
CLINICAL ARTICLE

Morel-Lavallée lesion: A review

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Abstract

Morel-Lavallée lesions are a relatively rare clinical problem, referring to closed degloving injuries around the pelvis or proximal femur, but have also been described in other areas of the body.

The pathology involves a shearing of the hypodermis from the underlying fascia, with disruption of the perforating arteries and lymphatic plexus. In the early stages, there is predominantly accumulation of haematoma in the newly formed cavity, which is later reabsorbed and replaced by slow-leaking lymph.

The diagnosis of an acute Morel-Lavallée lesion is clinical. The chronic Morel-Lavallée lesion, however, can pose diagnostic difficulties due to its resemblance to other soft tissue tumours.

Introduction

Morel-Lavallée lesions are closed internal degloving injuries, secondary to trauma of the proximal femur and pelvis, where the subcutaneous tissue is torn away from the underlying fascia,¹ creating a cavity filled with haematoma and lymph.^{2,3}

Some authors have described Morel-Lavallée lesions in other areas of the body, e.g. the spine, abdomen, knee, lower limb and scapula,⁴ but strictly speaking these are not Morel-Lavallée lesions.

Synonyms include post-traumatic soft tissue cyst, pseudocyst, Morel-Lavallée effusion, and Morel-Lavallée extravasation. Related post-traumatic entities described as ancient haematoma and chronic expanding haematoma can be found in the end stage of some long-standing Morel-Lavallée lesions.^{4,5}

The purpose of this article is to review the management of Morel-Lavallée lesions.

Pathogenesis

A classic Morel-Lavallée lesion consists of a fluctuant, subcutaneous, cystic structure, underlined by a fibrous capsule filled with sterile haemolymphatic or serohaematic content.⁶

The high incidence of Morel-Lavallée lesions around the proximal femur and pelvis has been attributed to the exposed nature of the trochanteric region and proximal thigh, the great extension and firm attachment of the iliotibial band, as well as the relative mobility of the regional skin and the rich pattern of the dermatomal vascularisation.^{6,7,8} The cause of the degloving injury is usually due to a force vector plus a compression and shearing force⁹ in a cylindrical structure, causing a severe traumatic separation of the pannus adiposus of the hypodermis from the underlying deep fascia. This separation of the hypodermis causes a disruption of the vascular and lymphatic plexus, perforating through the fascia lata. A dead space or cavity is created that fills with haematoma, lymph and a mixture of liquefied viable and necrotic fat and debris.^{1,2,4,10}

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Accumulation may develop slowly from shearing of the lymphatics or rapidly from trauma to arterial beds.¹¹

The subcutaneous and dermal tissue of the thigh is largely supplied by perforating musculocutaneous and fasciocutaneous vessels. After disruption, a less organised dermal plexus remains, that becomes the only blood source to the superficial tissues, in some cases leading to skin necrosis.¹²

Factors in the blood clotting cascade and blood breakdown products are said to be involved in a chronic inflammatory reaction. This may cause further bleeding, and a peripheral capsule with granulation tissue, capillaries and fibrosis originates. This reaction may account for the occasional slow growth and self-perpetuation of this haemolympathic mass.^{4,5,6}

Clinical diagnosis

Morel-Lavallée lesions are uncommon but form part of the major soft-tissue injuries around the proximal femur and thigh. Even with underlying pelvis fractures the entity may be missed on initial evaluation as bruising may take several days to develop, which makes the clinical diagnosis difficult.⁵ Hudson *et al*⁹ reported a delay in diagnosis for one-third of the patients in their series. He also found an increased incidence in females, as did Tsai *et al*. This is attributed to the difference in anatomical distribution of subcutaneous fat.^{13,14}

The patient can present immediately with a Morel-Lavallée lesion or days after the trauma. The average size is 30 x 12 cm.¹ Acute presentation encompasses a soft fluctuant area of variable ecchymosis;¹ skin hypermobility;^{3,13} soft-tissue swelling; palpable bulge; abrasions; local contusions and hypoaesthesia near the region of trauma.^{9,12}

Chronic Morel-Lavallée lesions are defined as presenting one month to many years after the initial trauma event. The lesions may decrease in size, remain stable or show a slowly progressive enlargement. As it enlarges and becomes chronic, it may become painful and firm, and this may lead to a misdiagnosis of a soft tissue tumour. Morel-Lavallée lesions are not associated with lymphadenopathy. Further long-term presentation includes contour deformity, infection or necrosis of the underlying skin.^{5,12,15}

Diagnostic procedures

A variety of diagnostic modalities have been used to aid in the diagnosis. These include non-invasive approaches such as ultrasound, magnetic resonance imaging (MRI) and computed tomography (CT) scans, as well as invasive procedures such as injection of contrast medium into the cavity.⁸ MRI is the diagnostic image modality of choice in the assessment of a Morel-Lavallée lesion in the hip region.⁶ The diagnosis can usually be made on the history, examination and characteristic location, supplemented by the MRI features if necessary.

