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

Xiaoyue Hu¹,², Miao Jing², Jun Feng¹ and Jihong Tang¹

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

Date received: 14 November 2019; accepted: 17 February 2020

¹Department of Neurology, Children’s Hospital of Soochow University, Suzhou, Jiangsu Province, China
²Department of Neurology, Wuxi Children’s Hospital, Wuxi, Jiangsu Province, China

Corresponding author:
Jihong Tang, Department of Neurology, Children’s Hospital of Soochow University, 303 Jingde Road, Suzhou, Jiangsu Province 215003, China.
Email: tjhzsh@126.com

Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 License (https://creativecommons.org/licenses/by-nc/4.0/) which permits non-commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the SAGE and Open Access pages (https://us.sagepub.com/en-us/nam/open-access-at-sage).
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. Its actual incidence is estimated to be at least 20 to 30 cases per 100,000 individuals, 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; 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. Male predominance of NA has been noted in both adults (2:1) and children (2.3:1.0). 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. 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. 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. 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). However, a recent study showed that the prognosis of NA in children is not favorable. 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.
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 bronchi-tis 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 his 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, rhi-novirus, 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. 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.

NA is reportedly more frequent in males than females across all age groups. 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; 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. 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. Thus, the right-side muscles are probably more likely to be involved in this disease.

In accordance with other literature, 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. Cranial nerve involvement is rare in idiopathic NA (17%); it is more common in hereditary NA (56%). Patients with NA who have hepatitis E virus usually exhibit asymmetric bilateral brachial plexus involvement and damage outside the brachial plexus, 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%. 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/I      | 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)| Fever and 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             | 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.) (twice) | Acute phase       | Full recovery                  | 6 months  | [25]  |
| 7 years   | F/r (leg)| Epileptic episode                  | Methylprednisolone pulse therapy (i.v.) + IVIG + TAC replaced | Acute phase       | Full recovery                  | 2 months  | [26]  |
| 4 years   | M/r      | Family history                     | Methylprednisolone pulse therapy (i.v.) (twice) | 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)
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.

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

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.
As previously reported, 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. 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.

**Acknowledgement**

We are grateful to the patients for their cooperation.

**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.

**Funding**

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the Science and Technology Program of Suzhou (No. SS201866), the Science and Technology Project of the Health and Health Committee of Jiangsu Province (No. H2018010), Wuxi Young Medical Talents (No. QNRC021), Wuxi Maternal and Child Health Research Project (No. FYKY201904), and Wuxi Medical Development Discipline (No. FZXK001).

