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Case Report

Rare Benign Vascular Tumors of Unusual Location: Two Cases Report

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ABSTRACT

Vascular lesions are classified according to their tumoral, malformative or reactive origin. Their clinical incidence and their frequency in pathological recruitment are also highly variable, with some lesions being rarer or even exceptional. We report the observation of 2 rare benign vascular tumors of unusual localization.

Epithelioid hemangioma is a benign rare vascular neoplasm. It is defined by the presence of well-formed vascular channels lined by epithelioid endothelial cells. Pathologically, it has a wide morphological spectrum making diagnosis difficult. In some cases, immunoreactivity anti-FOSB is important in differential diagnosis with other malignant forms, namely epithelioid hemangioendothelioma and angiosarcoma. The pathogenesis is controversial; however, several studies argue for a neoplastic origin in 48% of cases by showing a variety of gene fusions depending on the anatomical site of the tumor. We report the observation of a huge retroperitoneal epithelioid hemangioma in a 43-year-old woman, a presentation never reported in the literature.

The second observation was a 29-year-old female patient who consulted for a right chest wall mass that was painful on exertion. A large excision was performed and pathological examination of the surgical specimen confirmed the diagnosis of intramuscular hemangioma. It is a unique benign soft tissue vascular tumor characterized by an infiltrative architecture. It can occur in any skeletal muscle but lower limb muscles are the most common sites accounting for over 50%. An intra-thoracic location is reported for the first time *via* our observation.

Keywords: Benign vascular neoplasm; Epithelioid hemangioma; Retro-peritoneal space; Intramuscular hemangioma; Chest wall

INTRODUCTION

Vascular tumors are rare subset of soft tissue tumors. They are defined as tumors that show endothelial differentiation. They are a heterogeneous group of neoplasms both in terms of degree of vasoformative morphology, biological behavior and genetic abnormalities [1,2]. Vascular lesions remain difficult to diagnose due to their rare incidence and overlapping clinical, radiographic and histological features.

According to WHO 2020 soft tissue tumors, vascular tumors are classified according to their malignant potential into benign tumors represented by hemangioma, tumors of intermediate malignancy also called locally aggressive tumors represented by hemangioendotheliomas, and malignant tumors, which are angiosarcomas. The term benign vascular lesions include benign vascular tumors that are secondary to clonal cell proliferation and vascular malformations that are malignant developments of vessels during intra-uterine life due to mosaic mutations [3].

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In this article we report two observations of rare benign vascular tumors of unusual location with a review of the literature.

CASE PRESENTATION

Case report 1

Patient aged 43 years, healthy, with no particular history. She consulted for dorsal and lumbar spine pain with abdominal distension. An abdominal CT scan was performed and revealed a huge right retroperitoneal mass measuring 28 cm × 13 cm. It was heterogeneous with hyperdense and hypodense areas. The tumor displaces the right kidney anteriorly and medially, crushes the vena cava posteriorly without invading it, displaces the digestive structures anteriorly to the left and inferiorly, and comes into contact with the anterior abdominal wall (Figure 1). The tissue areas are enhanced after injection of the contrast medium [4]. Given this radiological presentation, sarcomatous origin was the most likely hypothesis and the patient was admitted to the operating theater within a week without diagnostic biopsy. During excision attempts, significant adhesions were encountered with the right kidney. As the mass was well encapsulated, the surgical team opted for a lumpectomy alone without nephrectomy pending confrontation with the histopathological data [5].

