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Intranodal Palisaded Myofibroblastoma: Histopatologic and Clinical Review for Clinicians and Pathologists

İntranodal Palisad Myofibroblastoma: Klinisyenler ve Patologlar için Histopatolojik ve Klinik İnceleme

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ABSTRACT
Intranodal palisaded myofibroblastoma (IPM) is a kind of mesenchymal tumor with benign behaviour. The origin of this tumor is differentiated smooth muscle cells and myofibroblasts. This is a rare entity and differential diagnosis from malignant tumors is important for clinicians and pathologists. The etiology has not been explained adequately. Age range is between 2nd and 8th decades of the life and it is seen more frequently in males. Inguinal region is the most common localization but has been reported in other localizations including submandibular and retroperitoneal areas. Total excision of the tumor is the treatment in these cases. Recurrence is very rare and there is no report about metastasis. In conclusion IPM is a rare entity and it is commonly misdiagnosed as metastasis. Kaposi Sarcoma and schwannoma are the most common two spindle cell neoplasias. Be aware of this unique entity is important because wrong diagnosis causes dangerous management strategies.
Keywords: Intranodal palisaded myofibroblastoma, lymphadenopathy, benign entity, excision

ÖZET
Intranodal palisaded myofibroblastoma (İPM) benign gidişi olan bir mezankimal tümördür. Bu tümörün kökeni diferansiye düz kas hücreleri ve myofibroblastlardır. Nadir bir antitedir ve malign tümörlerden ayırımı klinisyen ve patologlar için önemlidir. Etiyolojisi tam olark açıklanamamıştır. Yaş aralığı 2. ve 8. dekad arasındadır ve erkeklerde daha sıktır. İnguinal bölge en sık lokalizasyondur ancak submandibular ve retroperitoneal bölgeler de dahil diğer bölgelerde de tanımlanmıştır. areas. Lezyonun total eksizyonu esas tedavidir. Rekürrens çok nadirdir ve metastaz rapor edilmemiştir. Özetle İPM nadir bir antitedir ve yanlış olarak metastaz şeklinde tanı konabilir. Kaposi sarkom ve şıvannom en sık rastlanan 2 iğsi hücreli neoplazilerdir. Bu antitenin farkında olmak yanlış tanı ve tedavi stratejilerine neden olmamak için önemlidir.
Anahtar kelimeler: İntranodal palisadlı miyofibroblastom, lenfadenopati, benign antite, eksizyon

Introduction

Intranodal palisaded myofibroblastoma (IPM) is an infrequent benign mesenchymal tumor and it is originated from differentiated smooth muscle cells and myofibroblasts[1]. Myofibroblasts are in fact mesenchymal cells and spectrum is between fibroblasts and smooth muscle cells. They are seen in normal and pathologic conditions[2]. The cell of origin of the tumor is still not decided. Either a modified smooth muscle cell from hilar vessels or a intranodal stromal cell with myoid differentiation (myofibroblast-like cell), but probably not both.

Initially, IPM has been named as malignant neurilemmoma by the 2 authors[3,4]. Palisaded myofibroblastoma term has been proposed by Weiss et al[5]. Suster proposed "intranodal hemorrhagic spindle-cell tumor with amianthoid fibers" and Lee proposed solitary spindle-cell tumor with myoid differentiation of the lymph node[6,7]. Currently, the term IPM is preferred nomenclature for this tumor based on its histologic features and strict intranodal location[2].

The purpose of this paper is to give message to clinicians and pathologist. For this aim etiology, pathogenesis, clinical presentation and management have been discussed.

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Etiology

The etiology has not been explained adequately. However inflammation may be important due to high serum inflammation markers at the diagnosis and decrease after excision of the lesion. As Bigotti stated in 1991, the increase in number of intranodal myofibroblast-like cells within inguinal lymph node may be attributed to the need for mechanical support by stromal myofibroblasts due to increased drainage at this site. Also, trauma has been suggested as an etiologic factor. Additionally, other mutagenic factors introduced to inguinal lymph nodes due to increased drainage may contribute to the tumor's etiopathogenesis[6-8].

Age-sex

Age for IPM shows wide range. Age range is from 2nd to 8th decade but generally this entity is seen between 4th and 6th decade of life. This entity has been reported in an infant. It has been reported male predominance[8-12].

