Case-based review

Case key about pseudolipomas pathogenesis

Giulio Gualdi1
Giovanni Damiani2
Paola Monari1
Marco Schiavolena3
Piergiacomo Calzavara Pinton4

1 Department of Dermatology, Spedali Civili of Brescia, Brescia, Italy
2 Department of Dermatology, University of Brescia, Brescia, Italy
3 Department of Pathology, Spedali Civili of Brescia, Brescia, Italy
4 Department of Dermatology, University of Brescia, Brescia, Italy

Address for correspondence:
Giulio Gualdi
Department of Dermatology, “Spedali Civili” of Brescia, Piazzale Spedali Civili, 1
25123 Brescia, Italy
Phone: +39 030 3995019
Fax: +39 030 3995302
E-mail: giulio.gualdi@libero.it

Summary

In soft tissues the most common benign tumor is lipoma and has an incidence of 1%. It affects the entire body and both males and females equally affected, especially in obese patients (1). This neoplastic lesion develops on the fat mesenchimal cells. Predominantly located in the subcutaneous tissue layer, lipomas are rarely present in intermuscular, subfascial, retroperitoneal, mediastinal, gastrointestinal or intraneural areas (2). There are sometimes multiple lipomas, usually painless, asymptomatic, and grow slowly and expand without infiltrating neighboring structures. Pains can occur only when the lesion compresses the nerves causing irritation. When lipomas are located between the skin and the deep fascia, the classical aspect includes a soft, fluctuant feel, lobulation and non-adherent of overlying skin. Lipomas are easily removed surgically because they are well demarcated, disc shaped, round or ovoid, lobulated, yellow masses with a doughty consistency (3). Among lipomas literature shows that some of that is different because are not constituted by a surrounding capsule on magnetic resonance imaging (MRI) and histologically less defined as lipomas (4). The first who suggested a relation between traumatic events and development of benign adipose tumors was Adair in 1932. In contrast to lipomas, pseudolipomas are usually encountered with a strong predominance in female patients (12:1). They are often localized at the lower extremity, and in trochanteric and gluteal regions, so pseudolipomas can be defined as a simple adipose tissue in an abnormal location subjected to a trauma (5, 6).

Although this topic are one of the most discussed, the etiology remained unknown, so in literature there are only hypothesis. However, the first description of PTL pathogenetic mechanism was proposed by Brooke and McGregor: they assumed that soft tissue trauma lead to a prolapse of adipose tissue through Scarpa’s fascia, and finally to pseudolipoma. Most of the information about the topic comes to us from the exposition of case reports asserting three different pathogenetic hypothesis: mechanical, endocrinological and inflammatory. Our case report confirms the inflammatory one.

KEY WORDS: pseudolipoma; post traumatic lipoma; inflammatory theory; squamous cell carcinoma; scalp tumor.

Methods

In December 2010 a 68-year-old male treated for chronic lymphatic leukemia was submitted to a surgical removal of the ulcerated nodular lesion (4x2 cm) in the front-parietal area. The histological exam confirmed the presence of a squamous cell carcinoma poorly differentiated with both lateral and deep borders free of neoplastic remaining (Figure 1). This surgical operation didn’t remove the aponeurotic galea. However, for the second time, one year later the patient went to our dermatological and surgical department with a bulky recurrence on the first removal scar (Figure 2). The patient was re-operated in order to remove this neoplastic lesion and in addition, this time, also the aponeurotic galea was removed. In the galea was found an abundant and lipomatous lesion (Figures 3, 4). A pathological report confirmed the total neoplastic removal and the presence of a pseudo lipomatous lesion under the recurrence of squamous cell carcinoma. A full-body examination of the skin was negative for plaque, scarring, pigmented change, discolored patch, skin thickening, or vascular birthmark.
Case key about pseudolipomas pathogenesis

Results

The skin lozenge (5.5 X 5.5 cm) borders were painted with India ink and analyzed. The pathologist said that the neoplasia was entirely removed and the borders are free of neoplastic remains. However, under the squamous cell carcinoma there’s a fat tissue without a capsule, which was deeply analyzed. Macroscopically the fat mass appeared yellow, not capsulated, soft at the cut and divided by thin fibrotic septa. With the macroscopic characteristics we ruled out both the symmetric lypomatosis and multiple and diffuse lypomatosis because of the number of lesions, the nevus lypomatous cutaneous superficialis for dermal location, and also classic lipomas for the encapsuled pattern (7-10). Microscopically we observed a lobular pattern (Figure 5) with white fat differentiated cells characterized by a diameter up to 200 µm and a single centrally positioned large lipid vacuole and peripherally placed cytoplasm and nucleus. The nuclei are uniform and there’s absence of nuclear hyperchromasia. The microscopy exam exhibited a high vascularization of this fat tissue but wasn’t sufficient to include it into the angiolypomas category, the fibrous septa were insufficient to described it as fibrolypoma (11). We excluded also the mixed variants of lipomas, the white fat cells...
left out the hibernoma, which is composed by brown fat cells. Although the only doubt is with a rare variant of fibrolipomas called sclerotic lipomas, which have a predilection to grow on the scalp or hands of men, this kind of tumor is composed predominantly of a sclerotic fibrous tissue with only focal lipocytic areas (12). In the lesion there was fat tissue with a lobular pattern and the fibrous aspect is confined only to a narrow spray which outlines the structure of a lobul. In conclusion, we inferred the diagnosis of pseudolipoma both from macroscopic and microscopic characteristics.

Discussion

Our experience seems to favor the inflammatory theory, because in non-pathological situations there isn’t adipose tissue under aponeurotic galea, but only periosteum that is composed of two layers: a fibrous layer, with few fibroblasts and many elastic and collagenic fibers, and a cambial one rich in fibroblasts. Adipocytes are absent. The initial report as to the possible role of trauma in the development of lipomas dates back to 1932 when Adair et al. described two patients with lipomas secondary to trauma (13). This case debating Meggitt and Wilson’s hypothesis which suggests that when excessive force is applied locally to adipose tissue, it may cause fat compartments to fracture, shearing of the anchoring between the skin and the deep fascia (14). This leads to pouting of adipose tissue, resulting in PTL after the bruising and haematoma settle down. Our hypothesis was introduced by Campiglio and Signorini: inflammatory background and chemokine network induced by the repairing process can cause the quiescent adipocytes or stem cells to proliferate excessively, finally forming the patient’s PTL (6).

There are many works that evaluated the association between adipogenesis and inflammation (15), in complex situations such as obesity or in laboratory experience with the use of highly flogogenic polysaccharides (16-18). This association was verified diminishing the concentrations of TNF alpha and IL6; the adipogenetic stimulation was dramatically suppressed (19-21). The connection between these two conditions is a change of the microenvironment which causes a stem cell or even a mature cell to develop into an adipocyte. The capacity of a mature cell to differentiate into another mature phenotype (even if it belongs to a different embryological layer) is called Transdifferentiation (22). Periosteum is composed of two layers populated by myofibroblasts and in literature it was demonstrated that in vitro myofibroblasts can develop into adipocytes when differentiation triggers change (23, 24). Moreover, Kim verified two important facts: adipocytes have inflammatory chemokine receptors and they can migrate following the chemokine gradients (25). These results seem to confirm the hypothesis that PTL is caused by an inflammatory condition, which causes a change of the differentiation environment. Recently Turg Carel et al. associated the translocation or partial loss of chromosome 12 with the formation of lipomas (26).

References

Case key about pseudolipomas pathogenesis