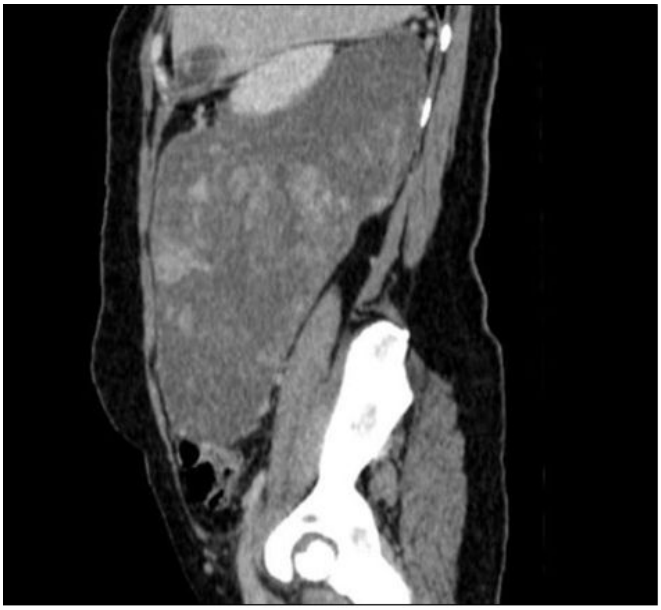

Figure 1: An abdominal CT scan: Right retroperitoneal heterogeneous mass, with hyperdense and hypodense areas. The tumor displaces the right kidney, displaces the digestive structures and comes into contact with the anterior abdominal wall.

Macroscopically, the tumor weighs 2000 g and measures 36 cm × 31 cm × 10 cm (Figure 2). It is a hard mass surrounded by a thick fibrous capsule. On opening, the tumor is made up of distinct and contiguous nodules of different sizes, with cystic remodeling measuring between 0.1 cm and 3 cm in length. No hemorrhagic or necrotic changes are seen (Figure 3). Histologically, it is a tumor proliferation arranged in ill-defined nodules, the site of a benign tumor proliferation of tightly packed epithelioid cells arranged in diffuse sheets. The tumor cells are medium-sized, monomorphic without cytonuclear atypia. The nucleus is round or oval with finely nucleated granular chromatin. The cytoplasm is abundant and eosinophilic with indistinct boundaries, showing intra-cytoplasmic vacuoles. The cells form lumens of variable size and shape without evidence of congestion. The stroma is edematous and fibrous (Figure 4). The modular architecture of the tumor evident macroscopically and microscopically, the proliferation of vasoformative monomorphic epithelioid cells with eosinophilic cytoplasm, the absence of stromal vascular remodeling and the absence of tumor necrosis support the diagnosis of epithelioid hemangioma. Further immunohistochemistry was performed to support the diagnosis, showing diffuse cytoplasmic staining with anti-EMA antibody, diffuse nuclear staining of the tumor cells with anti-CD34 antibody and focal nuclear staining with anti-ERG antibody. Morphological and immunohistochemical data confirmed the diagnosis of epithelioid hemangioma (Figure 5).

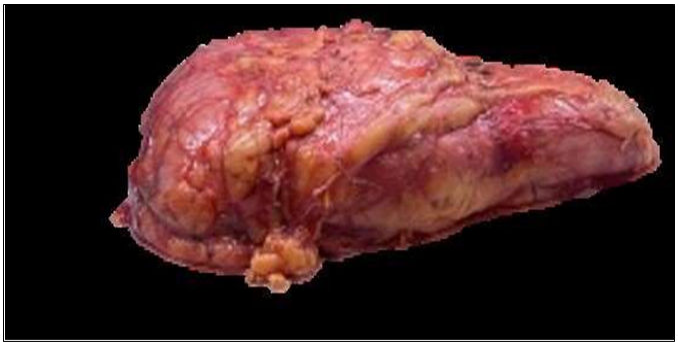

Figure 2: Retroperitoneal mass: Fresh macroscopic appearance: Well-encapsulated tumour measuring 36 cm long.

