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Case Report

Intraneural perineurioma affecting multiple nerves: a case report and literature review

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Abstract: Intraneural perineurioma is a neoplasm of perineurial cells, corresponding to WHO grade I. We present a case of intraneural perineurioma affecting multiple nerves, which usually involved one or two of major nerve trunks in one patient. We describe the clinical presentation, magnetic resonance (MR) neurography characteristics, and pathological characteristics. The differential diagnosis with other diseases, such as neurofibroma, Schwannomatosis and HNPP, will also be discussed. We also review the literature in efforts to highlight recent studies on intraneural perineurioma and heighten and awareness for the possible presentations of this disorder.

Keywords: Intraneural perineurioma, neuropathology, pseudo-onion bulbs, MR

Introduction

Perineurioma is divided into two forms: intraneural and extraneural (soft tissue) perineurioma. Intraneural perineurioma is a benign peripheral nerve sheath tumor of perineurial cell origin, first histologically identified by Imaginario in 1964 [1]. Intraneural perineurioma is an uncommon proliferation of perineural cells that form an onion bulb-like shape. These lesions are frequently misdiagnosed due to their rarity [2]. The disease progression is slow with increased involvement of the nerve, associated motor and, less frequently, sensory deficits. Previously published cases have involved one or two major nerve trunks, with less involvement in multiple nerves [3, 4]. We present a case of intraneural perineurioma affecting the cervical plexus, brachial plexus, intercostal nerves, spinal roots, sciatic nerve, nerves of extremities and etc.

Case report

History

A 22-year-old female presented with a 10-year history of paroxysmal acroanesthesia primarily localized in in distal limbs and accompanied with aching. There was no history of trauma and no family history of any neuromuscular symptoms. There was, however, the possible history of "infection of the upper respiratory tract" before the initial development of symptoms. Clinical examination revealed weakness and hypoesthesia of the limbs. There was some improvement following traditional Chinese acupuncture. Acroanesthsia appeared again five months prior to her examination and was accompanied by weakness of the ulna muscle of the left hand.

Examination

The left thumbnail was smaller than the right, suggesting that her weakness was long-standing. General physical examination was normal. Nerve conduction studies demonstrated velocity was reduced in bilateral median, ulnar, radial, tibial and sural nerves. There was no F wave in bilateral median and ulnar nerves and no H wave was recorded in the bilateral tibial nerves. MRI demonstrated enlargement of the nerves and abnormal hyperintense signal on the T2-weighted images including the cervical plexus, brachial plexus, intercostal nerves, spinal roots, sciatic nerve, and etc (**Figure 1**).

