Case Report

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Four cases of pediatric neuralgic amyotrophy treated with immunotherapy: one-year follow-up and literature review

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Abstract

Neuralgic amyotrophy (NA) is a neurological disease that occurs across all age groups, but its prognosis in children is controversial. The present report adds to the knowledge about its prognosis by describing four cases of pediatric NA in which the patients were treated with immunotherapy and followed up for 1 year. We also present a summary of relevant cases of pediatric NA treated with immunotherapy. The clinical features of the four present cases were similar to those of previously reported cases, and their symptoms improved after immunotherapy. At the 1-year follow-up, three of the children gained near complete recovery, and their improvement was significantly better than that observed at the 2-month follow-up. A review of the literature showed that most previously reported children with NA showed improvement after immunotherapy, but no more than half of the patients recovered fully. These findings indicate that in children with NA, immunotherapy is fairly effective and its benefits improve with time. Thus, long-term follow-up is needed in these patients to determine their prognosis.

Keywords

Neuralgic amyotrophy, pediatric, immunotherapy, prognosis, review, case report

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Introduction

Neuralgic amyotrophy (NA), also termed brachial neuritis or Parsonage–Turner syndrome, is a neurological entity characterized by the sudden onset of pain, usually in the shoulder or arm region, and subsequent muscular weakness, sensory loss, and atrophy.[1] Its actual incidence is estimated to be at least 20 to 30 cases per 100,000 individuals,[2] and it has been reported in all age groups. The median age at onset ranges from the second decade to the early fourth decade of life[3]; thus, it has rarely been documented in children. Among children with NA, an obvious biphasic age distribution has been observed in newborns (<8 weeks) and adolescents.[4] Male predominance of NA has been noted in both adults (2:1) and children (2.3:1.0).[4,5] The disease can be idiopathic or inherited; the latter, known as hereditary NA, is an autosomal dominant condition characterized by mutations in the *SEPT9* gene.[6] Idiopathic NA is a relatively common form of the condition that frequently occurs secondary to infections and immunization. Its occurrence is indicative of an immune-mediated pathogenesis, which is also supported by the presence of serum antibodies and the efficacy of immunotherapy.[7] Diagnosis is achieved in the clinical setting with the help of electrophysiological studies and magnetic resonance imaging (MRI), which are useful for excluding other differential diagnoses and confirming the presence of NA. Immunotherapy is usually effective in the early stage of the disorder, so prompt diagnosis is important.[8] The prognosis of childhood idiopathic NA is considered favorable, with full recovery reported in 63% of patients and partial recovery in 25%; in addition, recovery is quick (mean of 11.1 months).[4] However, a recent study showed that the prognosis of NA in children is not favorable.[9] Therefore, despite previous studies and reports on childhood NA, the treatment and prognosis remain unclear, and more reports would be useful to gain a better understanding of this disease and ensure that it is treated in a timely manner. The current report adds to the literature on childhood NA by describing four cases of NA that occurred secondary to respiratory infection.

We herein report our encounter with four cases of NA at the Children's Hospital of Soochow University. All four cases occurred in October 2018, and the patients were treated with immunotherapy. We first describe each case (Table 1) and then discuss and compare the findings with previous reports. Finally, we review relevant studies, including the present study, on the use of immunotherapy for NA in children.

Case reports

Case 1

A boy aged 2 years 3 months presented with a history of fever and cough that were followed by weakness in the right arm after 1 week. At the time of examination, he did not complain of pain in the right arm. His medical history and family history were unremarkable. Physical examination showed decreased strength in the proximal and distal muscles of the right arm (Medical Research Council [MRC] score, 2/5). The other limbs showed no abnormalities. Additionally, no abnormalities were found on blood tests, including a blood count, blood culture, liver function tests, creatine kinase measurement, and serology for related respiratory viruses, *Mycoplasma*, and hepatitis E virus. Cerebrospinal fluid (CSF) analysis revealed a normal white blood cell count and protein concentration. Electromyography (EMG) revealed damage to the upper, middle, and lower trunk of the right brachial plexus, but brain MRI showed no obvious abnormalities.

Table 1. Summary of clinical features and diagnostic studies of the four patients in the present study.

Patient	Clinical features	CSF examination	Abnormal EMG results	MRI findings
1	Right arm weakness without pain	Normal	Upper, middle, and lower trunk of the right brachial plexus	Normal
2	Right arm paralysis with no associated pain	–	Right axillary nerve and musculocutaneous nerve	Thickening in the right brachial plexus
3	Right arm paralysis, right leg paresis, no pain	WBC count = 52 cells/μL	Upper, middle, and lower trunk of the right brachial plexus	Normal
4	Left upper arm weakness without pain, left facial palsy, right lingual paralysis, true bulbar palsy	Normal	Upper trunk of the left brachial plexus	Normal

CSF, cerebrospinal fluid; EMG, electromyography; MRI, magnetic resonance imaging; WBC, white blood cell.

