Lymphology 25 (1992) 129-133

ULTRASONOGRAPHY OF EXTREMITY LYMPHEDEMA

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ABSTRACT

Ultrasonography of the extremities was performed in 91 patients with unilateral or bilateral peripheral lymphedema of the arms or legs. Linear 3.5 to 10 mHz ultrasonographic linear probes were used in accordance with standardized procedure. The data demonstrated a volumetric increase of the lymphedematous limb with increased thickness of both the subcutaneous and subfascial (muscular) compartments consistent with fibrosclerosis in both compartments with chronic disease. Whereas dermal thickening was minimal, subcutaneous and subfascial changes were more prominent in primary than secondary lymphedema.

By providing information about the volumetric and structural alterations with chronic lymphedema, ultrasonography safely and simply supplements conventional and isotopic lymphography in assessing patients with chronic lymphedema.

Lymphedema is the abnormal accumulation of proteins and water in the interstitial space as a result of insufficient lymphatic drainage. In the extremities, lymphedema is usually separated into primary (ductal aplasia, hypoplasia, or congenital malformations) or secondary abnormalities of the lymphatic system due to infection, inflammation, trauma, surgery, irradiation, or neoplasia. Classical imaging in patients with lymphedema entails conventional or direct lymphography (1) and more recently lymphoscintigraphy (2). These methods generally permit anatomic and functional insight into lymphatic perturbations. Because ultrasonography is a simple, safe, and easily repeated technique to examine the soft tissues, we evaluated the utility of this imaging technique for assessing the changes that occur in the superficial and subfascial (muscular) compartments with peripheral lymphedema.

MATERIALS AND METHODS

Ultrasonography of the lymphedematous extremity was carried out by placing a small parts 3.5-5 or 10 mHz linear probe directly on the skin and the different peripheral lymph segments examined as follows: upper extremity—transaxial scans were taken of both the forearm and arm 10cm proximal and distal to the humoral epicondyle with the arm fully extended and the hand open. Lower extremity: calf—transaxial scans were taken with the patient prone from the posterior portion of the calf, 20cm proximal to the lateral malleolus. The probe was placed to ensure that the tibia and fibula were at the same level with a frontal angle of 45°. Thigh_transaxial sections were taken from the anterior surface, 20cm cephalad to the head of the fibula. Ultrasound testing was done on a soft foam rubber table and excess pressure avoided so as not to distort the ultrasonographic images (3,4). A total of 91 patients were studied (32 men, 59 women) with a mean age of 42.5 years. Fifty nine had unilateral leg lymphedema; 18 had bilateral leg lymphedema; and 14 had unilateral arm lymphedema. With unilateral lymphedema, the contralateral limb was used for purposes of comparison whereas with bilateral leg lymphedema comparisons were made with subjects without edema of the legs.

RESULTS

Volumetric changes

Dermis—In general, dermal thickening in lymphedematous limbs was minimal with the average increase in thickness only 1-2mm (i.e., 3-4mm thick) compared with the normal dermis of 2mm thickness.

Subcutis —Thickness of the subcutaneous tissue was uniformly increased and except for the area adjacent to the tibial plateau was evenly distributed throughout. In primary lymphedema of the arm, the average increase was 21% (8 patients) whereas in secondary lymphedema, the average increase was 54% (19 patients). Similarly in leg lymphedema, the subcutaneous layer was increased 79% (50 patients) in primary lymphedema and 46% (16 patients) in secondary lymphedema *(Figs. 1-6).*

Muscle—Changes in the subfascial compartment were complex. In most patients with primary lymphedema muscle mass was increased but in 25% the muscle compartment was decreased or unchanged. In contrast, in secondary lymphedema muscle mass was increased in only one-half the patients and was the same or decreased in the others.

