etiologic factor, and in one patient cerebral palsy occurred as a complication of meningitis,

The severe quadriplegic cerebral palsy in patient 1 was complicated by bilateral hyperoxaluric nephrocalcinosis with early renal dysfunction and ultrasonographic evidence of adrenal haemorrhage. Patient 6 (Table 1) had a pelvic MRI showing atrophic undescended right testis located in the mid-inguinal region.

RESULTS

Of the twenty-one patients with spastic cerebral palsy, thirteen patients had evidence of brain atrophy (Table 1), and three of them had evidence of diffuse brain atrophy. One of the patients had also polymicrogyria and dilatation of the basal cisterns, one of the patients with diffuse brain atrophy also had bilateral parietal lobe white matter periventricular leukomalacia with mildly dilated lateral ventricle, and one patient also had bilateral parietal lobes deep white matter periventricular leukomalacia and mildly dilated lateral ventricles.

Two patients had arachnoid cyst including one with a large well-defined cyst in left temporal fossa causing atrophy of the temporal lobe, and a patient had arachnoid cyst in the left sphenoid 34 mm × 16 mm in diameter, and periventricular leukodystrophy-like changes of signal abnormalities in the occipital area.

One patient had isolated periventricular leukomalacia without obvious brain atrophy or other imaging abnormalities.

One patient, who had clear history of birth asphyxia, had diffuse hypodensity of both cerebral hemispheres sparing the cerebellar hemispheres and the basal ganglia suggesting hypoxic brain ischemia caused by birth asphyxia.

One patient had bilateral dilatation of the lateral ventricles, 14mm and mild dilatation of the third ventricles.

Table 1: The brain imaging studies of patients with spastic cerebral palsy.

S. No.	Sex	Age	Characteristics	Brain imaging
1	M	21 months	Birth asphyxia induced-brain damage. Severe quadriplegic type	MRI: Evidence of brain atrophy
2	F	N/A	Spastic quadriplegic type	Periventricular leukomalacia
3	M	2 years	Severe spastic type	Brain atrophy
4	M	N/A	Severe, with history of epilepsy	Brain atrophy
5	M	N/A	Birth asphyxia	CT-scan: Diffuse hypodensity of both cerebral hemispheres sparing the cerebellar hemispheres and the basal ganglia suggesting hypoxic brain ischemia caused by birth asphyxia.
6	M	N/A	Spastic quadriplegic type	CT scan: Evidence of diffuse brain atrophy. Pelvic MRI showed atrophic undescended right testis located in the mid-inguinal region
7	M	N/A		MRI showed brain atrophy with polymicrogyria and dilatation of the basal cisterns
8	F	1 year	Post-meningitis	MRI: Evidence of diffuse brain atrophy
9	F	N/A		CT-scan: Evidence of brain atrophy
10	F	N/A		CT-scan: Evidence of brain atrophy
11	F	N/A		MRI showed evidence of diffuse brain atrophy with bilateral parietal lobe white matter peri-ventricular leukomalacia with mildly dilated lateral ventricle.
12	M	10 months	Spastic	Ultrasound: Bilateral dilatation of the lateral ventricles, 14mm and mild dilatation of the third ventricles.
13	M	2 years	Spastic	Ultrasound was normal, but MRI showed evidence of brain atrophy.
14	F	6 years & 2 months	Spastic	MRI: Bilateral enlargement of the subarachnoid space in the frontal, temporal, & anterior parietal regions, mildly dilated ventricles.
15	M	4 years & 9 months	Spasticity in the lower limbs, mental retardation, delayed speech and gait abnormalities. He also had seizures that had already been controlled with sodium valproate.	CT-scan: Large well-defined arachnoid cyst in left temporal fossa causing atrophy of the temporal lobe.
16	F	N/A	Squint / torticollis	MRI: Signal intensity involving the right thalamic, midbrain and hypo campus region. Dilation of the right horn of the right lateral ventricle suggesting ischemic injury
17	F	N/A	Left Hemiplegia	CT scan: Large right sided temporo-parietal gliotic changes with large communicating porencephaly cyst
18	M	8 years	Left spastic hemiparesis causing gait abnormalities, seizures that had already been controlled with sodium valproate, and autism	CT-scan: Arachnoid cyst in the left sphenoid 34mmx16mm in diameter, and periventricular leukodystrophy-like changes of signal abnormalities in the occipital area.
19	M	3 years	Spastic	MRI: Evidence of moderate atrophic changes & bilateral parietal lobes deep white matter periventricular leukomalacia & mildly dilated lateral ventricles.
20	M	N/A	Spastic	CT-scan: Evidence of atrophic changes in the temporal region.
21	F	4 years	Spastic	CT-scan: Evidence of mild diffuse atrophic changes