Localization

Inguinal region is the most common localization of this unique tumor, but this tumor has been shown at submandibular region and retroperitoneum. Weiss et al. defined this status with the the presence of high numbers of stromal cells with myoid properties at inguinal lymph nodes. Drainage of lower extremity, genitalia, buttock, abdominal wall and also umbilicus is regulated by inguinal region lymph nodes and explains the localization of IPM in inguinal site. Other reported locations include pelvis, abdomen, and paratracheal region[2,13-18].

Cell of origine

Myofibroblasts and/or smooth muscle cell of lymph nodes are the origin of this unique entity[5,6,19,20]. Vimentin, actin and amianthoid fibers factor XIIIa are found to be positive while desmin is negative[6,8]. It has been shown that type I collagen is located at center of these fibers and type III collagen at periphery[21,22]. Actin positive/desmin negative myofibroblasts are detected in inguinal field as compared with other lymph node regions. The proliferation pnenotype of myofibroblasts has been suggested as a secondary to drainage function of inguinal lymph nodes[20].

Pathogenesis

The origin of IPM is myofibroblasts or smooth muscle cells of blood vessels in lymph node[23]. High number of myoid cells and myofibroblasts are found at inguinal lymph nodes and these cells form this lesion. Histopathologic/electron microscopic findings suggest that smooth muscle cells or myofibroblasts are essent

ial elements of IPM[22].

The association between IPM and Epstein-Barr virus (EBV) and/or human herpes virus 8 (HHV8) is not celar enough. However some reports suggest the association between EBV oncoprotein-EBNA3C and this phenomenon suggests the abnormalities seen in multiple cell cycle checkpoints. This finding suggests the association between environmental agents and genetic background. This may be related with transformation of preneoplastic hyperplastic entity to a neoplastic process[7,11,13,22,24,25]. Cyclin D1 overexpression may be growth factor for spindle cells[7,21,22,26]. Cyclin D1 shows high expression and is thougt as a player in promoting the growth of IPM but it has not been found genetic problems for cyclin D1. High expression of cyclin D1 is detcetd in more than 50% of the spindle cells and this expression suggests the role of cyclin D1 in tumor growth without genetic disturbance[22].

Beta-catenin (CTNNB1) is the key molecule for Wnt signaling pathway. It is well known that Wnt pathway is important in tissue homeostasis and also regulation of cell proliferation, and differentiation. Missense mutations or alternative molecular mechanisms of Wnt pathway cause abnormal stabilization and nuclear accumulation in a variety of epithelial cancers. Nuclear staining is important. Somatic b-catenin mutations have been reported frequently in IPM[27]. IPM is probably a monoclonal proliferation of myofibroblast-like cells whose pathogenesis is a mutation in beta-catenin. This would more conveniently explain cyclin D1 overexpression as this protein is downstream of nuclear beta-catenin. The etiology of the process is probably the introduction of mutagenic factors into a lymph node chain susceptible to increased drainage. In summary there is not strict association between EBV and the development of IPM. Cyclin D1 overexpression may independently influence the spindle cells as growth factor[21,22,24,28].

Clinical Features

IPM is generally seen at inguinal region but may be seen in other sites such as mediastinal and submandibular regions[23]. The tumor is presented as a solitary, firm, mobile, painless mass. Rarely, IPM has been reported at retroperitoneal and axillary regions and clinical presentation is depend on localization of tumor[1,2,16,25,29]. Tumor is typically unilateral, without pain, solitary, firm, and mobile. Patients' symptoms including pain, and compression to the local structures are more severe with increased size of the tumor. IPM is most commonly presented as local lymphadenopathy but may be multicentric and/or bilateral. At this situation IPM mimics malignant disorders[1,8,30-32,33].

Imaging

Typically IPM is seen as a solid mass but may show mixed echogenicity and lobulated margins ultrasonographically[31]. Elastography of the mass shows elastic properties. Sono-elastography has been proposed with promising and has been reported as useful for differentiating from malignant lesions in some tissues, such as breast, thyroid, salivary gland, and lymph node[34-37]. However elastographic findings have been reported in a case with IPM and it has been found that sonographic features, including spectral Doppler analysis and elastography, are useless for diagnosis of this rare tumor[19]. Computed tomography can be performed to exclude the other mass lesions. IPM is seen on CT as a well-demarcated tumor and is the most commonly used imaging modality[8,13,24,31].

