Ultrasonographic features in a patient with long-standing lymphedema

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Case

A 41-year-old male patient with a past medical history of fragile X syndrome, intellectual disability, Hodgkin's lymphoma, hepatitis C, and liver cirrhosis presented with altered mentation, breathlessness, progressive leg pain, erythema, and swelling for a week suggesting cellulitis with sepsis. He was normotensive but had tachycardic 125 beats/minutes, tachypneic 34 breaths/minutes, and a low-grade fever of 100.4°F. Upon examination, he was drowsy. Lower extremities were notable for 3+ pitting edema, especially on the left leg (Figure 1). Lab results were remarkable for WBC of 2.6 $\times$ 10^9/L, platelets of 31,000/µL, procalcitonin of 29 ng/mL, high anion gap metabolic acidosis with bicarbonate of 16 mmol/L and lactate of 8.3 mmol/L. Imaging was performed with ultrasonography (US) and computed tomography (CT), and results were typical for severe lymphedema (Figures 2–3). An isotonic fluid bolus and empirical antibiotics were started for the treatment of cellulitis with probable sepsis associated with chronic lymphedema; his hemodynamic status improved with this treatment.

Figure 1. Bilateral non-pitting edema extending from the leg to upper thigh, more prominent on the left side.

Discussion

Lymphedema (LED) is usually diagnosed by clinical history and physical examination; however, several imaging modalities, including lymphangioscintigraphy, CT scans, magnetic resonance imaging, and US, can help establish this diagnosis.[1,2] Ultrasonography may be the modality of choice due to its availability, non-invasiveness, cost-effectiveness, and its use to monitor therapeutic responses.[1] Although unable to visualize lymphatic truncal anatomy, LED can be indirectly diagnosed with the duplex US by evaluating surrounding skin, subcutaneous tissues, lymph nodes, and venous insufficiency, which is a more common cause of edematous limb.[2] Ultrasonographic findings suggestive of LED include skin and subcutaneous thickening, subcutaneous echogenicity, subcutaneous fluid accumulation, and stone-paved appearance.[1,3] In a study with 35 patients with secondary lymphedema, increased subcutaneous echogenicity, which is due to diffuse fibrosclerosis, was linearly correlated with the severity of LED according to the International Society of Lymphology (ISL) clinical stage which ranges from preclinical (stage 0) to lymphostatic elephantiasis (stage III).[3,4] Recently, ultrasound elastography has been suggested as a promising tool in moderate-to-severe LED to assess skin and subcutaneous tissue strain, in which lower strain represents stiffness and less deformity due to fibrosis.[5]

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DOI: 10.12746/swrccc.v10i43.1029

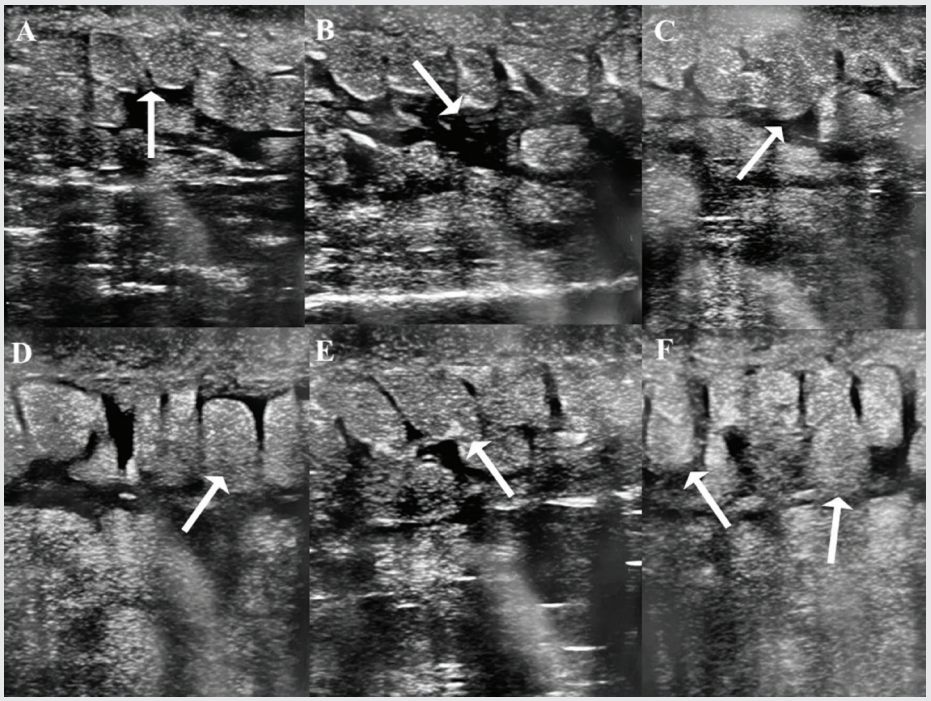

Figure 2 (Panels A–F). Ultrasonography of the lower limb showing increased skin and subcutaneous tissue thickness and increased subcutaneous echogenicity representing increased subcutaneous fluid in a cobblestone paved appearance (Arrows).

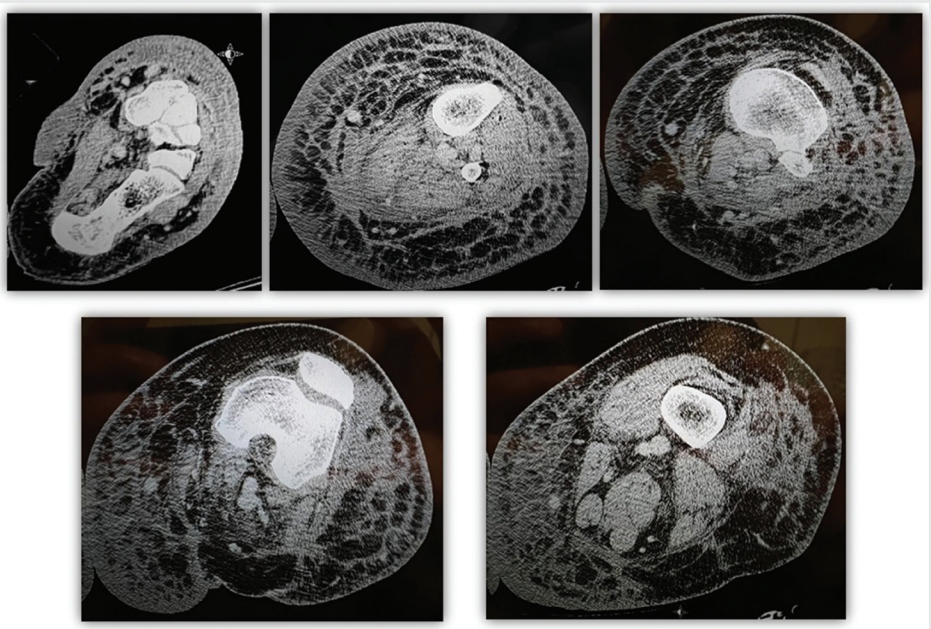

Figure 3. Computed tomography of the left lower extremity showing honeycomb pattern of subcutaneous edema and skin thickening extending from foot to upper thigh.

Consent: Informed written consent was obtained from the patient.

Keywords: Lymphedema, tissue fluid, ultrasonography, edema

Article citation: Guerin CM, Benjanuwattra J, Maylath JS, Abdelnabi M. Ultrasonographic features in a patient with long-standing lymphedema. The Southwest Respiratory and Critical Care Chronicles 2022;10(43):60–62
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Submitted: 3/3/2022
Accepted: 3/20/2022
Conflicts of interest: none

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