From the pseudolipoma to lipoma: staging of the typical radiological appearances. Pictorial essay.

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Abstract

Lipomas are benign mesenchymal tumors, which accounts for 50% of soft tissue tumors. Although the etiopathogenesis has not yet been fully understood, it is known that lipomas developed at a rate of 1% in the related localizations after traumas. Trauma initiates inflammatory reactions in fatty tissue and these can be the trigger mechanism for the development of lipomas. In this pictorial essay the objective was to present the imaging findings associated with the process of lipomas development. “The phases of lipomas formation” was schematized based on these data.

Keywords: lipoma, post-traumatic, ultrasonography, computed tomography, magnetic resonance imaging

Introduction

Lipomas are common benign mesenchymal tumors composed of mature adipocytes [1]. Soft-tissue lipoma accounts for almost 50% of all soft-tissue tumors. Lipomas predominantly occur in the subcutaneous tissue; intramuscular, intermuscular, subfascial, retroperitoneal, mediastinal, gastrointestinal and intraneural lesions have also been reported [2,3]. Although the etiology and pathogenesis are not clear, the incidence of post-traumatic lipoma is about 1% [1].

Benign lipomatous lesions involving soft tissues are classified into distinct categories: inflammation of fatty tissue, encapsulation of the affected area (pseudolipoma), lipomatosis, lipoma (simple lipoma, atypical lipoma) and the malignant ones are lipoblastoma and liposarcoma [1]. Although simple lipomas and well-differentiated liposarcomas are both grossly fatty masses, ultrasonography (US), computed tomography (CT) and magnetic resonance imaging (MRI) have been used to distinguish these two lesions [4-6].

The subcutaneous tissue localization of lipomas has attracted our attention and when the data of the current cases with lipomatous lesions were reviewed, we observed that these usually occurred after a trauma [7,8]. The reason for the uncontrolled formation and growth of lipomas is still unclear. In the last few years, various theories such as endocrine, dysmetabolic, and genetic causes have been discussed. It has been hypothesized that the triggering mechanism for the development of lipomas is activated by the release of cytokines and growth factors after trauma [9,10]. It has been suggested that the rearrangement of the lipomatous tissue after its transfascial herniation caused by a deep blunt injury supports the development of lipomas [11]. Also, the prolapse of normal adipose tissue outside its normal borders has been suggested to be the trigger mechanism for lipoma development [12].

The purpose of this illustrated article is to present the characteristic imaging findings of the lipomas formation stage by stage in cases with history of trauma or pathologies causing repetitive microtrauma. For this purpose we analyzed the US and/or MRI or CT aspect of 27 lipomas that appeared in relation with a trauma.
Lesions were grouped based on their imaging features:

**Group I, thickening of the subcutaneous fat tissue (lipodystrophy)**

Ultrasonographic examination performed within the first 6 weeks after the trauma. Sonography revealed only the thickening of the subcutaneous fat tissue as heterogeneous hyperechogenicity when compared to unaffected normal areas. There was no clear line of demarcation between the lesion and the normal tissue. The same thickening and minimal striations could be monitored on MRI T2-weighted (T2-W) and T1-weighted (T1-W) sequences (fig 1).

**Group II, globular thickening with inflammation and edema**

Despite the thickening and the continuity of the heterogeneous internal nature, increased echogenicity of the related area without clear demarcation was observed in addition to hypoechoic cutaneous striations (edematous changes) especially in the cases who presented at 6 weeks or 4-6 months after the trauma. US examination revealed a non-demarcated globularly thickened area. T2-W fat suppressed axial MRI showed increased signal consistent with inflammation and edema, whereas T1-W axial sections showed an oval, demarcated lesion (fig 2).

**Group III, near demarcated lesion**

On US examination, the persistence of thickened subcutaneous fat tissue can be observed. On MRI axial T1-W sequence, inflammation of the fatty tissues was, also no-
An easily recognizable demarcated mass was observed (fig 4). Demarcated but non-encapsulated lipomatous lesions were defined as the “Pseudolipoma” group.

**Group IV, demarcated but non-encapsulated lipomatous tissue mass**

On US/CT/MRI images of the lesion area, an easily recognizable demarcated mass was observed (fig 4). Demarcated but non-encapsulated lipomatous lesions were defined as the “Pseudolipoma” group.

**Group V, immature lipoma**

Lipoma: clearly demarcated capsulated lipomatous tissue mass. The irritating factor which causes repetitive trauma (such as exostosis) could also be visualized (continuous mechanical irritation) (fig 5).

**Group VI, mature lipoma**

This group consisted of cases with mature lipoma, easily recognized by US or other sectional methods; however, the propagation factor could not be visualized. In fig 6 typical encapsulated mature lipomas are shown.

In cases with hereditary multiple subcutaneous lipomas different stages of lesions evolution - from fatty thickening to encapsulated lipomas – can be observed (fig 7).

Being aware of the available data from the literature, and considering our experience, we propose an algorithm for the stages in lipomas development (fig 8).

According to this schema, the inflammatory changes, which progress with interstitial striations beginning with the first trauma, partially regress in the next stage and a demarcated lesion (first pseudolipoma and then lipoma) begins to develop.

In this pictorial assay, we showed the process of post-traumatic lipomas development by analyzing the imagis-
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Although the exact etiology of post-traumatic lipomas remains uncertain, there are theories regarding the mechanical effects of trauma on the formation of lipoma in the literature. Further studies with long follow-up periods, large series of patient groups, and histopathological data are required to verify this theory.

References


Fig 7. High-resolution superficial ultrasonography findings in a a 42-year-old man with hereditary multiple subcutaneous lipomas. Figures marked as a, b, c, and d show lipomatous lesions representing different four stages with different echogenicity and contour characteristics.

Fig 8. Algorithm for the stages in lipomas development