Intravenous immunoglobulin (IVIG) (2 g/kg) and methylprednisolone (4 mg/kg) were empirically administered; this was followed by oral prednisolone administration at a dose of 2 mg/kg, which was gradually tapered within 2 months. At the 2-month follow-up, the patient had muscle atrophy in the right supraspinatus and infraspinatus muscles and deltoid muscle and could not lift his shoulder and wrist; however, he was able to move the distal three fingers of his right hand. At the 1-year follow-up, the patient still exhibited marked wasting of these muscles, but he could move his fingers normally. However, his ability to lift his shoulder was still weak.

Case 2

A previously healthy 22-month-old boy was admitted with limb paresis that had occurred 2 weeks after he developed pneumonia. The limb paresis had been present for 6 days before admission to the hospital. Abnormalities on neurological examination were restricted to the right arm and involved both the proximal muscles (MRC score, 2/5) and distal muscles (MRC score, 4/5). His tendon reflexes were normal. As in Case 1, the results of his blood tests were normal. Lumbar puncture was not performed because we did not obtain consent. Brain MRI showed no obvious abnormalities, but spinal MRI revealed a region with thickening in the right brachial plexus. An electrophysiological study showed that the right axillary nerve and musculocutaneous nerve were injured. Treatment with IVIG (2 g/kg) and methylprednisolone (4 mg/kg) was effective in helping him regain muscle strength. At 2 months after onset, his proximal muscle strength had returned to the baseline level (MRC score, 3/5). At the 1-year follow-up, he had nearly complete recovery with slightly abnormal arm extension and mild muscle atrophy.

Case 3

A girl aged 4 years 3 months was referred to our hospital with a 3-day history of right limb weakness. She had developed bronchitis 10 days previously; this was accompanied by cheek ecchymosis, which had disappeared just before she was admitted to the hospital. She did not complain of pain on admission. Physical examination revealed the following neurological deficits: paralysis of the right arm (MRC score, 1/5), a lesser degree of paresis in the right leg (MRC score, 4/5), and no sensory involvement. Reduced reflexes were identified along the entire right arm. The results of blood tests for the following parameters were normal: hemoglobin concentration, glucose concentration, leukocyte count, sedimentation rate, liver and kidney function, blood culture, antinuclear antibodies, anti-DNA antibodies, and serology for hepatitis E virus, cytomegalovirus, Epstein–Barr virus, and *Mycoplasma*. Shoulder radiography and MRI of the whole spine and brain showed normal findings. CSF analysis revealed slight elevation of the white blood cell count (52 cells/μL), but the protein concentration was normal. EMG revealed several injuries to the upper, middle, and lower trunk of the right brachial plexus. IVIG was initiated, but her parents did not provide consent for steroid therapy. At the 2-month check-up, the patient could lift her shoulder slightly and move her fingers more effectively. At the 1-year follow-up, the patient could move her right leg freely and had almost gained full function of her upper limb, but she had slight difficulty in lifting the right arm sideways and showed slight atrophy of the deltoid muscle.

Case 4

An otherwise healthy 6-year-old boy presented to the Respiratory Department with a 3-day history of fever and cough. He was treated with latamoxef and azithromycin as well as nebulization with budesonide suspension plus albuterol and ipratropium bromide. However, the fever remained uncontrolled. On day 5, he developed progressive paralysis of the proximal left upper extremity that was associated with left facial palsy and right deviation of the tongue. He also experienced slurring while speaking and found it difficult to swallow. He did not complain of pain or paresthesia in the left arm. A neurologic examination demonstrated left proximal limb weakness (MRC score, 2/5). Cranial nerve examination revealed peripheral facial and lingual paralysis with true bulbar palsy. The patient was then taken to the Neurology Department for lumbar puncture and further examination. The results of the CSF analysis were unremarkable. Autoimmune encephalitis antibodies (e.g., N-methyl-D-aspartate receptor) and demyelinating antibodies (aquaporin 4 and myelin oligodendrocyte glycoprotein) were not detected in the serum or CSF. The results of routine blood tests and nucleic acid tests for microbiology, bocavirus, rhinovirus, hepatitis E virus, metapneumovirus, influenza virus, *Mycoplasma*, and *Chlamydia* pneumonia were normal. MRI of the whole spine and brain revealed no abnormalities. Based on these findings, treatment with methylprednisolone (4 mg/kg) and IVIG (2 g/kg) was commenced. EMG at 6 weeks after presentation showed a small degree of damage to the upper trunk of the left brachial plexus. The patient also received treatment from a physical therapist. After 2 months of undergoing this therapy, the patient showed improved swallowing function but still had facial paralysis. He was able to move his distal limbs, but he showed only slight improvement in his ability to lift his proximal limb. The 1-year follow-up examination showed that he could move his hands

and legs normally, but slight facial paralysis and muscle atrophy persisted.