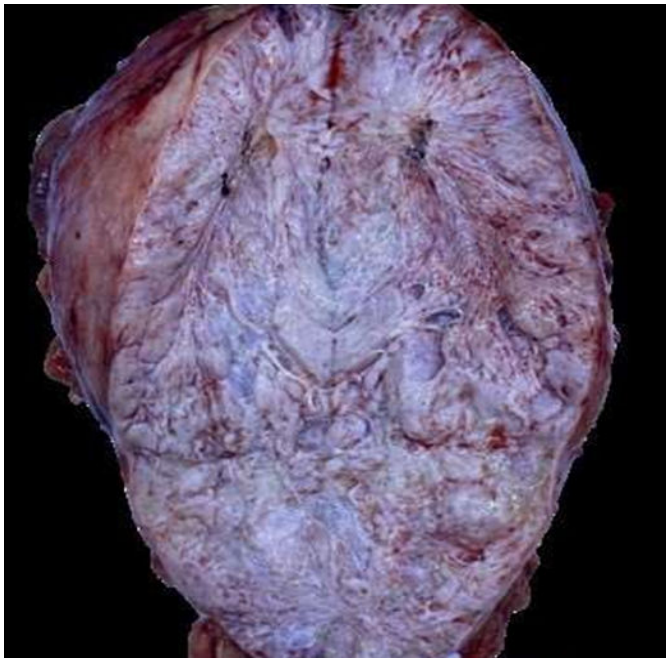

Figure 3: Freshly opened specimen: Distinct, contiguous nodules of different sizes, pearly white in color, with cystic remodeling measuring between 0.1 cm and 3 cm in long axis, with no signs of hemorrhage or necrosis.

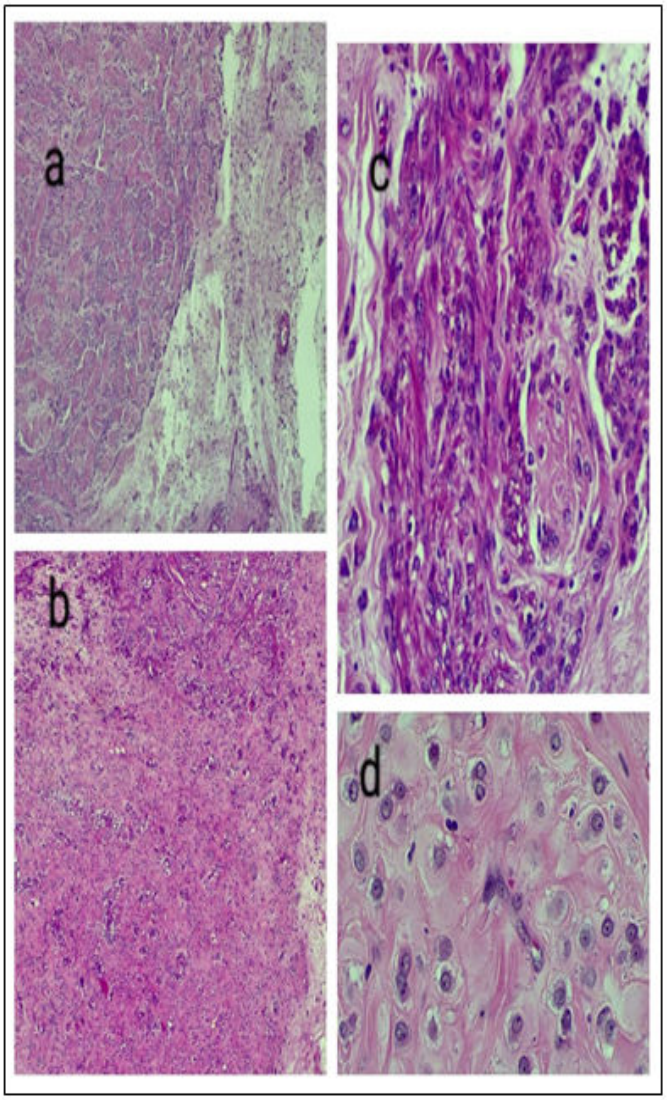

Figure 4: a) X4 tumor surrounded by a thick capsule; b) X10: Ill-defined proliferation of diffuse and nodular architecture; c) X20: Monomorphic epithelioid cells in small clusters with vascular differentiation forming lumens of variable size and shape; d) X40: Epithelioid. Cells without cytonuclear atypia with abundant eosinophilic cytoplasm.

