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ORIGINAL PAPER

Parotid Gland Nodular Fasciitis: A Clinicopathologic Series of 12 Cases with a Review of 18 Cases from the Literature

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Abstract Nodular fasciitis (NF), very uncommon in the parotid gland, is a benign myofibroblastic proliferation that may be mistaken for other neoplastic proliferations. The mass-like clinical presentation and histologic features result in frequent misclassification, resulting in inappropriate clinical management. There are only a few reported cases in the English literature. Cases within the files of the authors' institutions (retrospective) confined to the parotid gland were compared to cases reported in the English literature (Medline 1966–2014). The patients included five females and seven males, aged 11–70 years (mean 45.2 years). All patients presented with a mass lesion, present on average 1.9 months, without a documented history of trauma. The lesions were 0.7–5.2 cm (mean 2.2 cm). Seven patients had fine needle aspiration. The majority of the lesions were circumscribed (n = 9), composed of spindle-shaped to stellate myofibroblasts (MF) arranged in a storiform growth pattern, juxtaposed to hypocellular myxoid tissue-culture-like areas with extravasation of erythrocytes. Dense, keloid-like collagen (n = 7) and occasional giant cells were seen (n = 6). Mitotic figures (without atypical forms) were readily identifiable (mean 4/10 HPFs). By immunohistochemical staining, the MF were reactive with vimentin, actins, and calponin, while the histiocytes were reactive with CD68. All patients had surgical excision. One patient developed local recurrence (12 months later). All were alive and disease free at last follow-up, with a mean 133 months of follow-up. The principle differential diagnoses include fibrosarcoma, fibromatosis, pleomorphic adenoma, myoepithelioma, neurofibroma, schwannoma, solitary fibrous tumor, leiomyoma, fibrous histiocytoma and myxoma. NF of the parotid gland occurs in middle-aged patients who present with a mass (mean 2.2 cm) in the parotid gland of short duration (1.9 months). FNA misinterpretation frequently leads to excision. Separation from myoepithelial and mesenchymal lesions affecting the parotid gland results in appropriate management.

Keywords Nodular fasciitis · Parotid · Salivary gland · Soft tissue · Differential diagnosis · Immunohistochemistry · Myofibroblasts

Introduction

Nodular fasciitis (NF) is generally regarded as a benign, tumor-like proliferation of myofibroblasts, that typically occurs in the extremities, originally described by Konwaller et al. [1, 2]. The head and neck region is a common location for NF, particularly in children, but NF of the parotid gland is rare. Therefore, we undertook this study in order to more completely define the clinical, cytologic, histologic, and immunophenotypic features of NF of the parotid gland with a comparison to cases reported in the English literature.

Materials and Methods

Twelve patients with parotid gland NF were identified in the files of the Departments of Pathology within Southern California Permanente Medical Group and Johns Hopkins

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Table 1 Clinical cases of parotid gland nodular fasciitis

No.	Sex	Age (years)	Symptoms	Symptom duration (months)	Parotid gland lobe	Side	Size (cm)	Treatment	Follow-up (months)
1	M	36	Mass	2	Superficial	R	1.8	Excision	A, NED, 60.6
2	M	58	Mass	5	Adjacent	L	5	Excision	A, NED, 310.9
3	M	54	Mass, pain	0.8	Superficial	R	0.7	Parotidectomy	A, NED, 289.3
4	M	40	Mass	1.3	Adjacent	L	2.5	Excision	A, NED, 299.5
5	M	34	Mass	1.2	Adjacent	R	3	Excision	A, NED, 145.4
6	F	45	Mass, swelling	1	Deep	R	0.9	Excision	A, NED, 42.7
7	F	56	Mass	1	Superficial	R	nr	Excision	A, NED, 130.2
8	M	70	Mass, pain, tenderness, swelling	1.5	Deep	R	5.2	Superficial parotidectomy	A, NED, 103.8
9	F	49	Mass	nr	Superficial	L	1.1	Excision	A, NED, NR
10	M	11	Mass	3	Superficial	L	1.2	Excision	A, NED, 10.2
11	F	45	Mass, swelling	4	Parotid	L	1.5	Parotidectomy	A, NED, 55.5
12	F	44	Mass, pain, tenderness, swelling	0.2	Adjacent	R	1.6	Excision	A, NED, 6.5

M male, *F* female, *R* right, *L* left, *nr* not reported, *A*, *NED* alive, no evidence of disease

Medical Institutions between 1998 and 2014 (Tables 1, 2). Materials were supplemented by a review of the patient demographics (gender, age), symptoms at presentation (including duration); and past medical history (specifically, a history of trauma). In addition, we reviewed cytology, surgical pathology and operative reports and obtained follow-up information from the treating physician or the patient. Follow-up data included the exact location, size, treatment modalities, and current patient and disease status. Fine needle aspiration materials were reviewed (when available). Hematoxylin and eosin stained slides from all cases were reviewed for morphologic assessment of the established diagnostic criteria for nodular fasciitis. This clinical investigation was conducted in accordance and compliance with all statutes, directives, and guidelines of an Internal Review Board authorization (#5968) performed under the direction of Southern California Permanente Medical Group and the Code of Federal Regulations, Title 45, Part 46.

A review of the English literature between 1966 and 2014 was performed. Clinical series of "head and neck soft tissue tumors" were selected if critical information about parotid gland NF were included (Table 3) [3–17]. Foreign language articles were only included if they were published alongside an English translation and articles with limited or lacking parotid gland information or duplicate publications were excluded [18–24].

