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Proliferative Myositis: A Comprehensive Review of 33 Case Reports

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Proliferative myositis, a rare reactive intramuscular myofibroblastic proliferation, is not well recognized in clinical practice. It overgrows within a few weeks and expands the space between the muscle causing infiltrative-like border mimicking sarcoma. Knowledge of the natural history and pathology of proliferative myositis is essential in order to prevent misdiagnosis and unnecessary surgical resection. Thirty-three reported cases of proliferative myositis in PubMed and Web of Science databases from 2000 to 2018 had been reviewed with the main emphasis in clinical presentation, radiological and pathological findings, treatment, and prognosis. Both males (19 cases) and females (14 cases), predominantly the middle-aged and senior adults, were affected. Upper extremity and shoulder girdle were commonly involved. The chief complaint varied from either painful or painless mass. The traumatic injury was reported as a significant predisposing factor. The lesion typically proliferated and separated muscle bundle. Ultrasonography of the lesion revealed a characteristic "checkerboard pattern" on transverse view. The definite diagnosis was based on the demonstration of spindle-shaped fibroblast/myofibroblast admixed with giant ganglion-like cells in the biopsy. Immunohistochemistry may be useful diagnostic tool when the histopathology was inconclusive. Misdiagnosis of sarcoma occurred due to its rapid growth and infiltrative-like border. Watchful management without surgery was sufficient because of the potential for spontaneous regression. Thoroughly clinical examination and appropriate investigations, including imaging and histopathology, are crucial.

Keywords: Proliferative myositis, Pseudosarcomatous tumor, Spindle-like fibroblast/myofibroblast, Giant ganglion-like cell, Immunohistochemistry

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Introduction

Proliferative myositis (PM) is a rare, benign, intramuscular proliferation. Kern[1] first described the nature of this disease in 1960. This mass consists of spindle-like fibroblast/myofibroblast and giant ganglion-like cell.[2] Because of its rapid growth and infiltrative border, PM can be mistaken as sarcoma both clinically and pathologically. Therefore, it is classified as a pseudosarcomatous tumor. Other entities in this group are nodular fasciitis, proliferative fasciitis, myositis ossificans, and related lesions. PM is not widely recognized due to its rarity.

This article aimed to review clinical presentation, radiological and pathological findings, treatment, and prognosis of PM among 33 reported cases of PM from 2000 to 2018.

Proliferative Myositis Cases

Case reports of PM were retrieved from PubMed and Web of Science databases using the search terms "proliferative myositis" and "pseudosarcomatous tumor" from 2000 to 2018. In addition, original articles and review articles associated with PM and pseudosarcomatous tumor were analyzed to clarify certain topic.

Epidemiology

PM usually occurs in the middle-aged and senior adults, but very rare in childhood.[2,3] Only one affected infant was described during the review period.[3] The patient age ranged from 8 months to 78 years, with a median age of 54 years (Figure 1A and Table 1). Both males (19 cases) and females (14 cases) were affected. A slight male predilection (1.36:1) was observed. Most cases were sporadic, and no racial association was evident. This result was similar to the study from Enzinger et al.[2]

Signs and Symptoms

Clinical symptoms are almost nonspecific.[2,4] Most patients presented with rapidly growing painful or painless mass. Although PM is often painless,[2] 15 patients presented with painful or tender mass. It typically presents as a solitary mass ranging from 1 to 7 cm with an average diameter of 4 cm. Fourteen patients presented to clinicians within 2 weeks after the discovery of the mass due to its rapid enlargement.

Sites of Involvement

Any muscles can be affected,[4] and a wide range of anatomical involvement with upper extremity predilection was described (Figure 1B). Affected muscles included biceps brachii, brachioradialis and palm muscle in the upper extremity, sartorius, vastus intermedius and gastrocnemius in the lower extremity. Head and neck regions are uncommon locations. PM arising in the tongue (2 cases), sternohyoid (1 case), infrahyoid (1 case), sternocleidomastoid (3 cases), psoas major (1 case) and perirectal area (1 case) had been rarely documented. Detailed information of all cases is summarized in Table 1.