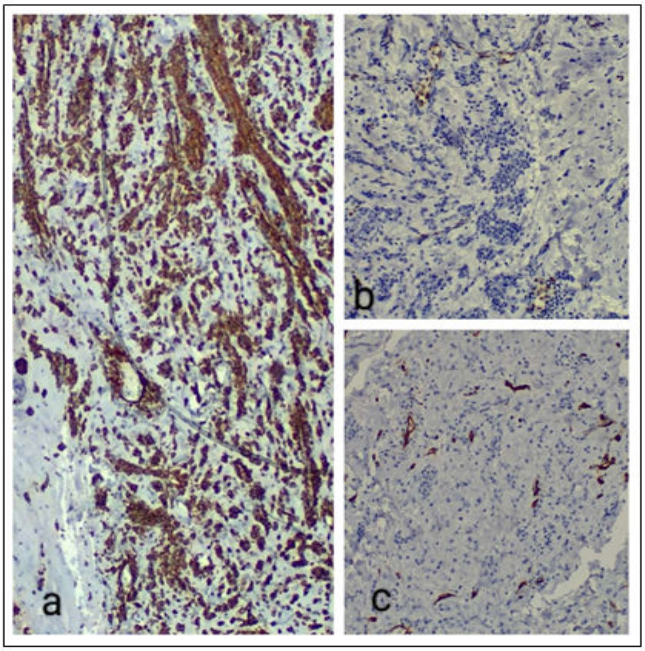

Figure 5: The epithelioid cells are a) AML+, b) CD34+; c) ERG+.

Discussion: Epithelioid hemangioma is a rare benign vascular tumor with an indolent course, first described in the 1960's [3]. This tumor typically occurs in the skin and subcutaneous tissue. It affects a wide age range from 10 to 75 years with predominance between the third and sixth decades and a slight female predilection [4,5]. Clinically it manifests as a single skin nodule in 80% of cases; multi-focality is encountered in pediatric patients, accounting for 50% of cases [6]. It is a reddish colored nodule, occurring in the head and neck region, particularly the peri-auricular skin, forehead and scalp. The size varies between 0.5 cm and 2 cm and rarely exceeds 5 cm [7]. Numerous locations other than the cutaneous form have been described, the most common being the bony location [8]. More rarely, the tumor develops inside the walls of arteries, in the heart, liver, trunk, and penis or inside mucous membranes such as the colon [8]. Although benign, epithelioid hemangioma of bone is the only clinical form that shows an infiltrative, locally destructive growth pattern, recurs in 11% and leads to metastasis in 2.7%. For this reason, WHO defines epithelioid hemangioma of bone as a locally aggressive tumor without being classified as benign, malignant or intermediate?

Our observation represents an unusual case never before described in the literature in terms of location, size and degree of compression of surrounding tissues and adhesion with adjacent organs. Retroperitoneal tumors are rare tumors. Their origin is dominated by primary sarcomas with an estimated frequency of 70%. The most frequent entities are liposarcomas, leiomyosarcomas and undifferentiated pleomorphic sarcomas. Benign retroperitoneal tumors are extremely rare. They are defined as any solid or cystic lesion arising in the retroperitoneal space with little or no potential for metastatic spread.

Information on benign retroperitoneal tumors is limited. However, the most commonly described tumors in the literature are benign lipomatous tumors, smooth muscle tumors, peripheral nerve sheath tumors and fibroblastic tumors [9]. Epithelioid hemangioma has never been reported in the retroperitoneal space.

