Otology

Pseudotumoural hypertrophic neuritis of the facial nerve

Neurite ipertrofica pseudotumorale del nervo facciale

E. ZANOLETTI, A. MAZZONI, R. BARBÒ[1]
ENT Unit, Department of Neurologic Sciences and Neurosurgery; [1] Neuroradiology Unit, Department of Neurologic Sciences and Neurosurgery, Ospedali Riuniti, Bergamo, Italy

SUMMARY

In a retrospective study of our cases of recurrent paralysis of the facial nerve of tumoural and non-tumoural origin, a tumour-like lesion of the intra-temporal course of the facial nerve, mimicking facial nerve schwannoma, was found and investigated in 4 cases. This was defined as, pseudotumoral hypertrophic neuritis of the facial nerve. The picture was one of recurrent acute facial palsy with incomplete recovery and imaging of a benign tumour. It was different from the well-known recurrent neuritis, hypertrophic neuropathy and perineuroma. A portion of the intra-temporal course of the nerve with concurrent dilatation of the osseous walls was diagnosed pre-operatively as facial nerve schwannoma. The pathological picture showed inflammatory hypertrophy which was not a schwannoma but was likely of viral origin, with degeneration-regeneration of fibres and new connective tissue. Resection of the involved portion of the facial nerve and autologous graft in two cases was performed, decompression with biopsy in the other two. No recurrence of new episodes of paralysis after surgery was observed.

KEY WORDS: Facial nerve • Facial nerve paralysis • Hypertrophic neuritis • Hypertrophic neuropathy • Facial nerve pseudotumour

RIASSUNTO

In uno studio retrospettivo della nostra casistica sulle paralisi ricorrenti del VII nervo cranico con eziologia tumorale e non, una nuova forma di neurite ipertrofica del nervo facciale è stata osservata in 4 casi. Tale forma è stata definita "Neurite pseudotumorale ipertrofica del nervo facciale". Il quadro clinico era di una paralisi acuta ricorrente del VII nervo cranico, con un recupero incompleto, l'aspetto radiologico di un tumore benigno con dilatazione del canale osseo di un tratto intratemporale del nervo facciale, ma istopatologicamente differente dallo schwannoma e dalle forme note di neuriti ipertrofiche, quali la neuropatia ipertrofica ed il perineuroma. Dopo rimozione del nervo ed un graft autologo in 2 casi ed una decompressione con biopsia negli altri 2, il quadro anatomo-patologico mostrava una ipertrofia infiammatoria pseudotumorale ma di probabile origine virale, diversa per caratteristiche dalle neuriti ipertrofiche e dalle lesioni tumorali, benché il quadro radiologico fosse indistinguibile.

PAROLE CHIAVE: *Nervo facciale • Paralisi del nervo facciale • Neurite ipertrofica • Neuropatia ipertrofica • Pseudotumore del nervo facciale*

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Introduction

The theory of the viral origin of idiopathic facial nerve palsy has gained ground following both experimental and clinical studies [1-4]. The viral theory implies that facial neuritis originates from *Herpes Simplex Virus* (HV) and *Varicella Zoster viruses* (HZV) and, to a lesser extent, Cytomegalovirus (CMV) and Borrelia which accumulate in the rhinopharyngeal mucosa and progress centrally along the nerves [1]. They stay in latency in the geniculate and meatal ganglion, until an increase of viruses and a decrease in the host's defenses allow reactivation of the infection. A viral ganglionitis occurs as the main pathogenetic mechanism of the neuritis and successively progresses to the other portions of the nerve.
Bell's palsy and Ramsay Hunt syndrome are the clinical expression of the viral ganglionitis [1 3 5], diagnosis being based on the assessment of the motor and sensory facial nerve loss and a positive enhancement of the intrameatal and labyrinthine tracts of the nerve at magnetic resonance imaging (MRI) [6-8]. Positive IgM serology, specific for HV or HZV and viruses in the saliva, have been found in the acute phase of the infection [5 9 10].
According to the literature, 4-7% of idiopathic facial palsy may recur at any age, at variable intervals of time, with inconstant number of episodes and a not easily predictable prognosis [1 3 11-13]. The pathogenetic mechanism of the recurrent facial palsies has not been well established, meatal and geniculate ganglionitis seems to be the underlying process but there are some aspects, such as the predictability of the recurrence, the evolution of the palsy, the therapy and details regarding pathology, which are debated [11-15].