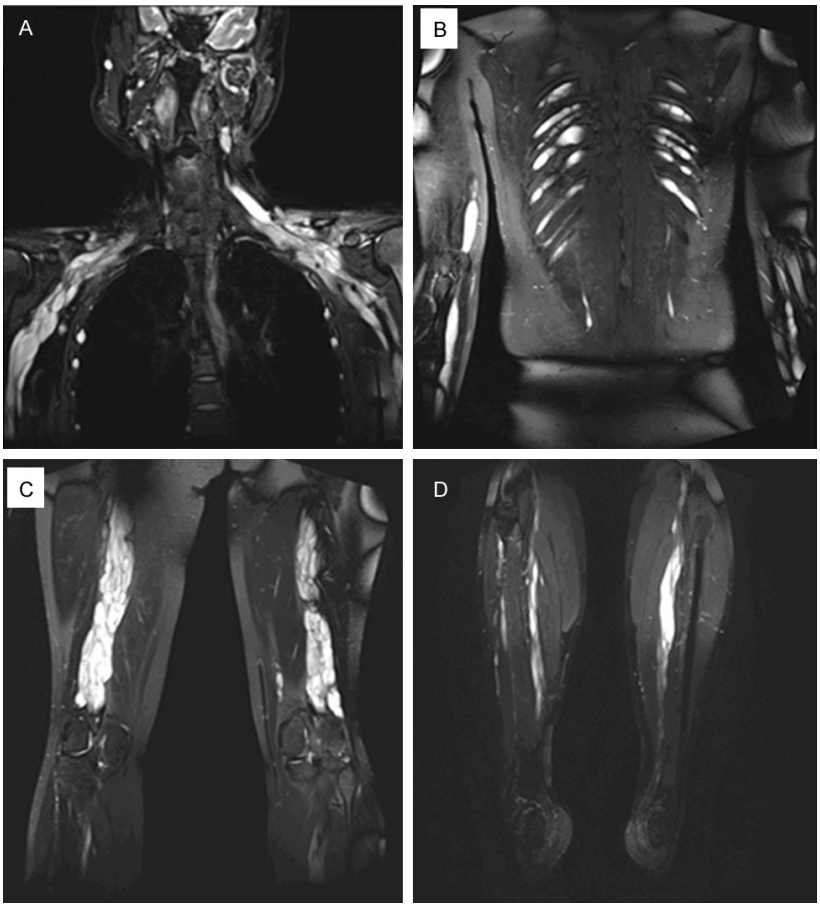

Figure 1. MRI demonstrated enlargement of the nerves and abnormal hyperintense signal on the T2-weighted images, including the cervical plexus (A), intercostal nerves (B), sciatic nerve (C), nerves of extremities (D).

Pathological findings

A biopsy of the right sural nerve was immersed in 10% buffered formalin and embedded in paraffin for processing and histopathological examination. Hematoxylin and Eosin (H&E) sections (**Figure 2A**) showed circumscribed lesions surrounded by fibrous connective tissue composed of layers of spindle-shaped cells around a central axon, forming structures described as pseudo-onion bulbs. These structures were also demonstrated in Epoxy section. Immunohistochemical staining showed that the perineurial cells were positive for epithelial membrane antigen (EMA, 1:100, Dako) (**Figure 2B**), collagen IV (1:100, Dako), CD34 (1:50, Dako) (**Figure 2C**), and the Schwann cells were positive for S-100 (1:100, Dako) (**Figure 2D**). Staining was negative for Myelin Basic Protein (1:50, Dako), P53, Smooth Muscle Actin (SMA), Cytokeratins (AE1/AE3) (1:200, Zymed), Laminin, Ki-67 (1:500; Dako).

Transmission electron microscopy (JEM-1400, Japan) showed concentric arrangements of perineurial cells, identified by their long, thin cytoplasmic processes, numerous pinocytotic vesicles, and fragmented basal lamina around a central axon with an accompanying Schwann cell (**Figure 2E**).

FISH analysis of paraffin sections was performed on the locus of chromosome 22 (probe, Vysis LSI EWSR1, USA). Slides were analyzed in a fluorescent (Olympus BX51) microscope with a triple band pass filter. A deletion of the locus (22q11) was negative (**Figure 3**). We then investigated the possibility of suffering from HNPP (hereditary neuropathy with liability to pressure palsy), which results from the loss of one copy of PMP22. The chromosome 17p11.2-p12 deletion was determined using seven microsatellite markers mapping described [5, 6]: D17S2217, D17S2218, D17S2220, D17S2226, D17S4A, D17S9A, and D17S9B. The result demonstrates no deletion on 17p11.2-p12, thereby excluding the possibility of HNPP (**Figure 4**). In order to distinguish with neurofibroma, the gene of NF2 was also detected. The PCR products of the exon2 and exon3 of NF2 gene was sequencing and analyzed by ABI3700DNA sequencer. Our results showed no mutations in exon2 or exon3 of NF2. These immunohistochemical, electron microscopic findings and molecular studies were consistent with H&E staining and all indicated perineurioma.

Discussion

Intraneural perineurioma is a neoplasm of perineurial cells, histologically corresponding to World Health Organization grade I [7]. This rare lesion accounts for about 1% of nerve sheath