The largest epithelioid hemangioma tumor described in the literature is 15 cm × 15 cm in size [3]. The tumor reported by the authors measures 36 cm × 31 cm × 10 cm, making it the largest epithelioid hemangioma. Histologically, it is characterized by a proliferation of epithelioid endothelial cells with discrete cytonuclear atypia. Depending on the architecture, a distinction is made between the lobular and the solid form of capillary hemangioma [10]. The lobular capillary hemangioma form is made up of mature open lumen vessels lined by epithelioid endothelial cells, which itself comprises the conventional subtype, the most frequent subtype in soft tissue, devoid of inflammatory infiltrate and a subtype called angiolymphoïd hyperplasia with eosinophilia, which accounts for 50% of all epithelioid hemangioma. It is distinguished by an inflammatory infiltrate rich in lymphoid follicles and eosinophilic cells. The solid form, or cell subtype, is characterized by diffuse proliferation of epithelioid endothelial cells, without open vessel formation. This is the most common subtype of epithelioid hemangioma of bone. In most cases, the lobular capillary hemangioma form and the solid form coexist within the same lesion in varying proportions. This is the same aspect reported in our observation [11].

Morphologically, the main diagnostic differential of epithelioid hemangioma, especially in its eosinophilic-rich inflammatory form, is with Kimura disease, a rare fibroinflammatory lymphoproliferative disorder. This disease is manifested by abundant vascular proliferation with histiocytoid endothelial cells with bulging eosinophilic cytoplasm and an eosinophilic infiltrate. However, Kimura disease occurs in the setting of familial allergy and atopy, manifests as painless subcutaneous nodules of the head and neck, regional adenopathy, and increased serum levels of Immunoglobulin E (IgE) and eosinophilic polymorphs [12].

However, when the diffuse architecture is more abundant, which is the case in our observation; differential diagnoses arise with malignant epithelioid vascular tumors, notably, anaplastic Kaposi's sarcoma, epithelioid hemangioendothelioma and epithelioid angiosarcoma. Indeed, the absence of cell pleomorphism, nuclear atypia, atypical mitosis patterns, tumor necrosis and low mitotic index, as described in our microscopic analysis study, allow the exclusion of malignancy [13]. However, in some situations, the distinction of epithelioid hemangioma, especially in its cellular variant, from other malignant epithelioid tumors makes use of an immunohistochemical panel that includes, besides the conventional vascular markers CD31, CD34, FLI-1 and ERG, the anti-FOS antibody and the anti-FOSB antibody. In the largest cohort published in 2022, involving 50 cases of epithelioid hemangioma, represented by its cutaneous form, 84% of cases showed a positive immunoreactivity for at least one antibody in 76%. Two-thirds of cases were FOSB positive, while 46% were immunoreactive for FOS. Co-expression of FOS and FOSB was found in 37%. [14]. A study published in 2017 of 24 cases of epithelioid hemangioma showed positive immunoreactivity for FOSB in 54%. This positivity was found in 75% (6 of 8 cases) of conventional epithelioid hemangioma, 10% (1 of 10) of solid epithelioid hemangioma and 100% (6 of 6) of epithelioid hemangioma of the angiolymphoïd hyperplasia type with eosinophilia [15]. A study published in 2018 showed that anti-FOSB immunostaining was diffuse with high intensity on 100% (15 cases) of epithelioid hemangioma in their angiolymphoïd hyperplasia with eosinophilia variants, whereas it was focal, low intensity on less than 5% of tumor cells in the malignant vascular tumors epithelioid hemangioendothelioma (4 cases) and epithelioid angiosarcoma (5 cases). The same study concluded that morphology is the main tool for differential diagnosis between epithelioid hemangioma and other malignant variants. Anti-FOSB immunostaining is sensitive for differentiation between epithelioid hemangioma, especially angiolymphoïd hyperplasia with eosinophilia, and other malignant vascular tumors [16].