Figure 1. Age With Sex Distribution (A) and Anatomical Location (B) of 33 Cases of Proliferative Myositis

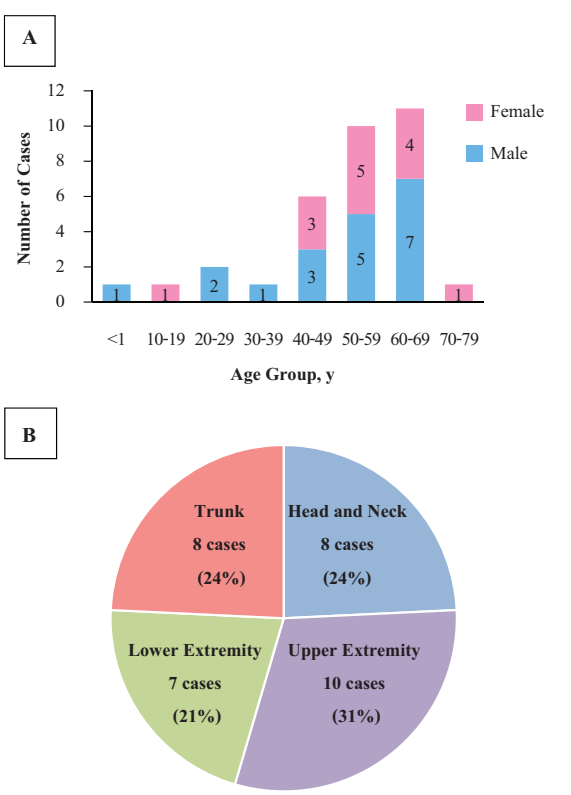

Table 1. Demographic Information of 33 Cases of Proliferative Myositis

Study	Age, y	Sex	Location	Pain	Largest Diameter, cm	Clinical Onset	Traumatic History	Treatment	Follow-up Duration	Outcome
Singh A, 2000[5]	46	F	Tongue, both sides	Yes	NA	NA	Yes	Wait and see	3 mo	Spontaneous resolve
Wong NL, 2002[6]	45	M	Palm, right	NA	1.5	1 wk	NA	Wait and see	33 mo	Spontaneous resolve
Wong NL, 2002[6]	78	F	Arm, right	NA	4	1 wk	NA	Wait and see	2 mo	Spontaneous resolve
Kent MS, 2002[7]	48	M	Pectoralis major, left	NA	7	4 wk	NA	Excision	1 y	No recurrence
Haloi AK, 2004[8]	28	M	Infrahyoid, left	No	6	2 y	No	NA	NA	NA
Wlachovska B, 2004[9]	56	M	Biceps brachii, right	Yes	7	4 d	No	Wait and see	2 mo	Spontaneous resolve
Pagonidis K, 2005[10]	52	F	Brachioradialis, left	Yes	NA	3 mo	No	Excision	2 y	No recurrence
Brooks JK, 2007[11]	65	M	Tongue, right	Yes	1	2 wk	NA	Excision	5 mo	No recurrence
Demir MK, 2007[12]	65	F	Biceps brachii, right	Yes	7	3 wk	Yes	NA	NA	NA
Fauser C, 2008[13]	64	F	Sternocleidomastoid, right	Yes	3	4 d	No	Excision	15 mo	No recurrence
Recarey FJR, 2008[14]	11	F	Gastrocnemius, left	No	6	2 mo	No	Excision	6 y	No recurrence
Wong NL, 2009[15]	53	F	Leg	NA	3	1 wk	NA	Wait and see	NA	Spontaneous resolve
Wong NL, 2009[15]	54	M	Thigh	NA	3	2 wk	NA	Wait and see	NA	Spontaneous resolve
Wong NL, 2009[15]	56	M	Arm	NA	3	5 wk	NA	Wait and see	NA	Spontaneous resolve
Yigit H, 2009[15]	48	M	Sternocleidomastoid, left	No	5	3 d	No	Excision	NA	NA
Znati K, 2010[17]	62	M	Neck	NA	2.5	2 wk	NA	Excision	NA	NA
Ergin M, 2011[18]	60	M	Latissimus dorsi, right	Yes	3	4 wk	No	Excision	6 mo	No recurrence

Table 1. Demographic Information of 33 Cases of Proliferative Myositis (Continued)