One patient had bilateral enlargement of the subarachanoid space in the frontal, temporal, & anterior parietal regions, mildly dilated ventricles.

One patient had signal intensity involving the right thalamic, midbrain and hypocampus region, dilation of the right horn of the right lateral ventricle suggesting ischemic injury.

One patient had a large right sided temporo-parietal gliotic changes with large communicating porencephaly cyst.

Three male patients had ataxic cerebral palsy including one patient with a clear history of birth asphyxia who had evidence of on generalized cerebral atrophy on MRI. The CT-scan in the second patients with ataxic cerebral palsy showed cerebellar atrophy with absence of the inferior vermis, and the posterior fossa was filled with CSF. The third patient had evidence of cerebellar hypoplasia, vermian hypoplasia with retro-cerebellar hypoplasia on MRI.

Five male patients who didn't have significant spasticity that were considered to have an unclassified type rather than hypotonic cerebral palsy. Four patients had evidence of brain atrophy including one patient with evidence of mild brain atrophy on MRI, one patient had mild dilatation of the third and lateral ventricles that were considered by the radiologist as and evidence of brain atrophy. In one patient, the CT-scan evidence of brain atrophy was associated with prominent lateral ventricles. One patient had left hemi-brain atrophy on CT-scan.

DISCUSSION

In this study, brain atrophy was the most important imaging abnormalities in children with cerebral palsy, occurring in 18 of the 29 patients in this study. Other brain imaging abnormalities include periventricular leukomalacia, arachnoid cyst, and cerebellar atrophy/cerebellar hypoplasia.

van Bogaert et al. (1992) reported 3 children with cerebral palsy who had normal brain CT-scans, but MRI revealed some CT-scan undetectable white matter abnormalities. The two patients with spastic diplegia showed bilateral abnormalities either in the subcortical regions or in the occipital periventricular areas. The patient with congenital hemiplegia exhibited unilateral abnormalities in the periventricular area. van Bogaert et al. suggested that MRI is probably more useful than CT-scan for the assessment of cerebral palsy [16].

In our study, isolated periventricular leukomalacia occurred in one patient with spastic cerebral palsy and occurred with evidence of brain atrophy in another two patients who also had spastic cerebral palsy.

In our study, polymicrogyria was observed on MRI in association with brain atrophy and dilatation of the basal cisterns in one patient with spastic cerebral palsy.

Truwit et al (1992) from the USA reported the MRI findings of 40 patients (aged 1 month to 41 years) with cerebral palsy. In 82% of the 11 patients who had been born before term, MRI showed periventricular white matter damage suggestive of hypoxic-ischemic brain injury. MRI findings in the remaining 29 patients who had been born at term included gyral abnormalities such as polymicrogyria, isolated periventricular leukomalacia, and watershed cortical or deep gray nuclear damage. In 16 (55%) of 29 patients born at term, MRI included intrauterine brain damage including developmental abnormalities [17].

In our study, imaging abnormalities in the three patients who had ataxic cerebral palsy included generalized cerebral atrophy, cerebellar atrophy with absence of the inferior vermis, and cerebellar hypoplasia, vermian hypoplasia with retro-cerebellar hypoplasia.