Immunophenotypic analysis was performed by a standardized BenchMark-XT^{TM} method employing 4 µm-thick, formalin fixed, paraffin embedded sections. Table 4 documents the pertinent, commercially available immunohistochemical antibody panel used. When required, cellular conditioning for antigen retrieval was performed by various standardized retrieval techniques, as standardized and validated in our laboratory. The antibody reactions were described as either positive or negative; nuclear, cytoplasmic, membranous or combination; and a percentage reported for the Ki-67 antibody. Standard positive controls were used throughout, with serum used as the negative control.

Results

Patient Demographics and Clinical Presentation

The patients included five females and seven males, whose ages ranged from 11 to 70 years of age, with a mean of 45.2 years. The mean age at presentation for females, 47.8 years, was slightly older than for males, 43.3 years, but this difference was not statistically significant. All patients presented clinically with a mass lesion, often documented by imaging studies (Fig. 1). Additionally, three patients also had associated pain, two had tenderness, and four had a swelling of the area. No patients had ulceration. No patients reported a history of antecedent trauma. The duration of symptoms ranged from 1 week to 5 months, with an mean of 1.9 months.

Treatment and Follow-up

All patients were treated surgically, with complete removal of the lesion. Only one patient had a recurrence in the same site 12 months later, although without disease after being followed for a total of 311 months. Overall, all patients

Table 2 Summary of this case series of parotid gland nodular fasciitis

Characteristic	Number (n = 12)
Gender	
Females	5
Males	7
Age (in years)	
Range	11–70
Mean	45.2
Female (mean)	47.8
Male (mean)	43.3
Symptom duration (in months)[a]	
Range	0.2–5
Mean	1.9
Female patients, mean	1.5
Male patients, mean	2.1
Anatomic side	
Left	5
Right	7
Anatomic site	
Superficial parotid gland	5
Adjacent-periphery of parotid gland	4
Deep lobe	2
Parotid gland	1
Size (cm)[a]	
Range	0.7–5.2
Mean	2.2
Female (mean) ($p = 0.094$)	1.3
Male (mean)	2.8
Patient follow-up (mean, months)[a]	
Alive, no evidence of disease (n = 11)	115
Patient with recurrence (n = 1)	311

[a] Not reported for all cases

Table 3 Summary of English literature review of parotid gland nodular fasciitis [3–17]

Characteristic	Number (n = 18)
Gender	
Females	10
Males	8
Age (in years)	
Range	2.5–73
Mean	29.4
Female (mean)	36.6
Male (mean)	20.4
Symptom duration (in months)[a]	
Range	0.8–12
Mean	3.8
Female patients, mean	3.9
Male patients, mean	3.8
Left, mean	1.1
Right, mean	4.3
Anatomic side[a]	
Left	3
Right	14
Anatomic site[a]	
Superficial lobe of parotid	6
Adjacent to parotid gland	7
Deep lobe of parotid	1
Tail of parotid	2
Parotid (not otherwise specified)	2
Size (cm)[a]	
Range	1–6
Mean	2.2
Female (mean) ($p = 0.022$)	1.6
Male (mean)	3.0
Left (mean)	1.6
Right (mean)	2.4
Patient follow-up (mean, months)[a]	
Alive, no evidence of disease (n = 10)	16.6
Alive, patient who had recurrence (n = 1)	18.0

[a] Not reported for all cases

were alive and without evidence of disease with a mean 133 months of follow-up.

Pathology

Cytology Findings

Fine needle aspirations were cellular, with the lesional cells arranged in thick groups with feathered edges. There was a background myxoid matrix, but true mucinous material was not appreciated. Detached fragments of collagen were present. The cells were spindled, short and plumb, showing eccentric nuclear placement. A bipolar appearance was common, with cytoplasmic extensions noted in several directions (Fig. 2). Several of the cells were binucleated or multinucleated, while true "multinucleated giant cells" were also occasional present. The nuclear chromatin was delicate to coarse with prominent nucleoli. Mitoses were occasionally present. Reduplicated or rounded basement membrane material was not seen. Normal glandular or acinar elements were present, but were separate from the proliferation; i.e., there was no blending of the epithelium with the spindled cell population.

Macroscopic Features

The majority of lesions were received as multiple, irregular fragments, described as soft or firm, glistening, fleshy,

Table 4 Immunohistochemical panel

Antigen/antibody/clone	Company	Dilution	Results (# of positive/# tested)
Vimentin (V9)	Ventana Medical Systems, Tucson, AZ	Neat	8/8 (100 %)
Smooth muscle actin (66.4.C2)	Leica Microsystems, Buffalo Grove, IL	1:200	8/8 (100 %)
Muscle specific actin (HHF35)	Enzo Life Sciences, Farmingdale, NY	1:100	8/8 (100 %)
Smooth muscle myosin heavy chain (SMMS-1)	Dako, Carpinteria, CA	1:100	4/4 (100 %)
Desmin (D33)	Dako	1:400	0/10 (0 %)
CD68 (PG-M1)	Dako	Neat	6/6 (100 %)
S-100 protein	Dako	1:2,000	0/9 (0 %)
Cytokeratin	Dako		
(AE1/AE3:M3515	Boehringer Mannheim	1:40	0/11 (0 %)
and CAM5.2)	Biochemicals, Indianapolis, IN	1:8	
CD117 (C-Kit)	Dako	1:400	0/5 (0 %)
p63 (7jul)	Leica	1:40	0/5 (0 %)
GFAP (6F2)	Dako	1:200	0/5 (0 %)
CD34 (QBEnd/10)	Ventana Medical Systems	Neat	0/9 (0 %)

rubbery, homogenous and solid to variegated masses, that were white, tan-pale, gray or brown. The excised specimens measured 0.7–5.2 cm in greatest dimension (Table 2), with a mean of 2.2 cm. Four cases were noted at the periphery of the parotid gland (adjacent), involving the capsule, but not seen within the parenchyma.