Structural changes

Ultrasonography usually depicted a mild hyperechogenic dermis, hypoechogenic subcutaneous layer, and reflective streaks and hyperechogenicity of the muscle compartment *(Fig. 1).* Although the changes in the subcutaneous and subfascial compartments were more consistently seen with primary as compared with secondary lymphedema, lymphedematous extremities often showed increased volume of both superficial and deep layers with diffuse hypoechogenicity often obscuring the distinction between the superficial and deep muscular fascia *(Figs. 2-6).* Increased streaking and "atypia" in both the superficial and deep compartments with chronic disease was consistent with diffuse fibrosclerosis *(Fig. 6).*

DISCUSSION

Conventional oil contrast lymphography and more recent lymphangioscintigraphy are first-line tests for assessing structure and function of the lymphatic system (1,2). Conventional lymphography, however, is cumbersome, associated with infrequent but definite complications, is not easily repeated, and at times is unsuccessful. Lymphoscintigraphy, on the other hand, provides a suboptimal view of the lymphatic collectors. Neither study enables visualization of the dermis, subcutaneous, or subfascial (muscular) compartments in lymphatic disorders. Other techniques such as lymphomanometry or direct pressure recordings of intralymphatic pressure and xeroradiography with its high cost and exposure to irradiation are less commonly utilized. Whereas ultrasonography does not yield direct information about lymphatic truncal anatomy and structure, this technique permits an accurate assessment of soft tissue changes in the superficial and deep layers. Moreover, because ultrasonography is safe and simple, it potentially can be used to evaluate the results of a wide variety of operative and nonoperative therapies commonly proposed for management of intractable lymphedema.

It is mandatory that in using ultrasonography for purposes of comparison among patients that a standardized approach as outlined be followed (5,6). Our findings indicate that dermal thickening is much less prominent than previously thought. Much of the thickness in the superficial layer with lymphedema, and especially secondary lymphedema, is in the subcutaneous tissue. In 80% of patients with

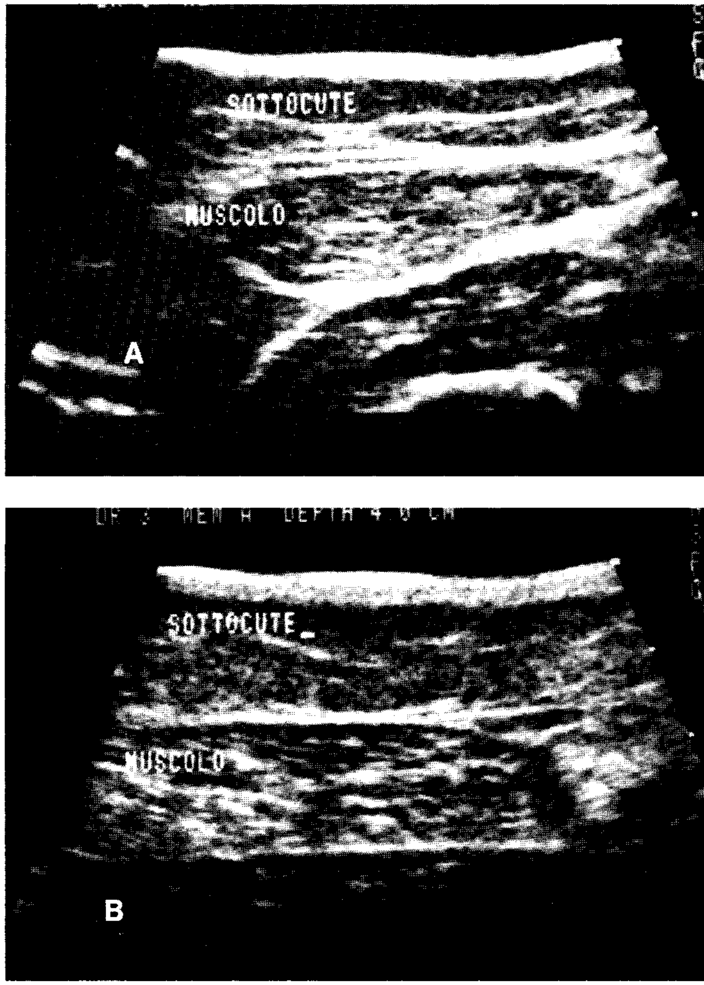

Fig. 1. Ultrasonography of the normal leg. A — transverse scan (10 mHz linear probe); B — longitudinal scan. Note hyperechogenic dermis, hypoechogenic subcutaneous tissue (sottocute) with hyperreflecting streaks and hyperechogenic muscle (muscolo).