The diagnosis of recurrent palsy is essentially clinico-radiological. Imaging shows the typical enhancement of a viral neuritis, which may persist for a long time even after the clinical signs have disappeared. There is generally no correlation between the positive enhancement and the recovery of the nerve.
In the present report, four cases are described of another type of recurrent facial palsy with a possible neuritic aetiology, a tumour-like imaging and a picture of localised inflammatory hypertrophy at pathology. In all these cases, a tumour was suspected pre-operatively. In two cases, a portion of the nerve was removed since the intra-operative findings still suggested a tumour, while in the other two cases, only a biopsy of the inflammatory hypertrophic tissue was obtained. We adopted, for this new entity, the descriptive term of "pseudotumoural hypertrophic neuritis".

Case reports

Case no. 1

A 38-year-old male presented with a 6 months' history of 2 recurrent episodes of left facial nerve paralysis, with sudden onset, each lasting more than one month. Five months before the paralysis, an ipsilateral tinnitus began and audiometry showed a high-frequency loss of 40 dB. At the first episode, with House-Brackmann 4 (HB) grading system paralysis, steroid therapy was promptly started. Recovery began one month after the advent of the clinical signs and led to 3 HB paralysis in 2 months. After 3 months, a new HB 5 paralysis occurred and began to recover after one month, until a HB 4 facial nerve function was achieved in 2 months. Three months after the second episode, computed tomography (CT) and MRI demonstrated an enhancing, tumour-like mass in the geniculate ganglion and the labyrinthine tract of the facial nerve. The bony walls of the fallopian canal were eroded around the mass without signs of infiltration. Surgery, with the middle cranial fossa approach, was performed 12 months after the first episode. The finding was a tumour-like enlargement of the geniculate ganglion, which was found to be an inflammatory proliferative lesion at the intra-operative histological examination. Removal of the mass, with the nerve, was performed, followed by sural nerve graft between the labyrinthine and tympanic tract.
Definitive histological examination showed chronic inflammatory cells, fibrosis due to proliferation of fibroblasts in the nerve. This pattern was defined as "pseudotumoural hypertrophic neuritis".
No recurrence of paralysis was recorded throughout the clinico-radiological follow-up, the last MRI being negative 9 years after surgery. Recovery of the nerve occurred within the first two years, at a HB 3 degree.

Case no. 2

A 22-year-old male presented with a life history of recurrent paralysis of the facial nerve, at the left side, and no associated symptoms. The first episode which occurred at the age of 2 months and was HB 4, lasted more than one month and recovered completely 2 months after onset. The second and the third episodes, both HB 4, occurred at the age of 2 and 4 years, respectively, each lasted one month and the nerve recovered up to HB 1 within 3 months. At the age of 10 years, the fourth recurrence occurred with HB 5 with a partial recovery to HB 3. The fifth episode occurred at the age of 20 years with a HB 5 paralysis. After 3-4 months, the facial nerve function was HB 4 and remained stable for the following 2 years. Imaging showed an enhancement in growth of the mastoid tract of the nerve, with enlargement of the bony canal (Figs. 1, 2). The mass harboured a cyst, containing fluid, half its diameter in width. The lesion was thought to be a schwannoma of the VII cranial nerve. It was removed with the mastoid portion of the nerve and a graft with the great auricular nerve was placed between the second genu and the extra cranial tract. Histological examination revealed an accumulation of inflammatory cells within a hypertrophic