Microscopic Features

The lesions were well circumscribed (n = 9; Fig. 3), while three were irregular and infiltrative into the surrounding soft tissues. Skeletal muscle was entrapped in four cases, with compression atrophy noted, while neural entrapment (n = 8) was a frequent finding (Fig. 4). Due to fragmentation a definitive margin status could not be determined with certainty.

The lesions were composed of a loosely cellular proliferation of tissue-culture-like myofibroblastic cells arranged in a loosely storiform growth pattern (Fig. 5), juxtaposed to hypocellular myxoid areas with extravasated erythrocytes. The lesional cells were spindled-shaped to stellate in appearance with oval nuclei, abundant basophilic cytoplasm and variably sized nuclei (Fig. 6). Occasional multinucleated giant cells were identified (Fig. 7) in six cases. Macrophages were focally abundant in two cases. Dense, keloid-like collagen was present in variable amount in seven cases (Fig. 8), although present to a lesser degree in the remaining cases. Mitotic figures were easily identified (range 1–14/10 HPFs; mean 4/10 HPFs), but without atypical forms (Fig. 7). The proliferation was adjacent to the parotid gland parenchyma in four cases, while identified within the parenchyma in the remaining cases.

Immunohistochemical Findings

While immunohistochemical studies were not necessary for the diagnosis of NF, the lesional MF cells were reactive for vimentin, smooth muscle actin (Fig. 9), muscle specific actin, calponin, and the histiocytes were positive for CD68 (Fig. 9). All cases tested were negative for desmin, S100 protein (Fig. 9), GFAP, CD117, p63, pan-cytokeratin, CD34 (Fig. 9), and Factor XIIIa. The immunostains, when positive, showed strong and diffuse cytoplasmic positivity.

Discussion

Definition and Nomenclature

NF was first described by Konwaler et al. [1], although referred to variably as pseudosarcomatous, proliferative and infiltrative fasciitis. This fibroblastic proliferation has recently been shown to have a well characterized molecular alteration, with fusion of *MYH9* with *USP6* [25]. While other abnormalities in the *USP6* gene (chromosome 17p13) are also noted in aneurysmal bone cyst, this sensitive and specific abnormality is a unique example of a self-limited human disease that shows a recurrent somatic gene fusion, and probably warrants re-classification as a clonal neoplastic disorder [25]. FISH evaluation of FNA material for *USP6* rearrangements may aid in accurate pre-operative diagnosis, as they are not detected in several histological mimics, such as desmoid-type fibromatosis, fibrosarcoma and myxofibrosarcoma [26].

It is postulated that NF arises from the superficial parotid gland sheath, with extension into the adjacent soft

Table 5 Combination of this case series and English literature review of parotid gland nodular fasciitis

Characteristic	Number (n = 30)
Gender	
Females	15
Males	15
Age (in years)	
Range	2.5–73
Mean	35.7
Female (mean) ($p = 0.169$)	40.3
Male (mean)	31.1
Symptom duration (in months)[a]	
Range	0.2–12
Mean	3.0
Female patients, mean	3.1
Male patients, mean	3.0
Left, mean	2.6
Right, mean	3.3
Anatomic side[a]	
Left	6
Right	19
Anatomic site[a]	
Superficial lobe of parotid	11
Adjacent to parotid gland	11
Deep lobe of parotid	3
Tail of parotid	2
Parotid (not otherwise specified)	3
Size (cm)[a]	
Range	0.7–6.0
Mean	2.2
Female (mean) ($p = 0.004$)	1.5
Male (mean)	2.9
Left (mean)	2.0
Right (mean)	2.3
Patient follow-up (mean, months)[a]	
Alive, no evidence of disease (n = 20)	65.8
Alive, patients who had recurrence (n = 2)	164.5

[a] Not reported for all cases

tissues and/or extension into the parenchyma of the parotid gland proper. Although trauma has been postulated as an etiology, none of the patients in this clinical series or the literature reported trauma, with one case postulated to arise from high cell phone use [14].

Epidemiology and Clinical Presentation

Although NF of the head and neck is relatively common, particularly in children, NF of the parotid gland is rare. We could only find single case reports and small or limited series of parotid gland NF in a review of the English literature (MEDLINE 1966–2014; Table 3). When our cases are combined with the literature, there is an equal gender distribution, with a mean age at presentation of 35.7 years (Table 5). Females are slightly older (mean 40.3 years) than males (mean 31.1 years), but this is not statistically significant ($p = 0.169$).