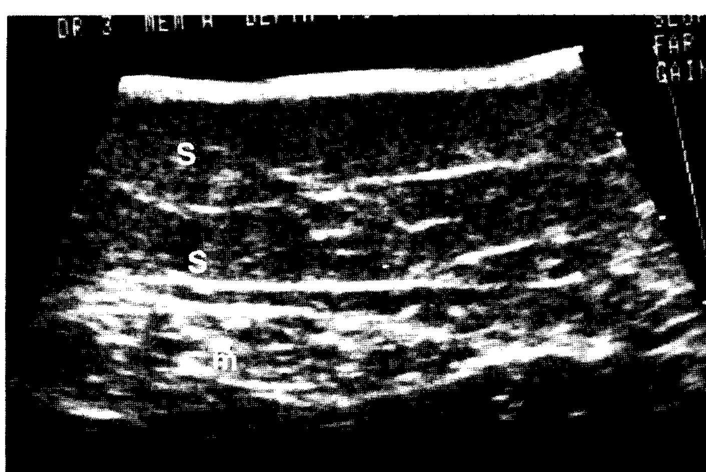

Fig. 2. Ultrasonography of leg with primary lymphedema — longitudinal scan. Note the increased thickness of the subcutaneous tissue (S) and decreased muscle thickness (M).

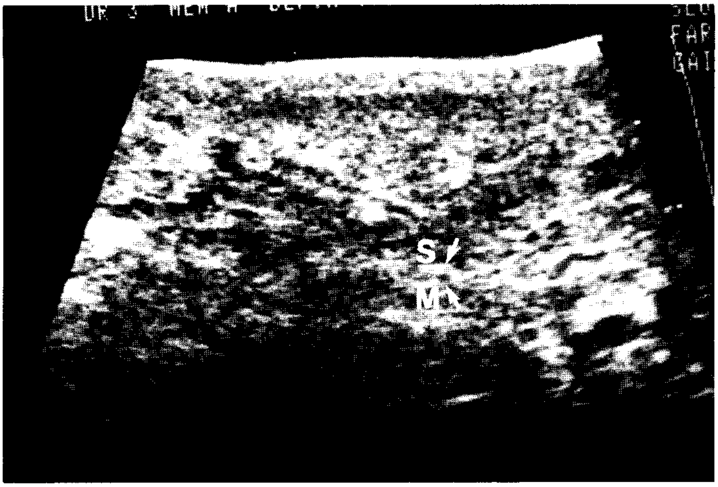

Fig. 3. Ultrasonography of leg with primary lymphedema — transverse scan. Note hyperechogenic subcutaneous tissue (S) and atypical muscular reticulum (M).