Lab tests

Coagulopathy-related haematoma are frequently disproportionate to the degree of trauma and may present without a traumatic cause. It is thus important to distinguish them from Morel-Lavallée lesions. Clinical distinction is usually on history and laboratory tests.^{6,16}

Needle aspiration

In uncertain cases (usually obese patients), a needle aspiration can be done on admission to confirm the presence of blood.¹

Biopsy

Biopsy may be needed to rule out soft-tissue tumour.⁸

Plain radiography

Plain radiography may reveal a non-specific, non-calcified soft-tissue mass that displaces but does not infiltrate the layers of fat.^{6,8}

Sonography

Ultrasound can confirm the presence of a suspected lesion and determine its size, volume and configuration. The lesion is located superficial to the muscle layer and deep to the hypodermis. Depending on the age of the haematoma, the ultrasound features may vary from anechoic to hyperechoic,^{8,11} and may contain connective tissue and fat globules.^{8,11,17}

In the chronic stage, an organised haematoma is seen with possible features of liquefied subcutaneous haematoma and sometimes a fluid-fluid level. Remnants of fat may be present in the wall of the mass and no Doppler flow activity is seen in the lesion or on the periphery.^{4,17}

Kalaci *et al* described features consistent with a complex cystic mass,⁸ which were also described by Gilbert, Bui-Mansfield *et al* in a 22-yr-old male who developed a Morel-Lavallée lesion ±1month after a motor vehicle accident.¹¹

CT

CT of a Morel-Lavallée lesion may show a fluid-fluid level, resulting from sedimentation of cellular blood components, and a capsule may surround the mass. Therefore, the finding of a capsule on imaging could be used to help select the appropriate course of treatment.^{11,17}

MRI

The appearance of Morel-Lavallée lesions on MRI is strongly influenced by the signal patterns of haemorrhage, and by those originating from the magnetic properties of the products of haemoglobin breakdown. The signal intensity of the content thus varies according to the age of the lesion.^{4,5,8}

Because the progression of the lesion from an initial hyperacute haematoma to a chronic organised haematoma and then eventually to a serosanguinous type lesion occurs slowly, it should be taken into account that the MRI features are not always specific, but can indicate mixed components of the different stages.

Initially in a hyperacute haematoma (hours old) the main component is oxyhaemoglobin, making the lesion hypointense on T1-WI (weighted images) and hyperintense on T2-WI. An acute haematoma (days old) is hypointense on both T1-W and T2-W MRI sequences.⁵

Subacute haematomas (weeks old) show homogenous hyperintensity on both T1-W and T2-W MRI sequences, due to the presence of methaemoglobin on the periphery, which produces a hypointense “concentric ring”. As the haematoma evolves, it becomes progressively encapsulated and the MRI features subsequently change.^{4,5}

A haematoma is considered to be chronic when it remains 1 month after the initial haemorrhage. The blood is mainly reabsorbed but the presence of haemosiderin deposits peripherally, granulation tissue, necrotic debris, fibrin and blood clots in the residual fluid are characteristic of a chronic organising haematoma.⁵ The lesion can be progressively encapsulated by a fibrous capsule and capillary formation within the lesion is possible.^{4,5} These lesions can appear homogeneously hypointense on T1-WI and hyperintense on T2-WI.

Ancient haematoma and chronic expanding haematoma have been described as post-traumatic entities and can be found in the end-stage of some long-standing Morel-Lavallée lesions.^{4,5,6,8}

As the lesion evolves, blood is largely reabsorbed and, due to the continuous leakage of lymph, replaced by a serosanguinous fluid. The lesion progressively becomes lined with a fibrous capsule.^{5,6} This may present with water-like MRI signal characteristics and a hypointense peripheral ring.⁶ Such lesions closely correlate with partially encapsulated serosanguinous fluid or seroma and probably accounts for the long-standing nature of the lesion.

MRI may help to characterise long-standing Morel-Lavallée lesions, particularly when progressive growth or pain clinically mimic soft-tissue tumour.

In a patient with a history of trauma to the proximal femur, the presence of a moderately expansive subcutaneous oval or fusiform encapsulated lesion (diameter ranging from 10-29 cm) located in the perifascial plane adjacent to the fascia lata, may reflect a long-standing Morel-Lavallée lesion.⁵ The adjacent muscles can be slightly deformed secondary to compression, and the presence of an internal septum has been described in some cases.^{4,5,6}

Longstanding Morel-Lavallée lesions also may present more atypical MRI features. These closed laceration lesions show a combination of perifascial dissection and closed fatty tissue laceration, with or without associated serous/haemorrhagic collection.

Another variation has a perifascial pseudonodular appearance and occasionally shows irregular peripheral enhancement and skin retraction.⁶ The MRI signal characteristics of subcutaneous fat necrosis are highly variable, depending on the age of the injury and may occasionally resemble a pseudonodular Morel-Lavallée lesion.