**ORCID iD**

Jihong Tang https://orcid.org/0000-0001-5656-4348

**References**

1. van Alfen N, van der Werf SP and van Engelen BG. Long-term pain, fatigue, and impairment in neuralgic amyotrophy. *Arch Phys Med Rehabil* 2009; 90: 435–439.
2. van Alfen N and van Engelen BG. The clinical spectrum of neuralgic amyotrophy in 246 cases. *Brain* 2006; 129: 438–450.
3. van Alfen N, van Engelen BG and Hughes RA. Treatment for idiopathic and hereditary neuralgic amyotrophy (brachial neuritis). *Cochrane Database Syst Rev* 2009; 3(3): Cd006976. doi: 10.1002/14651858.CD006976.pub2.
4. Host C and Skov L. Idiopathic neuralgic amyotrophy in children. Case report, 4 year follow up and review of the literature. *Eur J Paediatr Neurol* 2010; 14: 467–473.
5. van Eijk JJ, Groothuis JT and Van Alfen N. Neuralgic amyotrophy: an update on...
diagnosis, pathophysiology, and treatment. Muscle Nerve 2016; 53: 337–350.
6. Kuhlenbaumer G, Hannibal MC, Nelis E, et al. Mutations in SEPT9 cause hereditary neuralgic amyotrophy. Nat Genet 2005; 37: 1044–1046.
7. Moriguchi K, Miyamoto K, Takada K, et al. Four cases of anti-ganglioside antibody-positive neuralgic amyotrophy with good response to intravenous immunoglobulin infusion therapy. J Neuroimmunol 2011; 238: 107–109.
8. van Eijk JJ, van Alfen N, Berrevoets M, et al. Evaluation of prednisolone treatment in the acute phase of neuralgic amyotrophy: an observational study. J Neurol Neurosurg Psychiatry 2009; 80: 1120–1124.
9. Al-Ghamdi F and Ghosh PS. Neuralgic amyotrophy in children. Muscle Nerve 2018; 57: 932–936.
10. van Alfen N, Schuuring J, van Engelen BG, et al. Idiopathic neuralgic amyotrophy in children. A distinct phenotype compared to the adult form. Neuropediatrics 2000; 31: 328–332.
11. Seror P. Neuralgic amyotrophy. An update. Joint Bone Spine 2017; 84: 153–158.
12. Yin PQ, Sun YY, Chen HP, et al. Genomewide gene expression analysis of peripheral leukocytes in relation to the male predominance of Guillain-Barre syndrome: differential gene expression between male and female patients. Int J Neurosci 2016; 126: 531–541.
13. van Eijk JJJ, Dalton HR, Ripellino P, et al. Clinical phenotype and outcome of hepatitis E virus–associated neuralgic amyotrophy. Neurology 2017; 89: 909–917.
14. Naito KS, Fukushima K, Suzuki S, et al. Intravenous immunoglobulin (IVIg) with methylprednisolone pulse therapy for motor impairment of neuralgic amyotrophy: clinical observations in 10 cases. Intern Med 2012; 51: 1493–1500.
15. Nakajima M, Fujioka S, Ohno H, et al. Partial but rapid recovery from paralysis after immunomodulation during early stage of neuralgic amyotrophy. Eur Neurol 2006; 55: 227–229.
16. Morishima R, Nagaoka U, Nagao M, et al. Chronic brachial plexus neuritis that developed into typical neuralgic amyotrophy and positively responded to immunotherapy. Intern Med 2018; 57: 1021–1026.
17. Lejman T, Strong M, Michno P, et al. Septic arthritis of the shoulder during the first 18 months of life. J Pediatr Orthop 1995; 15: 172–175.
18. Estienne M, Scaioli V, Zibordi F, et al. Enigmatic osteomyelitis and bilateral upper limb palsy in a neonate. Pediatr Neurol 2005; 32: 56–59.
19. To WC and Traquina DN. Neuralgic amyotrophy presenting with bilateral vocal cord paralysis in a child: a case report. Int J Pediatr Otorhinolaryngol 1999; 48: 251–254.
20. Janes SE and Whitehouse WP. Brachial neuritis following infection with Epstein-Barr virus. Eur J Paediatr Neurol 2003; 7: 413–415.
21. Ooi MH, Wong SC, Clear D, et al. Adenovirus type 21-associated acute flaccid paralysis during an outbreak of hand-foot-and-mouth disease in Sarawak, Malaysia. Clin Infect Dis 2003; 36: 550–559.
22. Verheulpen D, Ribai P, Gerard JM, et al. Brachial plexus neuritis: is prognosis worse in children? Eur J Paediatr Neurol 2004; 8: 165–166; author reply 166.
23. Yilmaz C, Caksen H, Arslan S, et al. Bilateral brachial plexopathy complicating Henoch-Schonlein purpura. Brain Dev 2006; 28: 326–328.
24. Kotsopoulos I, Faber K, Raaijmakers J, et al. Idiopathic neuralgic amyotrophy in childhood. Neuropediatrics 2007; 38: 36–37.
25. Al Masri O, Fathallah W and Quader S. Recovery of tacrolimus-associated brachial neuritis after conversion to everolimus in a pediatric renal transplant recipient–case report and review of the literature. Pediatr Transplant 2008; 12: 914–917.
26. Yamada K, Mano T, Toribe Y, et al. MRI findings and steroid therapy for neuralgic amyotrophy in children. Pediatr Neurol 2011; 45: 200–202.
27. Mrowczynski OD, Langan ST and Rizk EB. Infant brachial neuritis following a viral pro- drome: a case in a 6-month old child and review of the literature. Childs Nerv Syst 2018; 34: 173–176.