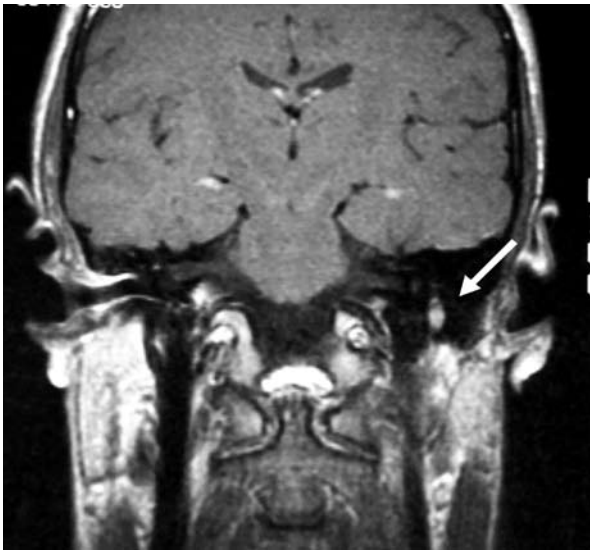

Fig. 1. Case 2: contrast enhanced T1 coronal MRI. Enhancing mass in third portion of left Fallopian canal.

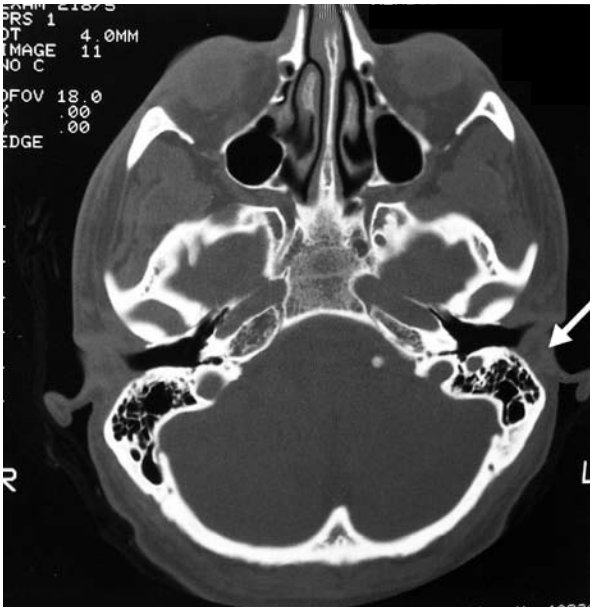

Fig. 2. Case 2: high resolution axial CT. Enlargement with clear margins of bony walls of third portion of left Fallopian canal.

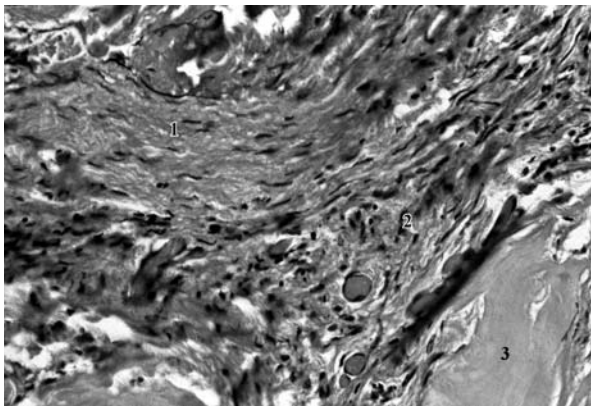

Fig. 3. Case 2: pathological specimen of nerve with hypertrophic and inflammatory pattern: nervous fibres (1), lymphocyte aggregates (2), collagenous fibres (3).

mass of fibroblasts. No tumour was found, the diagnosis was "Pseudotumoural hypertrophic neuritis" (Fig. 3). At 9 years follow-up, no recurrence of paralysis had occurred, MRI was negative and the facial nerve was HB 3.

Case no. 3

A 15-year-old male had a one-year history of 2 recurrent episodes of acute right facial nerve paralysis, each lasting one month, with an interval of 6 months between the two. The facial function was a HB 5, at the first and second episode, with a recovery to HB 4. Ipsilateral fasciculation and spasm at the *orbicularis oris* muscle appeared with the first episode and did not resolve until surgery. Moderate ipsilateral neurosensory hearing loss was present but no other accompanying symptoms. High dose steroids were administered after each episode. MRI, one year after the onset, showed an enhancing lesion in the internal auditory canal and enlargement of the bony walls without signs of active destruction. The diagnostic hypothesis was schwannoma. The intra-operative finding was a cystic lesion of the nerve. The cyst was removed without sacrificing the nerve. Histological examination described an arachnoid mass around nerve fibres with cells of inflammation (lymphocytes, macrophages) and a proliferation of fibroblasts. The diagnosis was "pseudotumoural hypertrophic neuritis" with a secondary cyst.
No recurrence of paralysis occurred during the 8 years' follow-up, the post-operative facial nerve function improved from 5 to 4 within 2 years and remained stable.