Imamura et al. (1992) from Japan reported the MRI abnormalities in 5 patients with ataxic cerebral palsy which included generalized atrophy of cerebellum (particularly in the anterior superior region) and slight atrophy of pons in a patient with inherited disorder. The second patient also had cerebellar atrophy especially in anterior superior region. In the third patient, CT and MRI showed generalized spino-ponto-cerebellar atrophy, and the patient received a diagnosis of Marinesco-Sjörgren syndrome. CT and MRI of the fourth patient showed generalized spino-ponto-cerebellar atrophy. The MRI of the fifth patient showed cranium bifida and agenesis of anterior medullar velum [18].

Campistol Plana et al. (1996) from Barcelona reported the CT-scan findings in 90 children with spastic cerebral palsy of the diplegic type. 51 of them had typical evidence of residual periventricular leukomalacia. Six of the 51 children were recognized in early in life by brain ultrasound. Campistol Plana et al. suggested that the CT-scan evidence of residual periventricular leukomalacia include slight ventricular dilation, subcortical atrophy or periventricular heterotopia [19].

In our study, imaging abnormalities in the two patients with hemiplegic cerebral palsy included large right sided temporo-parietal gliotic changes with large communicating porencephaly cyst in one patient, and arachnoid cyst in the left sphenoid observed in association with periventricular leukodystrophy-like changes of signal abnormalities in the occipital area, in the second patient.

Gururaj et al. (2002) reported brain imaging findings in 65 children (41 term-born and 24 preterm-born) patients with cerebral palsy from the United Arab Emirates. 23 patients had spastic diplegia, with 57% of them had marked periventricular leukomalacia, which occurred more in preterm-born patients. In 13 with hemiplegia, 12 had unilateral abnormalities on brain imaging. Patients with spastic tetraplegia had extensive, bilateral, diffuse brain damage. Extra-pyramidal cerebral palsy was much commoner in term-born patients and 80 % of them had marked abnormalities in the basal ganglia. Ataxic cerebral palsy was not associated with important correlation between the clinical manifestations and abnormalities on brain imaging [20].

The study of Gururaj et al. suggested that brain imaging findings were closely related to the type of cerebral palsy and the clinical abnormalities except in ataxic cerebral palsy [20].

Wu et al. (2006) from California University reported the brain imaging abnormalities in 78 infants with congenital hemiparetic cerebral palsy (55 had an MRI and 23 patients had a CT-scan). Perinatal arterial infarction was the most common brain imaging abnormalities occurring in 30% of term infants. Infants with right hemiparesis or moderate to severe weakness were more likely to show a perinatal arterial infarction on imaging studies. Periventricular white matter abnormalities occurred more in preterm infants. Brain malformations occurred in 14 patients, and included polymicrogyria, heterotopia, and schizencephaly. 14 patients who didn't have abnormalities on imaging study showed more likelihood to outgrow hemiparesis by the age of three years than patients who had abnormalities on an image study [21].

In our study, one patient has cerebral palsy occurring as a complication of meningitis during early infancy.

Tillberg and colleagues (2008) reported the occurrence of CNS infection during life in 13 children which resulted in the development of hemiplegic cerebral palsy [22].

Therefore, the pattern of brain imaging abnormalities was rather different from the pattern reported from other countries such as USA, Spain, Japan, and United Arab Emirates.

CONCLUSION

Brain atrophy was the most important imaging abnormalities in children with cerebral palsy, occurring in 18 of the 29 patients in this study. Other brain imaging abnormalities include periventricular leukomalacia, arachnoid cyst, and cerebellar atrophy/cerebellar hypoplasia. The pattern of brain imaging abnormalities was rather different from the pattern reported from other countries such as USA, Spain, Japan, and United Arab Emirates.

CONFLICTS OF INTEREST

None.

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