Study	Age, y	Sex	Location	Pain	Largest Diameter, cm	Clinical Onset	Traumatic History	Treatment	Follow-up Duration	Outcome
Talbert RJ, 2011[3]	8 mo	M	Gastrocnemius, left	No	4.5	4 mo	No	Wait and see	2 y	No further growth
Klapsinou E, 2012[19]	51	F	Latissimus dorsi, right	NA	NA	NA	Yes	Excision	NA	NA
Klapsinou E, 2012[19]	47	F	Inguinal region, right	NA	NA	NA	NA	Excision	NA	NA
Klapsinou E, 2012[19]	32	M	Axilla, left	NA	NA	NA	Yes	Wait and see	NA	Spontaneous resolve
Satish S, 2012[20]	65	M	Supraclavicular region	NA	3	NA	NA	Excision	NA	NA
Chawla N, 2013[21]	42	F	Forearm, left	No	2.5	1 mo	No	Excision	NA	NA
Franz D, 2014[22]	66	F	Psoas major, right	Yes	5.2	2 wk	No	Wait and see	2 mo	No further growth
Jarraya M, 2014[23]	52	F	Biceps brachii, right	Yes	6	3 wk	No	Wait and see	7 wk	Spontaneous resolve
Malhotra KP, 2014[24]	63	M	Sternocleidomastoid, left	No	2.2	10 d	No	Wait and see	2 mo	Spontaneous resolve
Zhang J, 2014[25]	56	M	Sartorius, left	Yes	5.2	2 wk	No	Excision	9 mo	No recurrence
Boroujeni AM, 2015[26]	59	M	Perirectal	Yes	NA	NA	NA	NA	NA	NA
Colombo JR, 2015[27]	51	F	Sternohyoid, left	Yes	2.2	3 d	No	Excision	3 mo	No recurrence
Binesh F, 2016[4]	66	F	Leg, right	Yes	NA	6 mo	No	Excision	NA	NA
McHugh N, 2017[28]	20	M	Latissimus dorsi, right	Yes	2.9	5 mo	Yes	NA	NA	NA
Wei N, 2017[29]	64	M	Brachioradialis, right	No	3	2 wk	NA	Excision	13 mo	No recurrence
Shi J, 2018[30]	69	M	Vastus intermedius, left	Yes	6	2 wk	NA	Wait and see	6 mo	Spontaneous resolve

Abbreviations; F, female; M, male; NA, not available.

Etiology

Many previous reports suggested that traumatic injury to the muscle is a predisposing factor for PM. McHugh et al[28] reported a case of PM occurring in a competitive rower who experienced repeated strains on his latissimus dorsi. Only 5 out of 26 patients reported prior trauma before mass development. Usually, there is no traumatic history to the site of the lesion.[31] Chromosomal abnormalities, including trisomy 2 and clonal translocation between chromosome 6 and 14, were also reported in literature.[32] Familial inheritance is not recognized. There was also a report of PM in a rheumatoid patient, suggesting that vasculitis may play a role in developing this disease.[33] Other proposed theories included local ischemia and paracrine myopathy.[13] However, the exact etiology is not yet established.[2, 10, 34]

Imaging

Radiographic findings in PM using various imaging methods are nonspecific. Histological confirmation is required for a definite diagnosis. A common imaging finding is an ill-defined intramuscular lesion associated with an inflammatory process. Characteristic ultrasonographic findings show scaffolding on longitudinal view, and "checkerboard pattern" on transverse view, defined by the non-uniform thickness of hypoechogenic geometric lines, that divide and encircle hyperechoic muscle bundles.[10, 16, 35] The continuity and fibrillary pattern of muscle fibers are mostly intact. It should be noted that these characteristic findings are not always present.[27]

PM, evaluated by computed tomography (CT), appears as a hypodense to isodense intramuscular mass with a poorly-demarcated margin. In one study, hypodense linear structures with a checkerboard-like pattern were evident.[16] There is no characteristic enhancement upon contrast injection. A mass with homogenous, heterogeneous, or no enhancement was described.[10, 16]

Other imaging modalities, preferably magnetic resonance imaging (MRI), may need to be performed. Although PM in this study appears as isointense comparing to the surrounding muscle on T1-weighted (T1w) image,[3, 9, 10, 14, 16, 23, 30] hypointense lesions have also been previously reported.[36, 37] PM also appears as a hyperintense mass[9, 10, 12, 14, 16, 28, 29] with variable hypointense linear structures[12, 16, 25] on T2-weighted (T2w) image. These linear structures were correlated with preserved continuity of muscle bundle, also known as a "stripe sign." This "stripe sign" is not specific for PM and can be found in other soft tissue tumors such as myositis ossificans and granular cell tumor.[38] Upon contrast injection, the lesion usually reveals homogenous enhancement[9, 12] but heterogenous enhancement can also be seen.[3, 4]