Case no. 4

A 9-year-old girl presented with a history of 2 acute recurrent facial palsies on the right side. A paralysis of HB 4 degree was observed in the first episode, which recovered to HB 2 in a month. The second episode occurred 2 months later, with a HB 4 paralysis recovering to a HB 3. Steroid therapy was administered in both episodes. The clinico-radiological diagnosis was geniculate ganglion schwannoma, as a localised enhancing mass with a benign aspect on the geniculate ganglion was revealed at MRI and CT, a localised enlargement of the Fallopian canal on the geniculate ganglion area and the labyrinthine tract, with a clearcut profile of the bone. Hearing was normal. Surgery was performed 2 months after the last episode, with a subtemporal access. The facial nerve was exposed from the labyrinthine to the tympanic tract. The geniculate ganglion was found to be homogeneously enlarged with oedema and obvious fascicles but no signs of tumour. Because of this picture and the slight paralysis, a biopsy was made only from the thickened perinevrium without damaging the nervous bundles. At histological examination a rich infiltration of lymphocytes was found, compatible with the regression phase of an inflammation. After surgery, no recurrence of paralysis was observed, facial function was HB 2, at 2 years of follow-up, and MRI was negative.

Discussion

Clinico-radiological picture

We described 4 cases of facial paralysis presenting with a similar picture of clinical, radiological, pathological and therapeutic aspects. This form of facial paralysis can be differentiated from both the tumour and non-tumour lesions.
The clinical picture involved a recurrent facial paralysis with acute onset, occurring at any age, and complete or partial regression within two months. A high frequency hearing loss occurred in 2 cases, with no other associated symptoms. The recurrence of paralysis occurred after a variable time (2 months - 2 years) with acute onset and a partial recovery to HB 3-4. Recovery to normal was not achieved with anti-viral and steroid therapy.
Imaging demonstrated an enhancing mass in the intratemporal course of the facial nerve, with an additional cystic component in 2 cases, and a concurrent dilation with a clearcut profile of the Fallopian canal or the internal auditory canal around the lesion. The lesion was centered on the geniculate ganglion in 2 cases, on the distal half of the internal auditory canal in one and the mastoid portion of the Fallopian canal in the other case. Surgery showed a localised hypertrophy of a portion of the nerve, with no possibility to separate the mass from the trunk. The Fallopian canal and the internal auditory canal were enlarged without signs of active osteolisis. At pathology, a thick fibrous mass was observed and, in 2 cases, a clear fluid cyst. A rich infiltrate of inflammatory cells was detected, with lymphocytes B and vessel proliferation, a reduced number of normal nervous fibres and degenerating-regenerating axons. The picture was one of chronic, aspecific hypertrophic neuritis. While the increase in mass of the nerve finds its correlate in this hypertrophic neuritis, the bone process of dilation could be a periostitis with a lytic-repair process of the bone.
The full picture of an acute recurrent paralysis, localised nerve and bone wall enlargement and a pathology of chronic, aspecific, hypertrophic neuritis was present in 2 cases, in which a nerve specimen was available. In the other 2 cases, the intra-operative finding was not typical of tumour and only a biopsy of the perineurium and the cyst wall was obtained. The inclusion of these cases within the pseudotumoural hypertrophic neuritis was supported by the clinico-radiological features as well as by the findings of a chronic aspecific inflammation around the nerve.

The portion of the nerve involved by the tumour-like mass was resected and replaced with an autologous graft in 2 of the cases. Recovery from the paralysis occurred within 6-12 months up to a HB 3 in 2 of the cases and to HB 4 in one case, and amounted to an improvement of one degree of the House-Brackmann scale. A HB 2 case remained unchanged.
The mean follow-up was 7 years. No recurrence of paralysis was observed and MRI was normal after 9, 8, 9, 2 years, respectively.