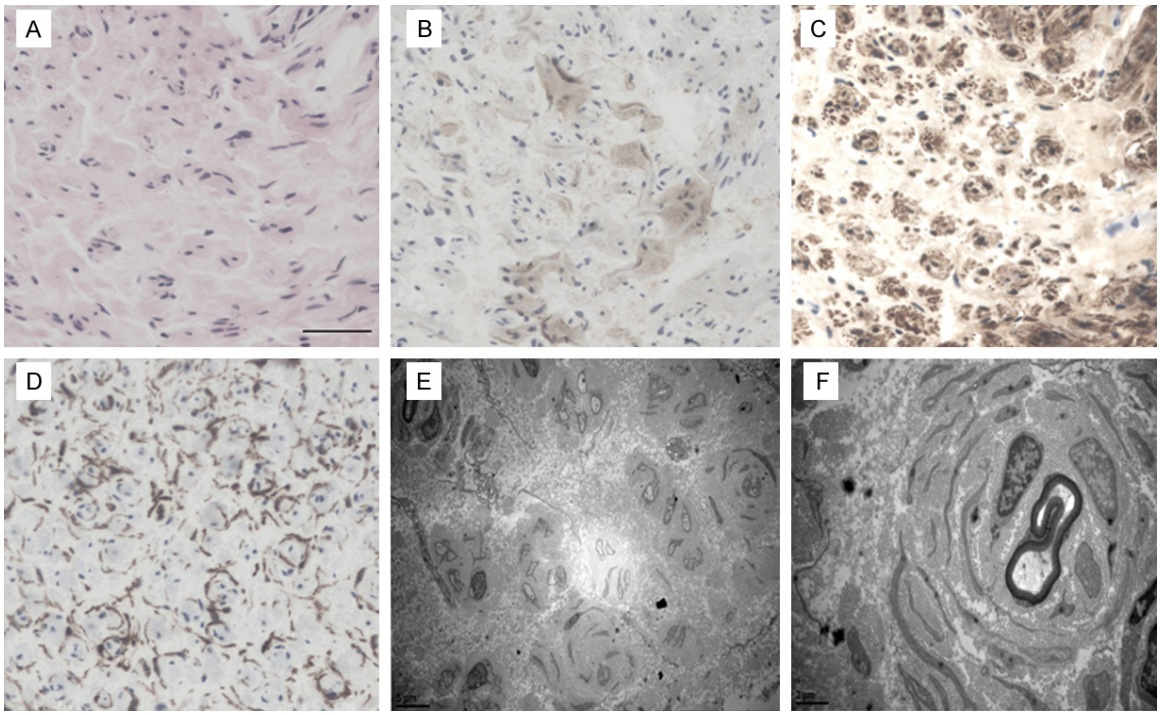

Figure 2. A: Hematoxylin and Eosin stain. Pseudo-onion bulbs structures with central axons were surrounded by swirls of spindle-shaped cells. B: Epithelial membrane antigen (EMA) positive staining shows pseudo-onion bulbs consisting of perineurial cells. C: S-100 staining highlights scattered Schwann cells. D: CD34 positive staining. Original magnification: ×400. E, F: Electron microscopy ultrastructure shows perineurial cells in a concentric arrangement, (E: ×3000; F: ×8000).

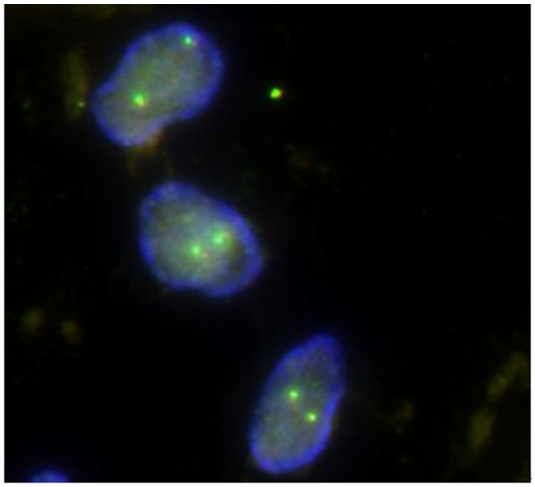

Figure 3. The chromosome of 22q11 was observed using EWSR1 hybridization. FISH image: none of the nuclei is monosomics (×1000).

neoplasm. The typical age of onset of intraneural perineurioma is either adolescence or young adulthood, and no sexual predilection has been demonstrated. The symptomatic presentation is usually a painless mononeuropathy with progressive weakness in the affected muscles. To date more than 120 cases of intraneural perineurioma have been reported [1-39] (**Table 1**). The affected patients are 2 to 73 years old, with the majority of cases reported in young persons (**Figure 5**). Involvement of the disease is usually limited to a single major nerve and is associated with a motor deficit (**Table 2**). Two cases were described by Emory and Nancy involving different nerves [3, 4]. Only one case has described the involvement of multiple nerves in a young patient [34]. The current report of intraneural perineurioma that we have described affected the cervical plexus, brachial plexus, intercostal nerves, spinal roots, sciatic nerve and etc. This is helpful to heighten the awareness of the potential presentations of intraneural perineurioma in multiple nerves.