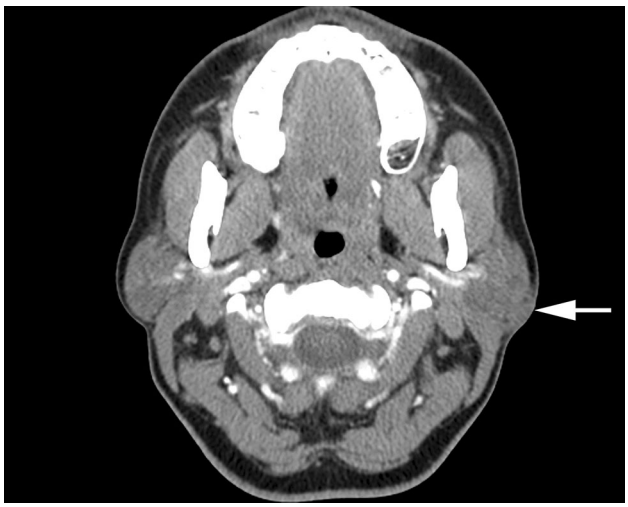

Fig. 1 A computed tomography scan of periparotid gland soft tissue enhancement, with extension into the periparotid and subcutaneous soft tissues (*arrow*)

The lesions are usually solitary masses that present with a rapid clinical onset. There is an overall average symptom duration of 3.0 months, but this is skewed by a couple of patients who had 12 months history. This duration of symptoms is longer than for extremity cases reported in the literature, where it is usually 2–3 weeks. Tenderness is infrequently reported (n = 2), while pain is seen in few patients (n = 4). No patients reported any trauma history.

Treatment and Prognosis

About 1–2 % of NF may recur, [2], while 6.7 % recurrence is noted in parotid gland lesions. This may be due to such a limited number of cases, although there are a greater number of fascial planes in and around the parotid gland which may result in a higher incidence of recurrence. Conservative excision can be performed if clinically indicated, although NF is generally considered self-limited with ultimate resolution without intervention. As the vast majority of cases (22 of 30; 73 %) were external and adjacent to the parotid gland, or only involving the superficial lobe, while an additional two cases involved the tail only, a limited excision can be achieved, without subjecting the patient to a total parotidectomy. No matter what therapy, none of the patients had any long term sequelae of parotid gland NF, followed up for >5 years (mean follow-up).

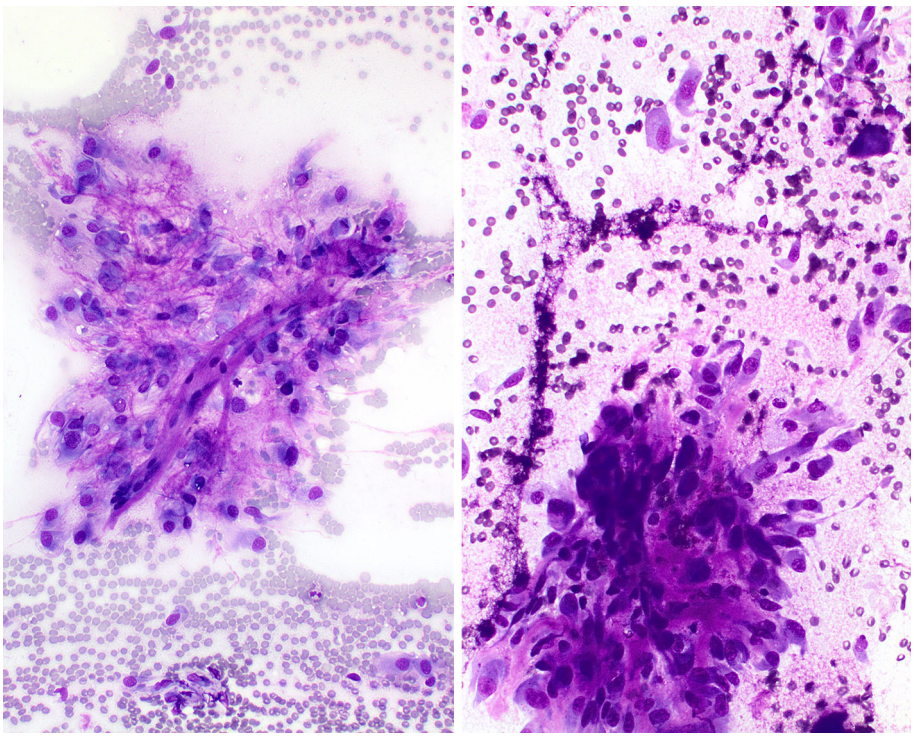

Fig. 2 Smears from parotid NF aspirations. *Left* note the feathering with a background of myxoid material. The cells are plasmacytoid. *Right* the bipolar cells have slightly more pleomorphism than the other case, but are still plasmacytoid with delicate nuclear chromatin distribution

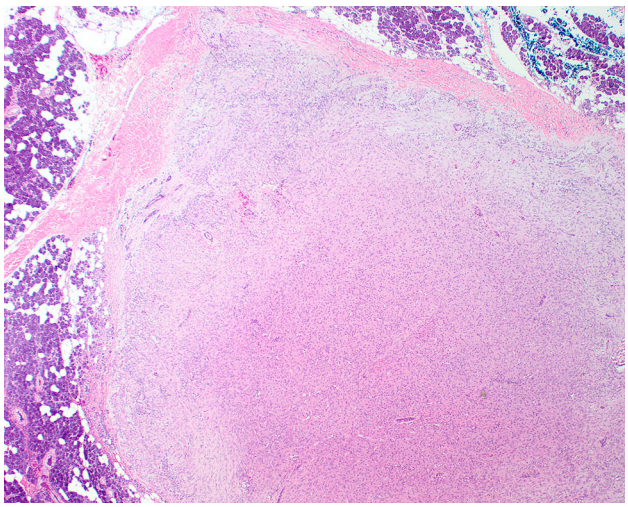

Fig. 3 There is a well defined and circumscribed appearance to the proliferation identified within the parotid gland parenchyma