Differential diagnosis

The differential diagnosis of the 7th nerve paralysis with a localised mass increase of the nerve is related to tumour and non-tumour conditions. The tumours are schwannoma, neurofibroma, solitary paraganglioma and metastasis, while the non-tumour conditions are localised hypertrophic neuropathy, perineuroma and the present form of pseudotumoural hypertrophic neuristis. Peripheral facial paralyses of non-tumuoral aetiology and no signs of hypertrophy, at imaging, are represented by viral and recurrent viral neuritis.
The differential diagnosis between the different forms of recurrent paralysis with mass increase includes the clinical picture, imaging, laboratory tests on biological fluid and pathology. Also the treatment, either medical or surgical, the outcome and long-term follow-up may involve a difference [11-16]. High dose steroids are given as medical treatment, while the surgical option includes partial or total decompression of the nerve, or removal of the tumour-nerve mass followed by a graft. The absence of recurrence of paralysis may reveal the success of the treatment.
"Hypertrophic neuropathy" is characterised by focal proliferation of fibroblasts disposing in layers in an onion-bulb shape. The pathogenetic mechanism involves continuous processes of myelinization and demyelinization of the nerve, though it is not clear whether this is a primary phenomenon or the consequence of recurrent inflammation [17-19]. Ultrastructural findings show a positive reaction to S-protein and absence of reaction with the epithelial antigens of the membrane, which indicates a proliferation of non-Schwann cells. Hypertrophic neuropathy exists in a "diffuse" pattern, which is often associated with chronic processes of congenital or acquired demyelinization occurring either at distant or contiguous portions of the same nerve, and a "localised" pattern which is more commonly associated with neuropathies of the brachial plexus and is rarely mentioned for the V, VII and VIII cranial nerves [17 18].
The "perineuroma" shows a proliferation of perineural cells and Schwann cells, shaping in an onion-bulb architecture, with a positive reaction to the epithelial antigens of the membrane and absence of response to protein S-100. These ultrastructural findings make the difference with the hypertrophic neuropathy. Perineuroma may occur primarily or be secondary to recurrent inflammation [19-22]. The Fallopian canal is enlarged both in hypertrophic neuropathy and perineuroma. Histological evidence for a positive diagnosis is necessary.
The clinical course of the paralysis is slowly progressive, with rare episodes of quick progression, in schwannoma, neurofibroma, paraganglioma and metastasis, it is progressive in the few cases of hypertrophic neuropathy and perineuroma described [17 20-22]. In our cases of pseudotumoural neuritis, the paralysis presented acute episodes with partial recovery and no slow progression. The location tends to be close to the stylomastoid foramen or the geniculate ganglion in metastasis. Hypertrophic neuropathy shows a long extension along the nerve.
Pseudotumoural hypertrophic neuritis appears to present the typical combination of acute attacks, partial regression and no progression, with an imaging of localised mass increase on any site of the bony course of the nerve. The small number of known cases does not allow this to be considered a new clinical entity, amenable to a clinico-radiological diagnosis and the histological examination is necessary. The rate of pseudotumoural neuritis may be underestimated, as cases with slight paralysis or advanced age are preferably submitted to observation and diagnosis at pathology is not obtained.

Pathogenesis, diagnosis and treatment of "pseudotumoural hypertrophic neuritis"

The theory of a viral aetiology of recurrent hypertrophic facial paralysis is not supported by serologic and morphologic studies as for viral neuritis, nevertheless it seems the most likely explanation for this form of paralysis. The viral neuritis of the facial nerve originates from HV, HZV, and rarely from CMV and Borrelia. The viruses stay in latency in the geniculate and meatal ganglion, until an accumulation of viruses and a decrease in host defences allow reactivation of the infection. The viral ganglionitis extends to the other portions of the nerve. An increased serous IgM, specific for HSV and HZV, as well as the virus itself in the saliva have been reported [1 3 4 9 10]. Despite their importance, further studies are necessary to confirm clinical relevance. The standard range of the serous level of the specific antibodies has to be established as well as their impact on diagnosis and treatment.
Bell's palsy and Ramsay Hunt syndrome are considered the clinical picture of this viral neuritis. Paralysis may recur at any age, with a 4-7% rate, with unpredictable episodes and prognosis.
If a viral aetiology is accepted, for the pseudotumoural hypertrophic facial nerve paralysis, the mass increase of the nerve has to be explained. The reactivation of the infection and the duration of the inflammatory changes would produce an accumulation of inflammatory cells, fibroblasts and macrophages with a localised peri- and intra-neural enlargement. The bony walls would be affected by a lytic and repair process [1 11-13 18 19]. It is, however, obvious that the specific mechanism differentiating the hypertrophic from the plain neuritis remains obscure and the concept of hypertrophic neuritis as a variation of the common neuritis needs further evidence.
The treatment of pseudotumoral neuritis is confronted with the pathological changes as well as the forseeable recurrence of the viral lesion. The choice of treatment involves an interaction between the clinical factors and the therapeutic options. The clinical factors are the recurrent paralysis with incomplete recovery, the degree of paralysis, the imaging of a localised mass with enlargement of its bony case. The therapeutic options range from medical treatment to more or less aggressive procedures such