Intraneural perineurioma can be misdiagnosed as other lesions, such as hypertrophic neuropathy, schwannoma and neurofibroma. The pseudo-onion bulbs of intraneural perineurioma are morphologically similar to the onions bulbs characteristic of Charcot-Marie-Tooth Disease

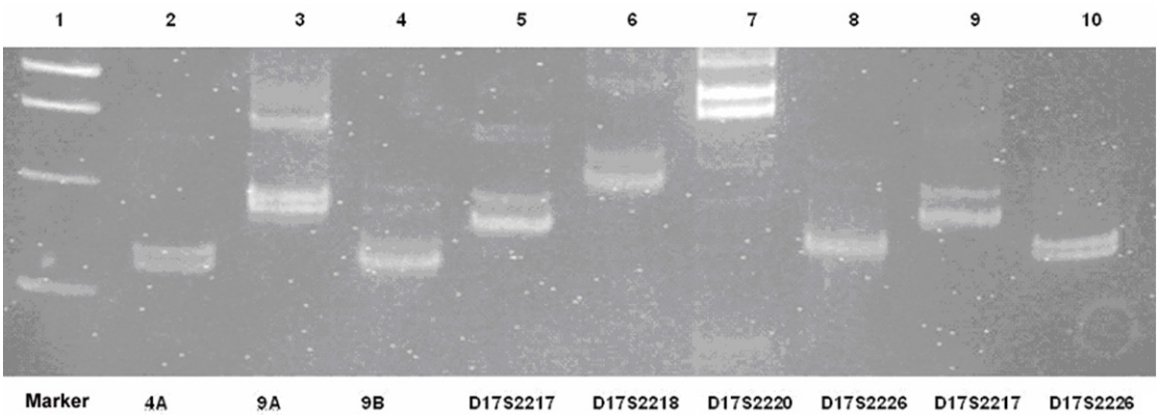

Figure 4. 1: Marker; 2-8 (patient): D17S4A, D17S9A, and D17S9B, D17S2217, D17S2218, D17S2220, D17S2226, ladder 9-10 (Normal control): D17S2217, D17S2226. 2-7: Each of markers shows two bands. There is no deletion on 17p11.2-p12.

Table 1. Intraneural perineuriomas reported after 1995

Authors	Age (y)	Sex	Affected nerves	Size (cm)	Immunohistochemical staining
Gruen, et al [18]	4	F	Median	4	EMA (+), S-100 (-)
	14	F	Peroneal	8	
	8	M	Peroneal	9.4	
	4	M	Median	2	
	12	M	Sciatic	-	
	38	F	Median	-	
	21	F	Brachial plexus	4.5	
	16	F	Peroneal	5	
	37	M	Ulnar	-	
	37	M	Radial	-	
	16	M	Median	3.5	
	38	F	Radial	3.81	
	8	F	Peroneal	6.35	
	43	F	Peroneal	8	
Simmons, et al [33]	17	F	Femoral	12	EMA (+), S-100 (Sc+)
	12	F	Sciatic	12	
	26	F	Brachial plexus	4	
	19	M	Brachial plexus	-	
Jazayeri, et al [23]	53	M	Median	5×4	EMA (+), S-100 (Sc+), P53(-)
Heilbrun, et al [20]	28	F	Peroneal	4×4	EMA (+), S-100 (+)
Alfonso, et al [8]	2	F	Median	1×1	EMA (+), S-100 (-)
Douglas, et al[15]	26	F	Unnamed nerve	1×0.75	EMA (+), S-100 (+)
Huguet, et al [21]	64	M	Dentary nerve	2×1.5	EMA (+), Vimentin (+), S-100 (Sc+), NF (+) in axons
Isaac, et al [22]	2	F	Radial	2	EMA (+), S-100 (-)
Cortes, et al [13]	9	F	Radial	3.5	EMA (+), S-100 (-)
Merlini, et al [28]	7	F	Sciatic	-	EMA (+)
Nancy, et al [3]	5	M	Radial	-	EMA (+), S-100 (-)
	12	M	Brachial plexus	-	EMA (+)
	14	M	Tibial and peroneal	2	EMA (+), Amyloid (-)
Nguyen, et al [30]	30	F	Radial	-	EMA(+), S-100(-)
Smehak, et al [35]	15	F	Laryngeal nerve	4	EMA (+), CD56 (+), vimentin (+), S-100 (-), CD34 (-)
Rocha, et al [31]	47	F	Lingual apex	1	EMA (+), S100 (Sc+)
	36	M	Lower lip	0.5	