The last atypical features to be identified by Mellado corresponded to an infected Morel-Lavallée lesion. It may present as a thick enhancing peripheral capsule with internal septations; an inflammatory reaction in the adjacent fatty tissue and fascia; peripheral fluid leakage and sinus tract formation.⁶

Treatment

The management of acute Morel-Lavallée lesions is controversial. In the last decade, there has been an evolution in the surgical treatment from aggressive debridement with wound healing by secondary intention, to a more minimally invasive approach. The main concerns in acute lesions are possible sepsis, skin and soft-tissue sloughing and the timing of surgery for the associated fractures. Surgery can be performed as a single step or two-stage procedure, depending on the extent of the injury.^{1,3} Studies have been done to determine the outcome of different approaches to treatment, but unfortunately these are few and mostly retrospective.

Morel-Lavallée lesions are uncommon but form part of the major soft-tissue injuries around the proximal femur and thigh

Most authors have agreed that, once the injury is identified, the haematoma must be evacuated and necrotic tissue removed.^{1,7} Neglected lesions can become infected, complicating the management further.¹ It can be assumed that colonisation can take place some time after the haematoma is created, probably as a result of circulating bacteria that are present after major trauma.¹ Accordingly, it would seem that if the diagnosis is made early, there is less time for colonisation to occur and there may be a lower rate of infected lesions if treatment is initiated sooner, rather than later.¹

Tseng and Tornetta conducted a level 4 prospective/retrospective study in 2006, involving 19 patients with Morel-Lavallée lesions. Fifteen patients had associated pelvic or acetabular fractures, and the degloving lesion averaged 30 x 12 cm. They performed percutaneous drainage via two 2 cm incisions (one distally and the second as supero-posteriorly as possible) on all the lesions within the first three days. In some cases where the lesion was extensive, three incisions were used, but the author does not mention the specific size of those lesions.

The haematoma then gets drained with a suction catheter inserted from the one end of the lesion to the other. A plastic brush for debridement of the fatty tissue was used and the wound was then washed with pulse lavage from distally to proximally, depending on the direction in which flow was most adequate. Rinsing was done until the fluid came out clear (usually 5ℓ of saline was enough). The wounds were closed and a percutaneous drain was placed across the lesion for up to two weeks or until drainage was <30 ml in 24 hrs. Postoperatively the drain was connected to wall suction and a cephalosporin was given for 24 hrs intravenously.¹

Minimally invasive surgery (e.g. percutaneous fixation of the posterior ring of the pelvis) was performed together with the percutaneous debridement. None of these patients with percutaneous fixation had wound breakdown. Extensive surgery (e.g. acetabular fixation) was performed >24 hrs after the removal of the suction drain, and in three patients that had surgical incisions directly through the Morel-Lavallée lesion no deep infection occurred postoperatively.

Only three out of the 19 patients (15%) had a positive culture from their debridement specimens. Hak *et al* had a significantly higher incidence of infection post debridement (11/24; 46%).³ This was most likely due to his delayed surgical and/or extensive open debridement of the Morel-Lavallée lesion after ±13 days, which can lead to disruption of the remaining subdermal plexus and violation of the already traumatised skin.^{1,10}

Early percutaneous drainage proved to be safe and effective in minimising complications such as infection and skin necrosis, and Tornetto's approach to the surgery in either one or two stages was well tolerated and safe.¹ His approach is the current preferred method of treatment.

Conservative treatment with compression dressings have proven to be successful in variable case studies in the treatment of small, acute Morel-Lavallée lesions.^{13,18,19} This can be combined with repeated ultrasound guided aspiration on an outpatient basis, until the lesion resolves.¹⁸

Chronic Morel-Lavallée lesions present a cosmetic problem due to the huge contour deformity^{5,9,12,15} and the recurrence after drainage. The lesion is resistant to conservative treatment because of the thick fibrous capsule containing it and the continuous leak of lymph that makes its resolution difficult.¹⁶

Hudson had success with liposuction in improving the silhouette of chronic lesions but only when the defect was soft with a smooth contour. An open incision was required for irregular firmer deformities or where fat necrosis and heterotopic calcification occurred.⁹

Complete excision of the lesion with or without cutaneous fascial sutures to obliterate dead space^{7,8} appears to be the most successful and is the current preferred method of treatment.

In 2006 Shai *et al* used talc sclerodhesis to limit the potential space for fluid accumulation, and all four of the chronic lesions he treated resolved. One patient developed infection and was treated with open debridement.¹⁶

In conclusion, a high index of clinical suspicion is needed in the acute setting with early minimally invasive percutaneous debridement. Depending on the extent of the surgery, fixation can be done at the same time as the debridement or up to two weeks post debridement. Fluid for cultures must be sent during debridement and treated accordingly. Chronic lesions must be distinguished from soft tissue tumours and final treatment lies with surgical excision,^{7,8} although talc sclerodhesis can be considered as an alternative.¹⁶

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