Histopathologic features

Palisaded myofibroblastoma terminology has been proposed due to its myofibroblastic origine and prominent palisaded spindle cells in lymph nodes. Tumor is located in the groin below the inguinal ligament and Typically capsule is well preserved and a single lymph node is involved and typically skin is not infiltrated[38]. Amianthoid fibers in the lymph node and hemosiderin-laden histiocytes, spindle cells are characteristic for this entity and typically amianthoid fibers are acellular and also eosinophilic amorphic material is seen in stellate-shaped collagenous bundles[6,7,21,23,24]. Electron microscopic studies are important to differentiate myofibroblastic and smooth muscle differentiation[21]. There is a narrow rim of lymphoid tissue with marginal sinuses and lesion is separated by a psuedocapsule and lesion has fibroblast-like spindle cell with focal nuclear palisading areas which are named as Antoni-A pattern as detected in schawannomas[13]. The spindle cells have mitotic activity but show elongated nuclei with a coarse chromatin and spindle cells have perinuclear vacuolization with milder nuclear pleomorphism. Interstitial hemorrhage and hemosiderin deposits frequently seen and mast cells can be present[24]. In summary there is short fascicular, whorled, and occasional fibromatosis-like growth pattern of spindled cells with scant, eosinophilic, fibrillary cytoplasm with perinuclear vacuoles, and frequently perinuclear intracytoplasmic inclusions, elongated, cytologically bland nuclei with occasional grooves or inclusions. These exhibit a tendency to palisade within fascicles and whorls; scattered eosinophilic stellate-shaped and elongated structures are composed of central hyalinized and peripheral fibrillary collagen.

Histochemical and immunohistochemical Features

Gomori-trichrome stain is used to determine the collagenous nature of these structures[22]. Spindle cells are stained with smooth muscle actin (SMA), muscle-specific actin and vimentin. It is known that actin (+),

desmin (-) myofibroblasts are more frequent in inguinal lymph nodes as compared to other sites and this finding supports the predilection of this tumor for inguinal region[5,19,20]. Desmin, c-kit (CD117), carcino-embryonic antigen, keratins, CD34, calponin, S-100, HMB-45, and EBV latent membrane protein, herpes simplex virus (HSV) type II and human papilloma virus (HPV) are found to be negative in spindle cells [1,13, 23]. CyclinD1 expression with low Ki67 index is observed in the nuclei and approximately 10-50% of spindle cells. As mentioned before CyclinD1 expression suggests the possible role of the cell cycle regulatory genes in the pathogenesis of IPM. Neoplastic cells are positive for beta-catenin and the Ki-67 index is generally low; less than 1%[13,17,22]. There is relation between nuclear beta-catenin and cyclinD1 expression within cells[27].

Electron Microscopic features

Phenotypic features and immunohistochemical profile is important but electron microscopy is confirmative and may be useful for the diagnosis of IPM with 2 typical properties. 1-Well-described amianthoid fiber is a collagen fiber with 80 to 150 nm width[10,39], 2-The spindle cells show both myofibroblastic and also have smooth muscle cell differentiations characteristics[23,39]. The diameter of the collagen fibers in the collagen bodies is less than those of classic amianthoid fibers and are therefore are not strictly amianthoid fibers, but amianthoid-like collagen fibers. Electron microscopy also shows smooth muscle features including external lamina and subplasmalemmal vesicles. Eyden failed to identify the myofibroblast cell-specific intercellular junction composed of myofilaments and fibronectin fibrils in a case of IPM[39]. There is cytoplasmic intranuclear pseudo-inclusions associated with cytoplasmic invagination and contours of the nucleus are irregular. Cytoplasm contains moderate to well-defined rough endoplasmic reticulum and a few mitochon-dria. Focal densities are related with smooth muscle myofilaments and these are characteristics of the myofibroblastic differentiation[31].

Table 1 shows typical histopathological, electron microscopic and fine needle aspiration cytologic findings[15].