Pathologic Features

Macroscopic

An attachment to fascia may be noted, and specifically with the parotid gland lesions, attachment to the superficial lobe of the parotid gland fascia was noted in 22 patients. The lesions were well defined, but may have an infiltrative border, especially as it may expand into the parotid gland parenchyma or into the adjacent soft tissues. The tumors were usually small (2.2 cm), similar to cases reported in the literature from other anatomic sites (2.3 cm) [2]. This may be due to a facial location, where a mass lesion is more likely to be discovered clinically while still a small size. Further, female patients had a statistically significantly smaller mass (1.5 vs. 2.9 cm) than male patients ($p = 0.004$). This may be due to a difference in attention to cosmetic findings (such as a mass) affecting the face.

Microscopic

As previously reported [27], there seems to be an arc of development, from a myxoid lesion, through an intermediate stage and ending as a fibromatous lesion. While there is considerable morphologic overlap, there tends to be a greater degree of myxoid change with a more haphazard architecture, extravasated erythrocytes and immature appearing fibroblasts at the beginning (patients who have a short symptom duration). The proliferation passes through a higher degree of cellularity with less background material, before finally appearing more compact with greater deposition of keloid-like collagen. Mitoses are usually easily identified, but do not include atypical forms. The absence of atypical mitoses may help with separation from malignant lesions in the differential diagnosis. Entrapment and involvement of the salivary gland parenchyma should not be viewed as "destructive" growth, but instead as an expansive and pushing growth into the parotid parenchyma. Similarly, entrapment of nerves or skeletal muscle (including atrophy of the muscle) does not equate to a malignant lesion.

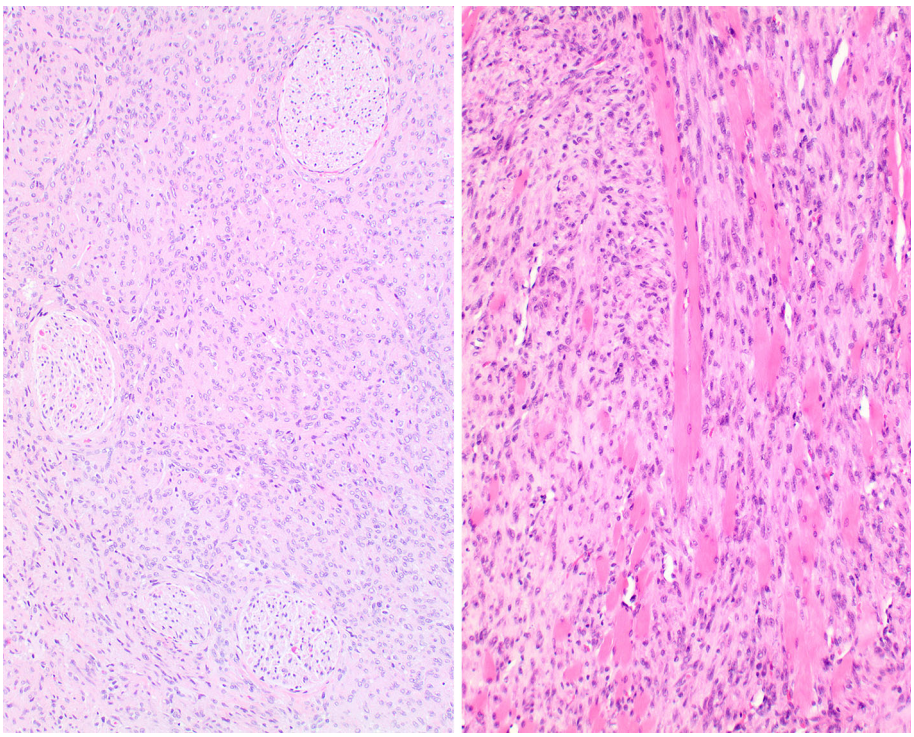

Fig. 4 *Left* several nerves are surrounded by the spindled to epithelioid proliferation of NF. *Right* the spindle cell proliferation is noted expanding between the skeletal muscle bundles

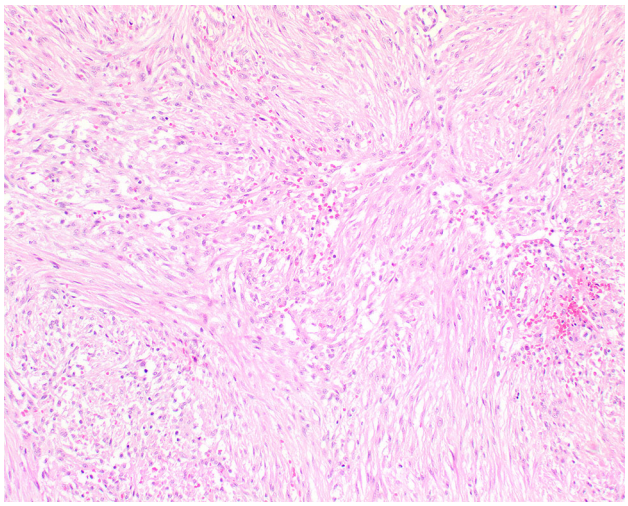

Fig. 5 The characteristic tissue-culture appearance with loosely arranged to storiform spindled cells showing extravasated erythrocytes

Immunohistochemistry

Immunohistochemistry is not required for the diagnosis of NF. However, in a core needle biopsy or in an exuberantly proliferative spindle cell lesion, a limited panel of pan-cytokeratin and S100 protein, along with muscle specific actin and CD68 would provide two negative and two positive results, respectively. These results would help to narrow the differential diagnosis considerably. If further difficulties remain, adding selected antibodies would allow for a definitive diagnosis. In very rare cases, FISH for *USP6* rearrangements may be valuable.