Positron emission tomography (PET) was also performed in some previous studies and revealed PM as a "hot spot". However, PET is not recommended because of its inability to differentiate malignancy from inflammation.[10]

Gross Pathology

On gross inspection, a mass appeared white to grey.[3, 20, 21, 27, 29] Poorly-demarcated masses were more common than well-circumscribed ones. It typically replaced a variable proportion of a muscle resulting in induration of the involved muscle. PM often replaced almost of the belly of a small or flat muscle. However, in larger muscles, wedge-like involvement of PM insidiously underneath a deep fascia cause a progressive depressed central portion of the belly.[2]

Histopathology

Biopsy of the mass is mandatory. Definite diagnosis of PM is primarily based on the presence of 2 characteristic cells, including spindle-shaped fibroblast/myofibroblast and giant ganglion-like cells.[2, 6, 15, 19] These cells proliferate and spread skeletal muscle bundles apart, producing a checkerboard-like pattern.[2, 3, 7, 11] Although surrounding skeletal muscle bundles are mostly left intact, some secondary regions of atrophy or focal necrosis may be noted.[2, 11, 14, 23] The stroma varies from myxoid to collagenous. The border of masses is typically ill-defined or infiltrative. The traditional microscopic features, including cytologic atypia, cellularity, mitoses, necrosis, and infiltrative growth, do not always indicate a malignant potential in the soft tissue tumor's biology and can be misinterpreted.

Spindle-shaped fibroblast/myofibroblast cells are more substantial and generally larger than the usual fibroblasts.[6, 15] Although they vary in shapes and sizes, most cells have elongated shapes with round to oval nuclei and 1 or 2 prominent nucleoli. Their basophilic cytopltasms are well-delineated with occasional long cytoplasmic processes.[6, 15] Their nuclei reveal a fine chromatin pattern. Mitoses are present, and may be numerous, but are not atypical.

Giant ganglion-like cells are large, at least twice the size of spindle-shaped cells, with triangular or polyhedral shape and basophilic cytoplasm.[15] These cells morphologically resemble ganglion cells or neurons. They contain 1 or occasionally 2 eccentrically located vesicular nuclei with numerous mitotic figures and sometimes look bizarre. Because of these appearances, they can be mistaken as rhabdomyoblasts or ganglioneuroblasts.[39, 40] Although the characteristic finding, as mentioned earlier, may be of concern for malignancy, the presence of fine chromatin, thin-smooth nuclear membrane, and absence of atypical mitotic figure favors a benign process.[19]

PM and proliferative fasciitis have similar histologic features. The distinctive feature between them is that the former involves muscle while the latter involves fascia and subcutaneous tissue.

Differential Diagnosis

Spindle cell lesions with overlapping histologic features with PM include nodular fasciitis, desmoid fibromatosis, or adult-type fibrosarcoma. Giant/bizarre ganglion-like cells may be sometimes confused with rhabdomyoblasts in rhabdomyosarcoma, or ganglion cells and neuroblasts in ganglioneuroblastoma.

Nodular fasciitis typically tend to show a well-demarcated border, whereas PM commonly shows an infiltrative border. Nodular fasciitis predominates in young to middle-aged adults without gender predilection. Most cases are derived from subcutaneous origin, but a minority of cases are intramuscular. Spindle cells in nodular fasciitis resemble those in PM due to their shared myofibroblastic nature, but nodular fasciitis lacks giant ganglion-like cells. Extravasated erythrocytes and lymphocytes are frequent findings in nodular fasciitis.

Desmoid fibromatosis is an infiltrative fibroblastic/myofibroblastic neoplasm with a significant potential for local recurrence. It is divided into 3 forms: abdominal wall, intra-abdominal, and extra-abdominal forms. Extra-abdominal desmoid fibromatosis occurs in the same anatomical distribution of PM including upper extremity, chest wall, thigh and head, and neck. It is a poorly demarcated tumor composed of spindle-shaped myofibroblasts arranged in sweeping fascicles without giant ganglion-like cells.