Vencio, et al [36]	59	F	Dental	-	EMA (+), S-100 (-), NF (-),
Lee, et al [24]	16	M	Sciatic	-	EMA (+)
	11	F	Peroneal	0.5	
Mauermann, et al [27]	12	F	Sciatic	1.3×15	EMA (+), S-100 (-)
	7	M	Sciatic	1.5×12	
	2	F	Lumbosacral plexus	1.5×12	
	12	F	Sciatic	4×0.8	
	35	M	Tibial	16×0.7	
	31	M	Sciatic	8	
	15	F	Sciatic	>25	
	56	M	Radial	15	
	19	F	Femoral	-	
	40	M	Median	4.5	
	7	F	Sciatic	6.5	
	40	M	Trigeminal	5	
	34	M	Sciatic	23.5	
	12	F	Brachial plexus	9.7	
	35	M	Brachial plexus	15	
	30	F	Radial	12	
	14	M	Sciatic	32	
	30	M	Sciatic	5	
	12	M	Brachial plexus	14	
	34	M	Radial	3	
	8	F	Sciatic	3.5	
	19	F	Ulnar	6	
	25	F	Ulnar	2.5	
	38	F	Radial	-	
	15	M	Median	-	
	11	F	Brachial plexus	-	
	13	F	Peroneal	6	
	35	F	Ulnar	-	
	8	M	Sciatic	6.8	
	13	F	Ulnar	7.8	
	37	M	Tibial	7.2	
	12	M	Sciatic	20	
Sachanandani, et al [2]	23	F	Median	-	EMA (+), S-100 (-)
Cornelis, et al [12]	4	M	Median	1.5×4	claudin-1 (+), laminin (+), s-100 (-), glut1 (-), NF (-)
Lequint, et al [25]	12	F	Brachial plexus	-	-
Li, et al [26]	23	F	Facial	-	EMA (+), S-100 (-)
Almefty, et al [9]	27	F	Third cranial nerve	1.5	EMA (+), S-100 (-)
Christoforidis, et al [11]	59	F	VIIIth cranial nerve		EMA (+), S-100 (-)
Siponen, et al [34]	18	F	Multiple orofacial		EMA (+), S-100 (-)
Miyahara [29]	11	F	Sciatic	-	EMA (+), S-100 (-)
Cruz [14]	12	M	Tongue	0.6	EMA (+), S-100 (-)
Boyanton [10]	6	F	Tongue	0.8×0.6	EMA (+), S-100 (-)
	5	M	Ulnar	1.5	
Santos, et al [32]	42	M			EMA (+), S-100 (-)
Dundr, et al [16]	16	M	Mucosa	1.5×1	EMA (+), S-100 (-)
S Yamada, et al [38]	30	F	Nose	0.5×0.3	EMA (-), S-100 (-), Glut-1 (+)
HY Gu, et al [19]	47	F	Tongue	1.5	EMA (+), CollagenIV (+), S-100 (-), CD34 (-)
WL Xiao, et al [37]	46	F	Tongue	1.1×1.1	EMA (+), S-100 (+), CD34 (+)