as decompression, removal of the mass with nerve preservation, radical removal of the tumour-nerve mass with nerve graft. Considering the pros and cons of each option is a step of the decision.
The observation and medical treatment, in the case of recurrence, present the incovenience of a dubious or complete lack of improvement, no conclusive diagnosis and the risk of a further recurrence for which the treatment has proven to be unsuccessful. We believe that observation alone may be discussed in the case of slight paralysis but with the readiness to switch to another option.
Decompression as used in recurrent viral neuritis shows no evidence of benefit [14 15] and leaves a lesion which is unknown and interferes with fibre regeneration.
Removal of a visible growth, if at all possible, may damage the nerve and leave pathological tissue with the potential of recurrence [16]. It seems a convenient option when a lesion with partial paralysis can be resected and the nerve preserved.
The complete removal of the tumour-nerve mass with nerve graft eliminates the inflammatory tissue that represents an obstacle to the regeneration of the fibres and allows their physiological repair. It remains to be assessed whether nerve replacement prevents further recurrences by removing the site of silent viruses. Our experience seems to support tumour-nerve removal + graft in the cases with HB 3 paralysis or worse [13]. The HB 2 cases present the dilemma of each one of the mentioned options. The choice of treatment depends on several factors. In old age there is less chance of a successful graft. The degree and duration of both the current and the previous paralyses allow the regeneration potential of the nerve to be foreseen. Operative finding of a tumor-like mass *vs*. oedema, the technical difficulty of placing a nerve graft are other factors for decision.

Conclusions

A tumour-like lesion of the intra-temporal course of the facial nerve was studied in four clinical cases and called recurrent pseudo-tumoural hypertrophic neuritis of the facial nerve. The picture was one of a recurrent acute facial palsy with incomplete recovery after the second episode and imaging of a benign tumour of the nerve. There was a localized enhancing growth of the nerve with a consensual dilation of the bony wall of the Fallopian or the internal auditory canal. The histological findings were consistent with chronic aspecific hypertrophic neuritis. The origin was not investigated and was considered to be viral since it was analogous with the facial neuritis.
Other hypertrophic neuropathies of the facial nerve are known and in their localised form may be misdiagnosed as a benign tumour or pseudotumoural neuritis. The differential diagnosis was mainly with the schwannoma and was established only following histological findings on the surgical specimen. The recurrent acute paralysis and the laboratory tests for a viral infection, although significant in principle, were not considered decisive for a positive diagnosis.
The treatment, in two cases, was resection of the tumour-nerve mass with nerve grafting, allowing a functional recovery of the degree expected in a nerve graft in a tumour condition. Plain decompression was adopted in one case of predominantly cystic change of the nerve and in one case of inflammatory hypertrophy of the geniculate ganglion. The picture of acute recurrence and no slow progression, localised enlargement of the facial nerve and chronic inflammatory hypertrophy are typical features, although it is not still a matter of discussion whether this can be considered a new clinical entity.

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Address for correspondence: Dr. E. Zanoletti, U.S.C. ORL, Ospedali Riuniti, largo Barozzi 1, 24121 Bergamo, Italy. Fax +39 035 269288.
E-mail: elisabetta.zanoletti@tiscali.it - antonio.mazzoni@libero.it