The pathogenesis of epithelioid hemangioma isn't well established. The high frequency of previous history of trauma, infection and or arteriovenous shunt, argue for a reactive origin of epithelioid hemangioma [17]. However, over the last decade, numerous *in situ* hybridization studies have shown rearrangements involving *FOS* family genes in 30% of cases, particularly in clinical forms with multifocal and locally aggressive presentation. Earlier studies found GATA6-FOXO1 translocations in 18% of cases [18]. Genetic abnormalities remain undetermined in more than 50% of cases [19,20]. Indeed, no genetic abnormality is specific for epithelioid hemangioma; however, *FOS* gene abnormalities are more recurrent in this entity than in other vascular and perivascular tumors. The *FOS* gene family includes 4 members: the *FOS* gene on chromosome 14q24.3, the *FOSB* gene located on chromosome 19q13.32, and the *FOSL1* and *FOSL2* genes. The rearrangements involve only the *FOS* gene in two thirds of the cases and the *FOSB* gene in the remaining third. Multiple fusion partners of the *FOS* gene have been identified, namely the FOS-VIM translocation, the most frequent rearrangement, the FOS-LMNA, FOS-MBNL1, or FOS-lincRNA [21]. Recurrent rearrangements of the *FOSB* gene are the translocation t (19;19) (q13.2; q13.2) or interstitial deletion of chromosome 19 (q13.2-3), resulting in the fusion of ZFP36-FOSB, and less frequently the translocation (q25; q12), resulting in the fusion of the two genes WWTR1-FOSB [22].

The *FOS* genes encode leucine zipper proteins that can dimerized with JUN family proteins forming the AP-1 transcription factor complex that controls the transcription of genes involved in cell growth, differentiation and apoptosis. Wild-type FOS proteins have a highly conserved nucleotide sequence that makes the FOS protein the target of the proteasome for degradation. Mutant FOS proteins lack this sequence and thus escape degradation. Stabilization of the FOS protein results in stimulation of the Notch pathway, angiogenesis and endothelial growth *via* increased VEGF [23].

Morphologically, epithelioid hemangiomas with *FOS* and *FOSB* gene rearrangements are characterized by diffuse architecture, higher cellularity, moderate cytonuclear atypia, moderately high mitotic index and focal areas of necrosis. In bone epithelioid hemangioma, characterized by solid architecture, *FOS* gene rearrangements are identified in 59% of cases; this may explain the low anti-FOSB immunoreactivity (10%), as described above, reported in this solid subtype. The ZFP36-FOSB fusion is identified in penile locations alone representing two thirds of cases [24].

Although the eosinophil-rich inflammatory form shows 100% immunoreactivity for the FOSB antibody, none of the cases show a rearrangement for either the *FOS* or *FOSB* genes by FISH. This result implies that the overexpression of the FOSB protein is secondary to epigenetic modifications or point mutations not detected by FISH. Some epigenetic changes may be transient, which is an argument for a reactive origin [25].

Molecular biology may be indicated to rule out differential diagnoses such as the (p36; q25) WWTR1-CAMTA1 translocation found in over 90% of epithelioid hemangioendothelioma. No genetic abnormality is specific for angiosarcoma; some recurrent abnormalities may guide the diagnosis in difficult situations such as *MYC* gene amplification, and *KDR* gene mutation [26].

In general, treatment is based on complete surgical removal. In some situations, management is based on age, single or multifocal nature, anatomical site and clinical symptomatology. Young children may show spontaneous regression. In situations where, surgical removal is not possible or may result in severe morbidity, treatment is based on interventional radiology and/or medical treatment with sirolimus or interferon with therapeutic response. Radiotherapy and microwave ablation have been used, alone or in combination, with success [27].

Conclusion: An epithelioid hemangioma in the form of a huge retroperitoneal mass is a case reported for the first time by the authors. It is a benign vascular proliferation whose incidence is poorly defined due to the rarity of the tumor. This tumor is very heterogeneous both clinically and histopathologically. The identification of fusion genes allows a better understanding of the pathogenesis of this tumor and provides important diagnostic information. However, the diagnosis of epithelioid hemangioma is purely morphological, with molecular biology being used in cases of aberrant morphology or when biopsy material is limited.