Giant ganglion-like cells in proliferative fasciitis and PM may mimic and be mistaken for sarcomas, most commonly rhabdomyosarcoma or ganglioneuroblastoma. The diagnostic pitfall that is most likely to encounter in childhood cases because rhabdomyosarcoma is a more common condition than that of proliferative fasciitis and PM. However, the ganglion-like cells lack cross-striations and show more cytoplasmic basophilia than that of rhabdomyoblasts. Ganglioneuroblastoma comprises of fascicles of Schwann cells with variable numbers of ganglion cells, resembling PM in a limited biopsy. Immunohistochemistry is very helpful to distinguish these entities.

Adult-type fibrosarcoma is a non-pleomorphic spindle cell sarcoma, commonly in the deep soft tissue of extremity or trunk. The lesion is typically composed of spindle cells arranged in parallel or herringbone pattern, without ganglion-like cells. Spindle cells in adult-type fibrosarcoma are more cellular than that of proliferative fasciitis and PM.

Although case reports of misdiagnosed PM rarely occur, 2 misdiagnosed cases as malignant neoplasm were documented during 2000 - 2018. Binesh et al[4] reported a 66-year-old woman developed a fast growing, painful and ill-defined mass at the right leg for 6 months with a clinical diagnosis of malignancy. Pleomorphic sarcoma was later diagnosed based on the excisional specimen. Fortunately, the patient refused chemotherapy and was referred to another clinician. After a pathological review, PM was finally diagnosed instead of pleomorphic sarcoma,

highlighting the importance of careful histological examination. Zhang et al[25] also described a 56-year-old man underwent radical surgery of a malignant mass at left sartorius based on the MRI finding. However, the final diagnosis of PM was inferred based on a pathological evaluation, signifying the limitation of radiological investigation in this setting. Because PM can easily mimic sarcoma/malignancy, consideration of clinical presentation, radiological characteristics and histopathology are key to assigning a correct and definite diagnosis.

Immunohistochemistry

Immunohistochemical stains may be helpful when the histological findings are undetermined. The immunohistochemical profiles of spindle cells in PM and proliferative fasciitis are similar to those of myofibroblasts in nodular fasciitis.[31] Strong and diffuse immunopositivity for vimentin, smooth muscle actin (SMA) and muscle-specific actin (MSA) are observed in the spindle cells.[9, 11, 21, 28, 41, 42] Desmin, h-caldesmon, S100 protein, myogenin, MyoD1, CD34, β-catenin, and keratin are not typically expressed. The ganglion-like cells in proliferative fasciitis and PM do not express neuroendocrine or muscle markers.

Nuclear staining for β-catenin, as a result of a dysregulation of the Wnt signaling pathway, is a unique phenotype of desmoid fibromatosis.[43] Spindle cells in desmoid fibromatosis also express SMA, MSA, and calponin.

Absent expression of myogenin and MyoD1 in ganglion-like cells further excludes the possibility of rhabdomyosarcoma.[43] Ganglion cells in ganglioneuroblastoma express neuroendocrine markers (synaptophysin, chromogranin A, neuron-specific enolase and CD56), whereas background Schwann cells are diffusely positive for S100 protein, unlike the spindle cells in PM.[44] Spindle cells in adult-type fibrosarcoma do not show specific immunophenotype. Focal positivity of SMA or CD34 is occasionally encountered.

Immunohistochemical findings in PM and differential diagnosis are summarized (Table 2).

Treatment

Regarding the available data, 13 patients underwent watchful monitoring with follow-up periods ranging from 2 to 33 months. The lesions in 11 patients regressed spontaneously. No further growth of the lesion was observed in 2 patients. Local excision was performed in 16 cases, and no recurrence was documented during follow-up periods ranging from 3 months to 2 years. Multiple reports also noted that PM has a benign origin, and it has a tendency to regress spontaneously.[2, 5, 6, 9, 15, 23] Therefore, it is reasonable to advocate for passive observation if the mass does not affect function.

Table 2. Immunohistochemistry of Proliferative Myositis and the Differential Diagnoses

Immunohistochemistry Marker	Proliferative Myositis	Nodular Fasciitis	Desmoid Fibromatosis	Rhabdomyosarcoma	Ganglioneuroblastoma
SMA	+	+	+	Variable	-
MSA	+	+	+	Variable	-
Vimentin	+	+	+	+	-
Keratin	-	-	-	Rarely	-
S100	-	-	-	-	+
Desmin	-	-	Rarely	+	-
Myogenin	-	-	-	+	-
β-catenin	-	-	+ (Nucleus)	-	-

Abbreviations; SMA, smooth muscle actin; MSA, muscle specific actin; +, positive; -, negative.