Discussion

We have herein reported four cases of childhood NA that presented with near complete recovery after immunotherapy at the 1-year follow-up.

NA is classically considered an adult pathology. Children with idiopathic NA comprise a subgroup distinct from adults who are affected by the condition, especially with regard to the occurrence of pain, which is always absent or difficult to assess in children.[10] None of our four pediatric patients reported feeling pain, and no signs of pain were observed. Weakness, pain, and sensory involvement are the prominent features in adults with NA; in contrast, pain and sensory involvement are often rare or not noticeable in children. Thus, diagnosis of this condition in children is more difficult. Although EMG significantly reduces the incidence of misdiagnosis and diagnostic delay, it is necessary to develop noninvasive diagnostic techniques for pediatric patients. Ultrasonography is a promising noninvasive technique for studying peripheral nerves in patients with NA.[5]

NA is reportedly more frequent in males than females across all age groups.[11] This was also true among our cases; the male: female ratio was 3:1. This sex distribution is similar to that of Guillain–Barré syndrome. Both NA and Guillain–Barré syndrome are more likely to affect men, while other immune-related diseases are usually more likely to affect women. Matrix metalloproteinase 9 has been found to play an important role in the male predominance of Guillain–Barré syndrome[12]; thus, differences in the expression and activation of cytokines between male and female patients may be a possible cause of the sex-based difference in the incidence of NA.

Three of four children in our study were affected on their right side; this is consistent with the observation in adults. One review pointed out that this distribution pattern mainly exists in school-age children and attributed it to the development of right-handedness.[4] However, the children whose right side was affected in the present study were younger than school age. In contrast, other studies have shown that irrespective of whether an individual is right-handed, the disease tends to occur on the right side.[2] Thus, the right-side muscles are probably more likely to be involved in this disease.

In accordance with other literature,[4] the upper trunk of the brachial plexus nerve showed the most involvement in our patients. Two of our patients had extensive damage to the middle and lower trunk. Moreover, two patients had lesions in regions other than the brachial plexus; this means that extra-brachial nerves could also be involved. The patient in Case 3 also had slight leg weakness, which is sometimes the only symptom in some variant cases of NA.[7] In Case 4, extensive injury to the cranial nerves, including the facial and bulbar nerves, was observed. Cranial nerve involvement is rare in idiopathic NA (17%); it is more common in hereditary NA (56%).[2] Patients with NA who have hepatitis E virus usually exhibit asymmetric bilateral brachial plexus involvement and damage outside the brachial plexus,[13] but none of the four patients in this case series were positive for hepatitis E virus.

The estimated incidence of upper respiratory infection prior to NA among affected children is 34%.[10] In the present study, however, respiratory infections occurred in all children within 1 week before disease onset. Furthermore, the onset time was concentrated in early October 2018, and the children were concentrated in the same geographical region. Therefore, we deduced that undetected microorganisms and the

Table 2. Clinical features of pediatric neuralgic amyotrophy treated with immunotherapy.

Age	Sex/Side	Preceding event	Treatment	Time to treatment	Prognosis	Follow-up	Study
5 years	M/l	Upper respiratory infection	Steroids (i.v.)	Acute phase	Full recovery	3 months	[19]
15 years	F/r	EBV infection	Hydrocortisone (i.v.)	Acute phase	Nearly complete recovery	16 months	[20]
4.5 months	M/b	Oral polio vaccine	IVIG (twice)	Acute phase	No improvement	NA	[21]
9 months	F/l	Fever and rash	IVIG	Acute phase	Full recovery	NA	[21]
14 months	M/l (leg)	HFMD	IVIG	Acute phase	Full recovery	2 years	[21]
19 months	M/b (legs)	Febrile illness	IVIG	Acute phase	Partial recovery	NA	[21]
14 months	F/b (legs)	HFMD	IVIG	Acute phase	Full recovery	2 years	[21]
4.5 years	M/l	Respiratory infection	Methylprednisolone pulse therapy (i.v.)	Acute phase	Partial recovery	2 years	[22]
11 years	M/b	Henoch–Schönlein purpura	Methylprednisolone (high oral dose) and IVIG (twice)	Day 7 Day 14	No improvement	4 months	[23]
7 weeks	M/b	Fever	Prednisolone (i.v.)	Acute phase	Full recovery	1 month	[24]
8.5 years	M/r	Kidney transplantation, TAC-associated	Methylprednisolone pulse therapy (i.v.) + IVIG + TAC replaced	Acute phase	Full recovery	6 months	[25]
7 years	F/r (leg)	Epileptic episode	Methylprednisolone pulse therapy (i.v.) (twice)	Acute phase	Full recovery	2 months	[26]
4 years	M/r	Family history	Methylprednisolone pulse therapy (i.v.)	Acute phase	Partial recovery	6 months	[26]
6 months	F/r	Upper respiratory infection	Prednisolone	Acute phase	Nearly complete recovery	10 months	[27]
16 years	F/r	None	Plasmapheresis	3 months	Full recovery	3 years	[9]
15 years	F/l > r	None	Corticosteroids (oral)	6 weeks	Partial recovery	1.5 years	[9]
11 years	M/r	None	Corticosteroids (oral)	4 weeks	Partial recovery	9 months	[9]

(continued)

Table 2. Continued.