Case report 2

The patient was 29 years old. She presented with a right chest wall mass that was fixed in relation to the deep plane and mobile in relation to the superficial plan and painful on exertion. The thoracic CT scan showed a tumor of vascular origin without pleural invasion. The patient underwent a complete resection with healthy surgical margins of more than 1 cm. The authors received for pathological examination a mass of striated muscle weighing 65 g and measuring 10 cm × 6 cm × 2 cm. On opening, the striated muscle mixed with fatty tissue was found to be the site of multiple microcystic remodeling of variable size and shape with no hemorrhagic or necrotic remodeling (Figure 6). Histologically, the muscle tissue was dissociated by a benign non-encapsulated tumor proliferation, made up of small capillary structures associated with large anastomosing vascular spaces giving a cavernous appearance. They consist of a thin wall lined by a single layer of flattened endothelial cells. There is a proliferation of spindle-shaped endothelial cells mixed with mature adipocyte cells. The tumor cells are labeled with CD 34 and ERG (Figure 7). The diagnosis of intramuscular angioma was made.

Figure 6: Macroscopic image of chest wall intramuscular angioma after fixation, showing striated muscle with microcystic remodelling, without haemorrhagic or necrotic signs.

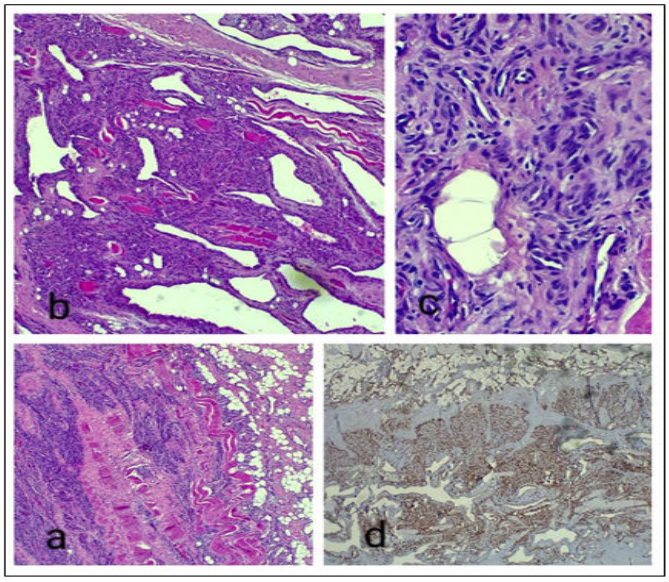

Figure 7: a) X4 striated muscle dissociated by unencapsulated benign tumor proliferation, b) X10 small capillary structures associated with cavernous spaces, bordered by a single bed of flattened endothelial cells, c) X20 proliferation of spindle-shaped endothelial cells mixed with mature adipocyte cells, d) X20, anti CD34 antibody: Intense and diffuse. Cytoplasmic.

DISCUSSION

Intramuscular angioma is a benign vascular tumor of the skeletal muscles. It is a very rare entity, accounting for less than 1% of all soft tissue hemangioma and usually occurs in young women under 30 years of age [19]. Given the rarity and lack of data on this entity it is considered by the international society for the study of vascular anomalies to be a provisionally unclassified vascular anomaly [20]. Intramuscular angioma can involve any skeletal muscle, however the lower limb is the most common site accounting for over 50% [21]. Several cases of intramuscular hemangioma have been described in the skeletal muscles of the head and neck, ENT muscles, abdominal muscles and paraspinal muscles. To our knowledge, this is the first observation that reports an intra-thoracic location.

The pathogenesis of intramuscular hemangioma is not clearly defined. Some authors argue for a post-traumatic origin responsible for an increase in angiogenic factors such as Vascular Endothelial Growth Factor (VEGF) and Matrix Metalloproteinases (MMP). Other authors suggest a congenital origin [22]. Recent molecular studies have revealed somatic mutations in the *MAP2K1* and *KRAS* genes similar to those detected in several types of arteriovenous malformations, suggesting an overlap between two conditions [20]. Tumor growth is slow and can be accelerated by trauma, growth spurt, and hormonal change including contraception [23]. When the tumor reaches a significant size, poor local circulation can be responsible for chronic anoxia of the myocytes with accumulation of metabolites which can be complicated by scleroderma of the whole muscle, fibrosis, calcification, ossification and bone lysis [22].

