Lumbar subcutaneous edema

Posterior lumbar subcutaneous edema (PLS) is a very frequent finding on MRI of spine and is usually considered an incidental finding.

Shi et al. 1) found that PLS edema occurred more frequently and more severely in overweight patients. It is in the extremely obese patients (BMI > 40), in which the most pronounced incidence of edema is expected. Obesity impedes lymphatic flow, leading to the collection of protein-rich lymphatic fluid in the subcutaneous tissues, which frequently results in lymphedema 2).

In addition, Shi et al. 3) found that PLS edema was significantly more severe in women and in older patients.

The degree of edema was not found to be correlated with sex or age in the study of Han et al. Furthermore, no correlation was found between edema or the fluid collection degree on MR images and clinical findings 4).

However, the greater severity of subcutaneous edema in the cases with lower back pain (LBP) might have been due to poorer lymphatic drainage in these patients due to pain-related physical activity restriction 5).

In the cases without other nearby abnormal findings, the posterior lumbar soft-tissue edema may have been caused by a general condition. Some conditions, such as diabetes mellitus (DM), liver cirrhosis (LC), congestive heart failure (CHF) or other heart problems, can cause diffuse subcutaneous edema in any region of the body, particularly in the dependent regions. Furthermore, 33 cases had an underlying disease in the study, and 28 of these had DM. Edema is common in patients with LC or heart failure; yet, in the present study, only five cases had LC and six had heart failure. However, this may be due to a lack of spine MR examinations in LC or heart failure patients.

Edema in CHF is the result of the activation of a series of humoral and neurohumoral mechanisms which promote sodium and water reabsorption by the kidneys and the expansion of extracellular fluid. Furthermore, edema in LC is the result of sodium retention and is caused by mechanisms that lower the effective arterial blood volume, such as the overproduction of vasodilatory factors, by a damaged liver. These factors primarily lead to a marked fall in mesenteric vascular resistance and blood pooling as well as to a decrease in total peripheral vascular resistance 6).

Clinical correlation

A clinical correlation is almost always required to identify the significance of this, in particular in the overweight, elderly and bedridden. However, before attributing this oedema to physiological phenomena, it is necessary to make sure that there are no local underlying disorders, such as myositis, recent surgery, interspinous infectious diseases, etc. 7).

Radiographic features

Cooper et al. 8), reported that lumbar edema accumulates at the subcutaneous fascia plane on CT images. Nevertheless, MRI is a well-established imaging modality for the evaluation of acute soft tissue disorders due to its excellent contrast resolution, high sensitivity for pathologic fluid accumulation, and its ability to depict abnormal tissue compartments. Furthermore, in this context, it has been shown to be superior to other cross-sectional imaging techniques, such as the ultrasound and the CT 9).
MRI

It is the same appearance as that of edema elsewhere in the body.

T2 and STIR: hyperintense signal

T1: hypointense signal

Due to its distribution it is sometimes referred to as tramp-stamp oedema.

On a routine basis, we are often confronted with the presence of a T2-STIR hypersignal at the subcutaneous lumbar fat, that is discovered by chance and that, in rare cases may be mistaken for inflammatory or infectious infiltration. This T2 hypersignal accumulates along the superficial fascia, separating the subcutaneous fat tissue into a deep and a superficial layer. Few studies have described this anomaly by slice imaging.

The severity of posterior lumbar subcutaneous edema and the volume of fluid collections on MR imaging are associated with increased weight \(^{10}\).

As opposed to the results published by Shi et al. Genu et al. did not find a correlation with sex. In fact, the non-equal distribution of fat tissue between the sexes may have had an effect on the occurrence of the oedema. In addition, a correlation was not found between this oedema and the time of the examination. The latter data seems to be biased since the time of the examination is not representative in the hospitalised patients due to prolonged bed rest \(^{11}\).