Age	Sex/Side	Preceding event	Treatment	Time to treatment	Prognosis	Follow-up	Study
14 years	M/r	None	Plasmapheresis	4 months	Full recovery	15 months	[9]
2 years	M/r	Respiratory infection	Methylprednisolone (i.v.) + IVIG	Day 14	Partial recovery	1 year	Present
22 months	M/r	Respiratory infection	Methylprednisolone (i.v.) + IVIG	Day 7	Nearly complete recovery	1 year	Present
4 years	F/r	Respiratory infection	IVIG	Day 10	Nearly complete recovery	1 year	Present
6 years	M/l	Respiratory infection	Methylprednisolone (i.v.) + IVIG	Day 2	Nearly complete recovery	1 year	Present

M, male; F, female; r, right; l, left; b, bilateral; EBV, Epstein–Barr virus; IVIG, intravenous immunoglobulin; i.v., intravenous; NA, not available; HFMD, hand, foot, and mouth disease; TAC, tacrolimus.

subsequent immune response could have been the underlying cause.

No specific drug treatment is available for NA. Because the disease is likely to be immune-mediated, immunotherapy (including IVIG, steroids, and even plasmapheresis) has been performed in adults. van Eijk et al.[8] reported good functional improvement in a study of 50 adult patients who were prescribed oral prednisolone. A smaller case series showed improvement in 9 of 10 patients who were administered IVIG and methylprednisolone pulse therapy.[14] Other reports have confirmed the efficacy of IVIG, but none of them were of high quality.[7,15,16] Further, early use of immunomodulators has been emphasized in some studies. Although one report described a patient who benefited from the combination of IVIG and methylprednisolone administered at 10 months after disease onset,[16] early treatment is still considered to shorten the recovery time of the disease.[8]

Reports on the efficacy of immunotherapy for pediatric NA are limited. Our search of PubMed for relevant cases of pediatric NA treated with immunotherapy (using combinations of the terms "neuralgic amyotrophy," "brachial neuritis," "Parsonage–Turner syndrome," "pediatric," and "children") revealed 10 studies (Table 2).[9,19–27] We excluded patients with osteomyelitis or septic arthritis because the combination of NA with these two disorders may indicate a different pathogenesis.[17,18] In total, 22 patients underwent immunotherapy among the selected studies and the present study. The mean follow-up time was 12.9 months. Among the 22 patients, 9 (40.9%) gained full recovery, but 2 (9.1%) showed no improvement. Near complete and partial recovery were achieved in five (22.7%) and six (27.3%) children, respectively. In general, immunotherapy was found to be favorable for recovery.

As previously reported,[9] the present study has confirmed that even after immunotherapy, more than half of children with NA still have some degree of sequelae. The percentage of patients who achieved full recovery in the present report is lower than that reported by Host and Skov.[4] This is probably because their study included several patients in whom NA occurred secondary to osteomyelitis and arthritis, which often has a high rate of full recovery. Although immunotherapy did not result in full recovery in our patients, three of the four children were living an almost normal life at the 1-year follow-up. Notably, all four children's symptoms showed significantly greater improvement after the long-term follow-up of 1 year than after the short-term follow-up of 2 months; thus, the prognosis seems to improve with time. Importantly, however, the efficacy of immunotherapy may be limited in the treatment of NA caused by the oral polio vaccine (live attenuated vaccine) and severe immune diseases such as Henoch–Schönlein purpura. Additionally, when NA occurs secondary to drug-induced damage, it is more important to remove the drugs from the system than start immunotherapy.

In conclusion, we have presented four cases of pediatric NA in patients who were followed up for 1 year and received immunotherapy. The clinical features of these patients were similar to those reported in previous studies. Based on these case findings and our review, it appears that the long-term follow-up prognosis of immunotherapy is fair. However, more cases and longer-term follow-up may be needed for a complete understanding of the factors that determine full recovery.

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Declaration of conflicting interest

The author(s) declare that there is no conflict of interest.

Ethical compliance

This study was approved by the Ethics Committee of the Children's Hospital of Soochow University. Written informed consent was obtained from the parents of all participants.

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