Cytology

Fine needle aspirations are performed on the vast majority of head and neck masses, with parotid gland lesions one of the most frequently sampled. Therefore, it is quite likely that a fine needle aspiration would be performed prior to any surgery. In about 3 % of cases, there is likely to be a significant spindle cell or mesenchymal component to the aspiration [28]. Familiarity with the features seen on fine needle aspiration material would assist in management planning [29]. Unfortunately, the aspirate material is frequently diagnosed descriptively, with a differential diagnosis list of several entities, the majority of which require surgery. It is important in a purely spindled cell aspirate, to include NF in the differential diagnosis, and to make this interpretation/diagnosis when the features are present. The natural arc of NF is to undergo involution with time, thereby avoiding unnecessary surgery.

Differential Diagnosis

NF in more usual locations is frequently incorrectly considered a malignant lesion due to its rapid growth, presence of a high cellularity, abundant mitotic figures, and capacity to be poorly circumscribed, and NF of the parotid gland is

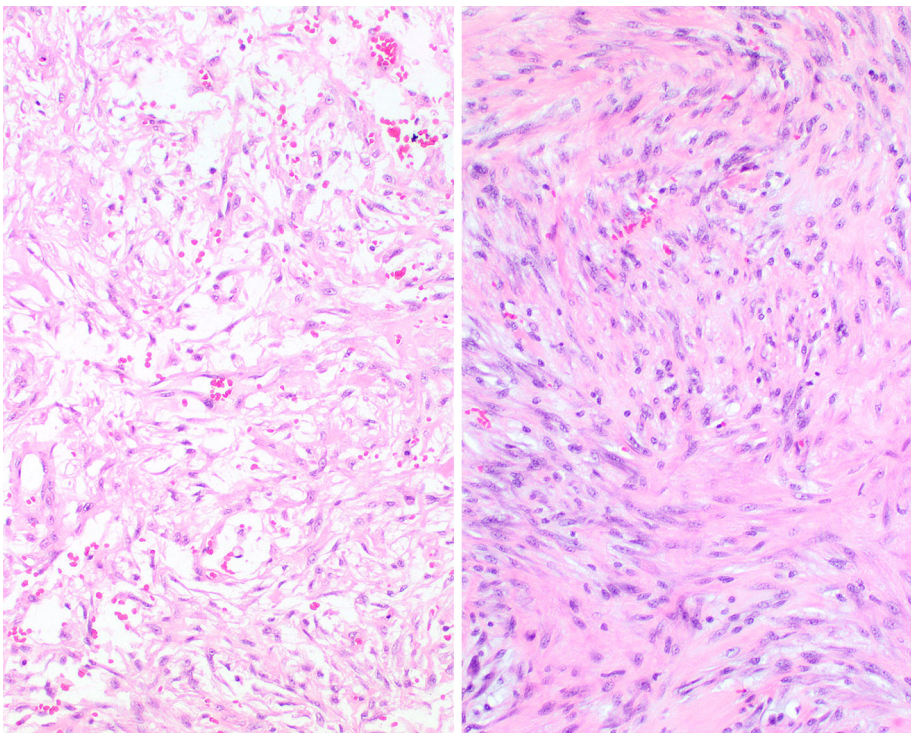

Fig. 6 The variable cellularity of nodular fasciitis can be seen in these two cases. *Left* hypocellular with myxoid change. *Right* hypercellular with elongated nuclei and small nuclei