Fine needle aspiration cytology (FNAC)

IPM should be considered in differential diagnosis of aspiration samples of inguinal lymph nodes by the pathologists and/or cytologists. Spindle cells without nuclear atypia with moderate cellularity and also fibrillar background material, hemosiderin granules, hemosiderin-laden histiocytes are seen in aspiration materials[1,14]. However cytology may not be diagnostic and surgical excision must be recommended[14,21].

Differential diagnosis

IPM may be reported as metastatic cancer. It is well known that inguinal lymph nodes have rich vascular components and immunohistochemical and/or electron microscopic findings show similarities between these lesions will be informative in differential diagnosis. For this reason experience of the pathologist is important in differential diagnosis[1,9,31-33]. The most important disadvantage of this is the report of a problem is to report IPM as metastatic disease or lymphoma. The most common two spindle cell neoplasias are KS and schwannoma and should be considered in differential diagnosis[6,7,13,23]. There is a long list of differential diagnosis of IPM and they are malignant mesenchymal tumors including leiomyosarcoma, intranodal schwannoma, dendritic cell sarcoma, inflammatory myofibroblastic tumor, solitary fibromastocytic tumor, solitary fibrous tumor hemangioendothelioma, dendritic reticulum cell tumors, all types of metastatic spindle cell lesions of lymph nodes, carcinomas including malignant melanoma metastasis, carcinoma with pseudosarcomatous features, metastatic carcinoma unknown origine and also lymphoproliferaive diseases[1,2,5-6,24,40].

IPM may be reported as KS may be misdiagnosed as IPM due to extravasated erythrocytes and hemosiderin pigments among the spindle cells[3,41]. HHV-8 is generally found as positive and this condition reduces the utility of this marker in the differential diagnosis og IPM, but the absence of nuclear palisading and amianthoid fibers in KS is important for the differential diagnosis. The absence of endothelial markers supports the diagnosis of IPM[42-44]. IMT is characterized by proliferation of spindle cells in the lymph node, a vascular structure laid down by compressed endothelium, and dense inflammatory cell component; amianthoid fibers observed in IPM are not present is important for differential diagnosis[41]. Detailed

immunohistochemical profile should be obtained in the presence of primary tumor. When a pathologist suspects from the IPM he/she reports this lesion as a low-grade spindle cell tumor and must recommend surgical excision[13,22]. IPM is well differentiated tumor with low proliferative activity and has benign biological behavior. It is important to distinguish it from primary and/or metastatic malignant lesions of the lymph nodes. Spindle-cell melanoma is another malignancy considered in differential diagnosis. Hemosiderin is frequently detected in IPM and it can mimic melanin and may be difficult to exclude malignant melanoma. In addition S-100 and HMB-45 positivity may be detected in spindle cells. However carcinomas express epithelial markers including keratin and EMA/cell atypia/pleomorphism and high proliferative activity are essential properties for carcinomas[40,43,44].

IPM has histomorphologic characteristics looking like IMT which is associated with a virus-induced alteration of cell cycle regulation and correct approach is important[21]. Table 2 shows differential diagnosis of IPM. In some cases with underlying neoplastic disease this tumor may be confusing for initial staging and/or relapse disease[18,42,45]. Table 3 shows histopathologic features of entities requiring to differentiate from IPM.

Treatment and prognosis

En bloc excision of the tumor is the treatment of choice in cases with IPM. It is not reported major peri/post-operative complications. The most important point is the correct diagnosis and to exclude all entities mimicking IPM. Surgery is the treatment of choice and it is not necessary to use drug and/or radiation treatment. Prognosis is excellent with complete excision and surgery is necessary in case of relapse[2,13,16].

Recurrence

Clinical outcome of IPM is benign, but this entity not infrequently mimic malignant/metastatic lesions. For this reason appropriate pathologic examination is very important and management depends on true diagnosis[24]**.** Recurrence is not frequent but it has been reportad in a few cases and local recurrence rate has been reported in about 6% of the cases but malignant transformation has not been reported[2,6,7,11,13,30,40]. Metastases have not been reported until today.

Conclusion

IPM is a unique histopathologic entity. The first step is correct diagnosis and exclusion of other entities, especially sarcomas and also malignant epithelial tumors. Inguinal region is the most common localization but has been reported in other localizations including submandibular and retroperitoneal areas. Total excision of the tumor is the treatment of choice.

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