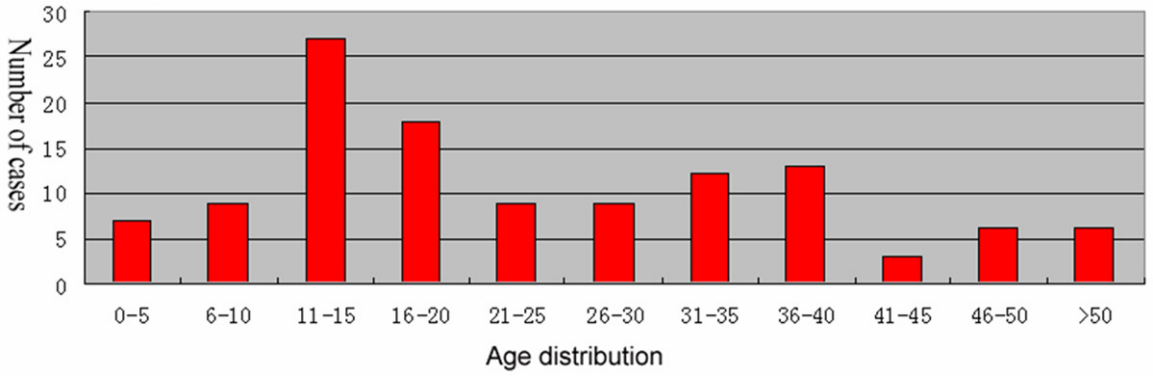

Figure 5. Age distribution of cases of intraneural perineurioma reported in the literature.

Table 2. Conclusion of affected nerves in reported cases of intraneural perineurioma

Affected nerve	No. of cases
Sciatic	21
Radial	15
Median	14
Brachial plexus	12
Peroneal	9
Ulnar	8
Tibial	7
Tongue	7
Posterior interosseous	5
Femoral	3
cranial nerve	2
Spinal roots	2
Dental	2
Facial	2
Digital	1
Mandibular	1
Acoustic	1
Popliteal	1
Sacral roots	1
Sural	1
Lumbosacral plexus	1
Laryngeal	1

and chronic inflammatory demyelinating polyneuropathy. While, there is whorl-shaped proliferations of Schwann cells in other lesions, not perineurial cells. So immunohistochemistry is very specific for the diagnosis of intraneural perineurioma, which is positive for Epithelial Membrane Antigen (EMA) and negative for S-100. While the perineural whorls surround a centrally placed axon with the myelin sheath and a small number of Schwann's cells that are reactive with S-100 protein. Collagen IV, CD34, CD56 and vimentin can be positive in some reports (**Table 1**) [2, 21, 35]. Also Claudin-1 and glut-1 can also be useful for the diagnosis of Sclerosing perineurioma [12], although sometimes these are not specific for intraneural perineurioma.

Ultrastructural examination found numerous pinocytotic vesicles and fragmented basal lamina around a central axon and accompanying Schwann cell. Some authors believe that the process of intraneural perineuriomas arises from repeated trauma. Although there is no deletion in the present case report, recent studies have identified a deletion in chromosome 22 (deletion of part or all chromosome 22) in the proliferative cells constituting intraneural perineuriomas [4, 17, 33]. This suggests that intraneural perineurioma represents a clonal expansion or neoplasm of perineurial cells [13]. Sometimes intraneural perineuriomas can be mistaken for HNPP which is characterized by focal myelin thickenings and abnormal folding. In order to exclude the diagnosis of HNPP, the method of STR-PCR or Real time-PCR is most commonly used. Schwannomatosis is also distinguished common misdiagnosis. The pathogenic gene, NF2, should be sequenced. If mutations in NF2 are identified, then the diagnosis of Schwannomatosis or Neurofibromatosis type 2 should be considered.

By demonstrating the incorporation or displacement of normal fascicles, MRI can also assist in the preoperative evaluation of whether the lesion is more likely to be an encapsulated neurilemmoma or an infiltrating neurofibroma [20]. In current case, MRI demonstrated enlargement of the nerves and abnormal hyperintense signal on the T2-weighted images including the cervical plexus, brachial plexus, intercostal

nerves, spinal roots, sciatic nerve, nerves of extremities, etc. The MRI findings were considered compatible with the results of biopsy examination including H&E, immunohistochemical and ultrastructural examination.

The treatment of intraneural perineurioma has been controversial. There is cause to excise the tumor and perform nerve reconstruction for the current case since the affected nerves are disseminated. About 5% of this disease can be local recurrence. The success in identifying this lesion depends on comprehensive analysis of the clinical symptoms, imaging and pathological study, as well as molecular cytogenetic studies.

Disclosure of conflict of interest

None.

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