However, a large mass may be painful or can compress nearby structure, causing cosmetic disfiguration. Conservative surgical excision is recommended in this regard. To the best of our knowledge, there is no report of malignant transformation. Hence, radical or extensive excision should be avoided. Long term follow-up is unnecessary.

Conclusions

PM is a self-limited, benign, reactive intramuscular process possibly preceded by trauma in a small number of patients. The disease affects both male and female adults typically over 40 years of age. Manifestation in childhood rarely occurs. Although upper extremity, as well as shoulder girdle, are common mass locations, a wide range of muscles can be involved. Different imaging modalities, along with a history of rapidly growing infiltrative mass, may facilitate provisional diagnosis of PM in some cases, but a tissue biopsy is mandatory for a definite diagnosis. Watchful management with follow-up is recommended because PM usually tends to regress spontaneously. Local excision may be essential in the case of a large mass causing malfunction or cosmetic disfiguration. Recognition of the nature of PM helps to distinguish it from sarcoma and prevent unnecessary extensive surgical operations.

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โพรลิเฟอเรทีพมัยโอไซติส: การทบทวนวารสารการรายงานคนไข้ 33 ราย

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โพรลิเฟอเรทีพมัยโอไซติสเป็นโรคที่เกิดจากไฟโบรบลาสท์/ไมโอไฟโบรบลาสท์แบ่งตัวเกิดเป็นก้อนในกล้ามเนื้อ พบได้น้อย หายได้เอง โตเร็วภายในสัปดาห์ คล้ายมะเร็งซาร์โคมา จึงจัดเป็นเนื้องอกซาร์โคมาเทียม วัตถุประสงค์ของการทบทวนเพื่อป้องกันการวินิจฉัยโรคผิดเป็นมะเร็งและรักษาโดยผ่าตัดแบบกว้าง ผู้นิพนธ์สืบค้นรายงานคนไข้จำนวน 33 คน จากฐานข้อมูล PubMed และ Web of Science ปี พ.ศ. 2543 ถึง พ.ศ. 2561 โดยทบทวนอาการ ผลตรวจทางรังสีวิทยา พยาธิวิทยา การรักษาและพยากรณ์โรค พบทั้งผู้ป่วยชาย (19 คน) และผู้ป่วยหญิง (14 คน) ส่วนใหญ่เป็นวัยกลางคนจนถึงผู้สูงอายุ พบโรคมากที่กล้ามเนื้อไหล่และรยางค์ท่อนบน มีอาการทั้งแบบปวดและไม่ปวด มีรายงานพบการบาดเจ็บสัมพันธ์กับการเกิดโรค โรคมักแทรกเข้าในกล้ามเนื้อเกิดลักษณะคล้ายตารางหมากรุกเมื่อตรวจอัลตราซาวด์ คำวินิจฉัยที่แน่นอนขึ้นกับการตรวจทางพยาธิวิทยา พบเซลล์ 2 ชนิดดังกล่าว การตรวจทางอิมมูโนฮิสโตเคมีช่วยในการวินิจฉัยเมื่อพยาธิสภาพไม่ชัดเจน การวินิจฉัยผิดเป็นมะเร็งซาร์โคมาพึงระวังอย่างยิ่ง เพราะโรคนี้โตเร็วและขอบไม่เรียบ การติดตามผู้ป่วยโดยไม่ต้องผ่าตัดเพียงพอต่อการรักษา การตรวจร่างกายร่วมกับการตรวจทางรังสีวิทยาและพยาธิวิทยาช่วยให้วินิจฉัยและรักษาโรคได้อย่างถูกต้อง

คำสำคัญ: โพรลิเฟอเรทีพมัยโอไซติส เนื้องอกชนิดซาร์โคมาเทียม เซลล์ไฟโบรบลาสท์/ไมโอไฟโบรบลาสท์ซึ่งมีรูปร่างคล้ายกระสวย เซลล์ขนาดใหญ่ที่มีลักษณะคล้ายเซลล์ประสาท การตรวจทางอิมมูโนฮิสโตเคมี

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