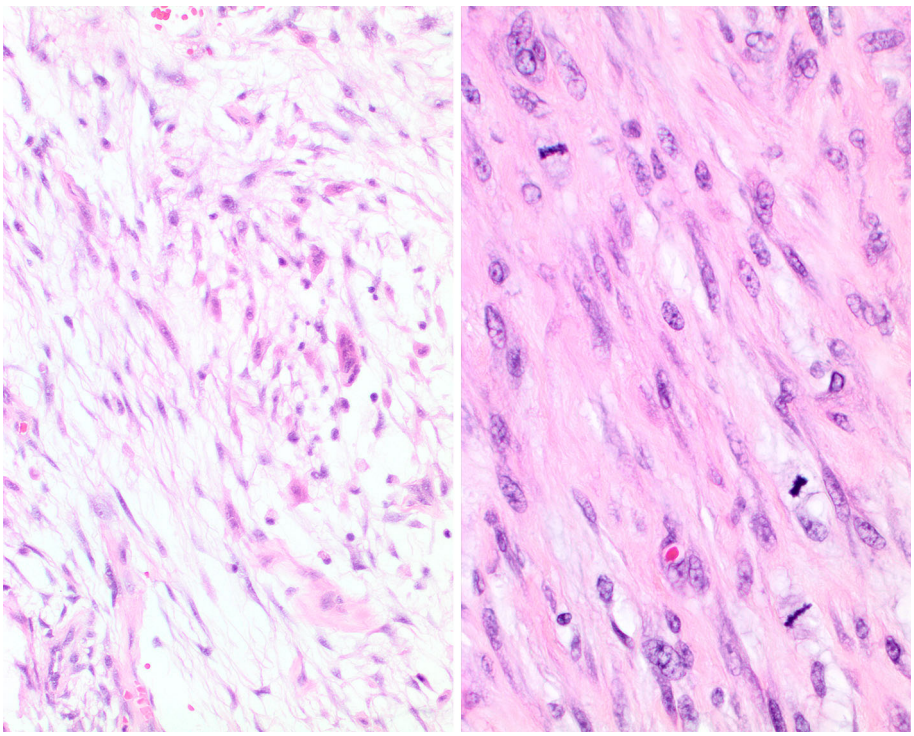

Fig. 7 *Left* isolated histiocytic giant cells could be seen. *Right* increased mitoses (three in this high power field) were seen in most cases, but no atypical mitoses were seen

frequently misdiagnosed. The most frequent misdiagnoses for parotid gland NF cases were (in order of frequency) fibrosarcoma, fibromatosis, pleomorphic adenoma, myoepithelioma, neurofibroma, schwannoma, solitary fibrous tumor, leiomyoma, fibrous histiocytoma, and myxoma. Other malignancies (dermatofibrosarcoma protuberans, myxofibrosarcoma, rhabdomyosarcoma, angiosarcoma) are rarely included in the histologic or cytologic differential diagnosis [11, 16, 18, 22, 23, 27, 28, 30]. Needless to say, this is a broad reactive and neoplastic differential

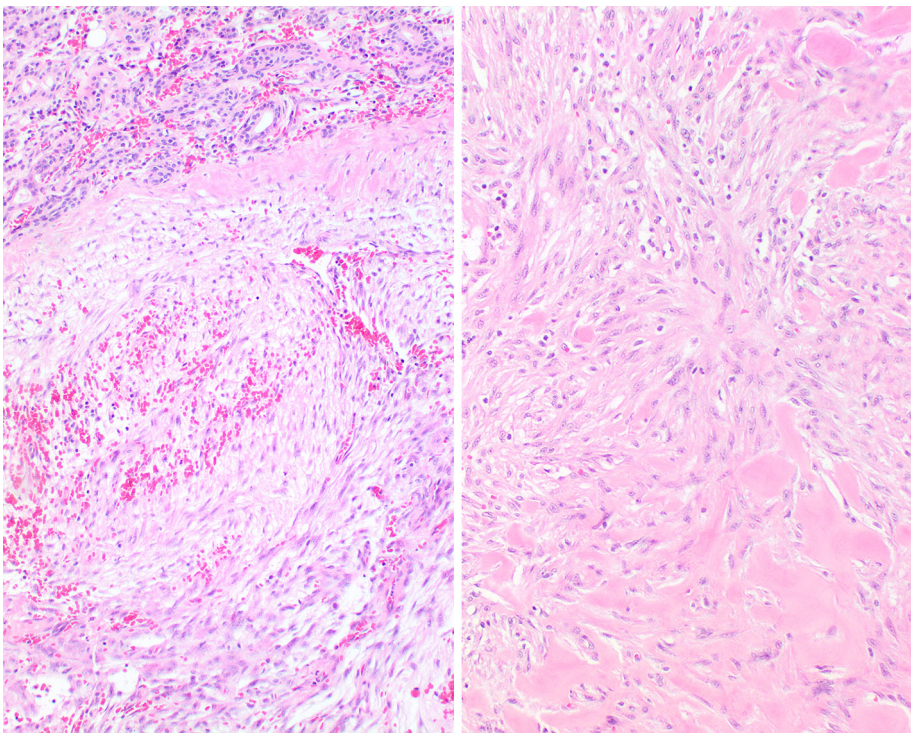

Fig. 8 *Left* a spindle cell population with extravasated erythrocytes (uninvolved parotid gland tissue at the *top*). *Right* dense, heavy, keloid-like collagen could be seen in most cases