Clinically, intramuscular hemangioma initially presents as a painless subcutaneous mass that progressively increases in volume with no skin change in the area. In advanced stages the patient reports muscle swelling, spontaneous pain and throbbing on palpation accentuated by physical effort [22]. Radiological examinations to support the diagnosis are Doppler ultrasound, which shows a tumor of vascular origin with high blood flow velocity, and contrast-enhanced CT scan, which confirms the vascular origin and determines the size and shape of the tumor [24]. Magnetic resonance imaging, MRI, is the most sensitive examination with 90% of sensibility. It can also be used to distinguish intramuscular hemangioma from soft tissue sarcoma by identifying the lobulated and compartmentalized nature of the lesion, which is found in hemangioma but not in sarcoma [25]. In some situations, MRI displays signs mimicking malignancy, like non- homogeneous lesions, blurred borders, high density shading in soft tissue, bone destruction and infiltration of nerve and arterial structures [22]. The diagnosis of certainty is based on histopathological examination. Although a benign tumor, histologically intramuscular hemangioma is characterized by non-encapsulated capillary proliferation, showing an infiltrative growth pattern, intraluminal papillary projections and perineural infiltration. In addition to these features, it is a proliferation of spindle-shaped endothelial cells arranged in small clusters or diffuse nappes most often associated with mature adipocytes. Intramuscular hemangioma is classified by Allen and Enzinger in 1972 into three subtypes according to the size of the vascular structures that constitute them: capillary-type (small vessels), cavernous-type (large vessels) and mixed-type. Immunohistochemical examination is used only to confirm the vascular nature of the proliferation by anti-CD34 antibody, anti-CD30 antibody and anti-Erg antibody. The differential diagnosis of intramuscular hemangioma depends on the location of the lesion. In general, malignancy should always be ruled out, mainly angiosarcoma, and Kaposi's sarcoma [26]. Intramuscular angioma is distinguished from angiosarcoma by the absence of cytonuclear signs of malignancy, namely anisokaryosis, hyperchromasia, and nuclear irregularity, the presence of multinucleated endothelial cells and tumor necrosis. Kaposi's sarcoma is characterized by vascular proliferation without muscle infiltration and anti-HVV-8 immunoreactivity. The differential diagnosis is also made with other benign conditions, mainly angiolipoma and intramuscular venous malformations. Angiolipoma is a proliferation of mature adipocytes and well-formed thin-walled capillary structures showing a predominantly lobular arrangement at the periphery of the lesion without a spindle-shaped proliferation of endothelial cells. It is also characterized by intravascular fibrin bridges and extensive thrombosis [27]. Venous malformations can be located intramuscularly, mainly in the lower limbs. They are painful due to intravascular coagulopathy responsible for thrombosis and phlebitis. Intramuscular venous malformations are not associated with either a capillary component or a proliferation of spindle endothelial cells [20].

The treatment of choice for intramuscular hemangioma is adequate surgical excision with safe surgical margins, a condition difficult to achieve for deep infiltrating hemangiomas. In our case, the patient underwent a wide excision with safety margins of more than 1 cm on pathological examination. The frequent recurrence, reaching 28%, is related to microscopic infiltration of the surrounding muscle tissue and minor feeder vessels [23]. Spontaneous regression or malignant transformation is exceptional.

CONCLUSION

We describe for the first time a case of intramuscular hemangioma of the chest wall; a rare benign vascular proliferation that most often occurs in the lower limbs. The patient underwent surgical excision with borders of more than 1 cm. The evolution was marked by a clinical improvement few weeks after the operation. Anatomopathological examination of the excision specimen allows the diagnosis to be made and the surgical margins to be estimated as the main risk factor for recurrence.

CONFLICT OF INTEREST

None.

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