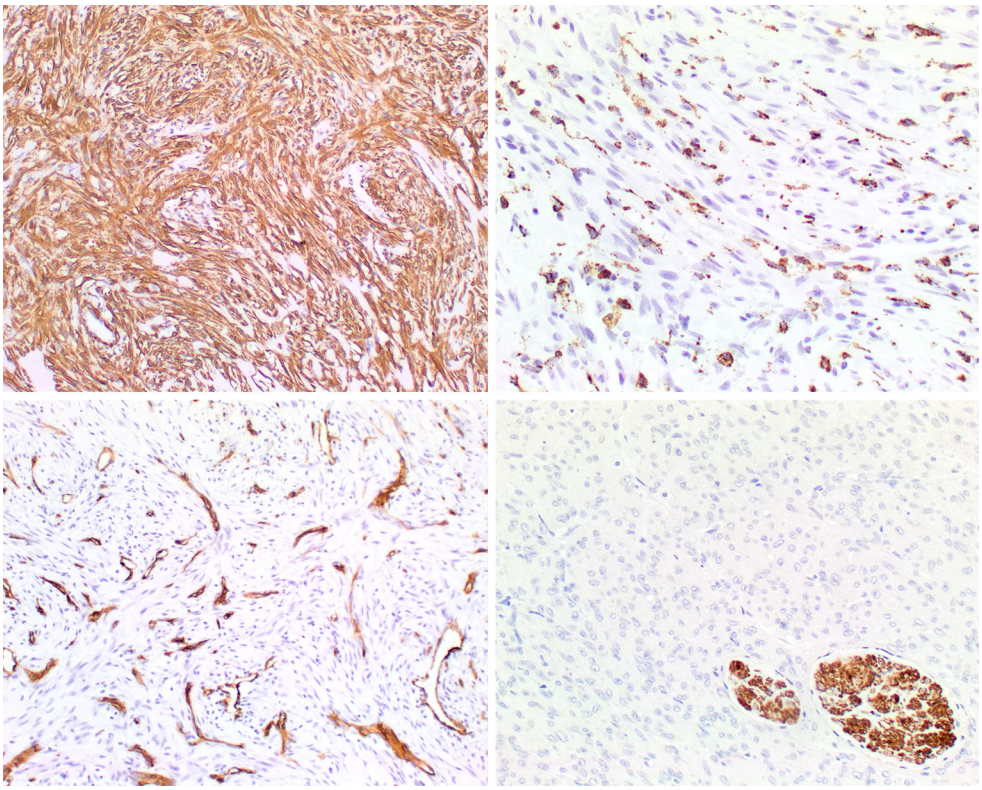

Fig. 9 The lesion cells showed the following immunohistochemistry results: *Left upper* smooth muscle actin positive. *Right upper* CD68 positive. *Left lower* CD34 negative in lesional cells (vessels are positive). *Right lower* S100 protein negative (nerve is an internal control)

diagnostic group. For the most part, the characteristic histologic features of a bland spindle cell population arranged in a patternless to fascicular architecture, with heavy, wiry, keloid-like collagen deposition in association with a rich vascular plexus can help to confirm the diagnosis with a limited, pertinent and focused immunohistochemistry panel.

Fibrosarcomas tend to be hypercellular with a more "herringbone" than storiform architecture. The nuclei are irregular and pleomorphic, with coarse, irregularly distributed nuclear chromatin. Tumor necrosis and atypical mitoses are features absent in NF [18]. Fibromatosis (desmoid or aggressive fibromatosis) are usually larger lesions, tend to involve the lateral neck and develop in young patients. Fibromatosis histologically usually shows a purposeful direction to the fibroblasts, prominent elongated vessels that are parallel to the direction of the fibers, limited collagen deposition, an "infiltrative" periphery, no pronounced myxoid substance and no "tissue-culture-like" appearance [31, 32]. The lesional cells show a strong nuclear β-catenin immunohistochemical expression, a finding lacking in NF [33]. This separation is important, as fibromatosis has a high local recurrence rate, often requiring much more aggressive surgery.

Pleomorphic adenoma (PA; benign mixed tumor) accounts for the vast majority of parotid gland tumors (up to 70 %). Therefore, a PA is a much more likely candidate to consider when there is a spindle cell proliferation. However, in general, a ductal or glandular component is seen, along with an intimate blending of the epithelial and mesenchymal elements (aspiration or excision samples). In a cellular PA or a myoepithelioma, only a spindled to oval cell population would be seen. PA does not show a tissue-culture like appearance and usually lacks extravasated erythrocytes and histiocytic giant cells. Furthermore, the cells in PA will show reactivity with epithelial markers (keratin, EMA, CK5/6, p63), S100 protein, GFAP, and muscle markers, different from the muscle markers and myofibroblastic reactivity seen in NF.

Schwannoma and neurofibroma are uncommon in the parotid gland or periparotid gland soft tissue. The tumors usually grow slowly and frequently are painful or tender; there is often acute, excruciating and radiating pain with fine needle aspiration. These tumors tend to be encapsulated and very well circumscribed, show nerve twigs at the periphery, tend to show palisading and Antoni A and B areas, while showing a blending of myofibroblasts and perineurites with nerve tissue. These tumors are SOX10, S100 protein and NFP positive, while lacking muscle markers and CD68 reactivity.

Solitary fibrous tumor tends to be quite cellular with short, wavy nuclei and heavy, wiry, refractile collagen deposition. There is a lack of destructive growth with limited mitoses. The strong reaction with CD34 and bcl-2 would help with separation from NF [30]. Desmin and other smooth muscle markers are generally positive in smooth muscle tumors (leiomyoma or leiomyosarcoma). Although myofibroblasts may occasionally be positive for desmin, we have not observed this finding in NF. A myxoma tends to be hypocellular, lacking any storiform architecture, giant cells or extravasated erythrocytes. There is a lack of muscle markers.

The general, imaging studies, clinical findings, and the histologic lack of nuclear atypia, a lack of necrosis, a lack of destructive infiltrative growth, and an absence of atypical mitotic figures should assist in separating NF from other malignant neoplasms, especially the sarcoma group. However, as parotid gland NF develops in a relatively young population, misdiagnosis as a sarcoma will often result in rapid and aggressive clinical management. Therefore, accurate diagnosis of NF is essential in order to avert inappropriately aggressive management.

Conclusion

In conclusion, NF of the parotid gland is rare, presents as a rapidly enlarging mass lesion of short duration in middle aged patients equally in both genders. Masses are smaller in females. NF should be considered in the differential diagnosis of a parotid gland spindle cell tumor, especially on FNA material, in order to potentially avert unnecessary surgery. If removed, there is a very low risk of recurrence (6.7 %).

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Conflict of interest None.

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