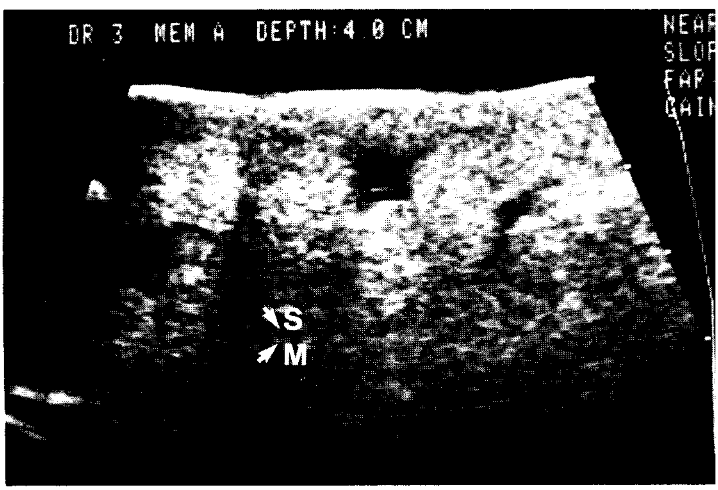

Fig. 4. Ultrasonography of leg with primary lymphedema — transverse scan. Note hyperechogenic subcutaneous tissue (S) and atypical muscular reticulum (M).

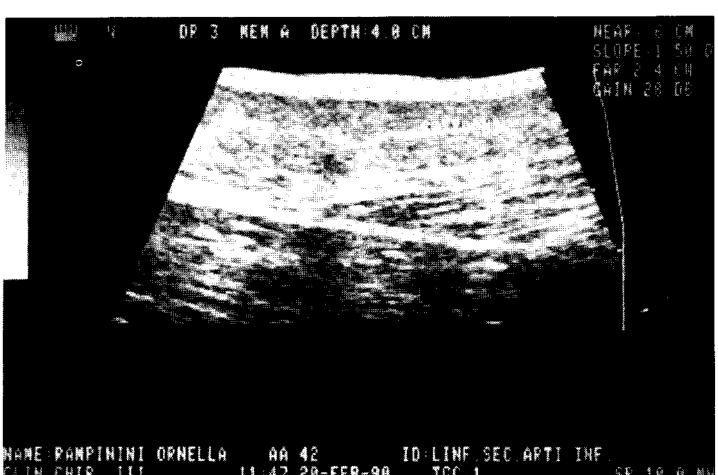

Fig. 5. Ultrasonography of leg with secondary lymphedema— longitudinal scan. Note hyperechogenic subcutaneous tissue (sottocute) and muscle with normal reticulum.

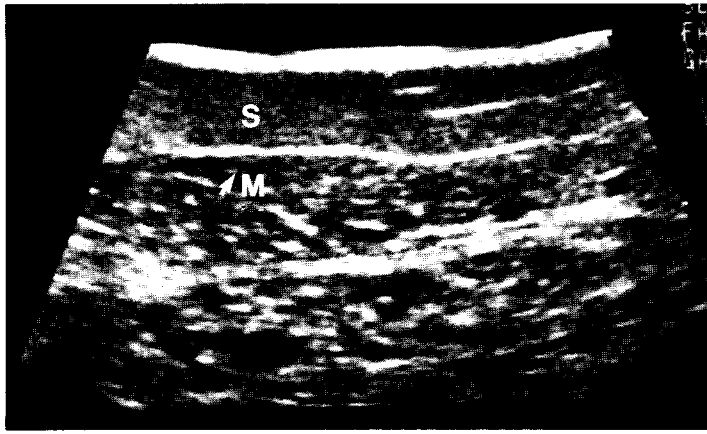

Fig. 6. Ultrasonography of leg with primary lymphedema — longitudinal scan. Note normal echogenic subcutaneous tissue (S) and muscular tissue (M) with atypical reticulum. Both the subcutaneous tissue and muscle demonstrate increased thickness.

primary lymphedema and in 50% of patients with secondary lymphedema, the subfascial (muscle) compartment is also volumetrically increased. Whereas ultrasonographic changes of hyperechogenicity and "atypia" suggest elements of fibrosclerosis in both these layers, it is possible that some of the volumetric increase in muscle mass is related to work hypertrophy as the patient negotiates a heavy, unwieldy extremity. Nonetheless, subfascial as well as subcutaneous alterations need to be reconsidered and followed after nonoperative and operative methods designed to alleviate peripheral lymphedema. Moreover, the architectural changes seen on ultrasonography are consistent with the pathogenesis of lymphedema, that is, of stagnant plasma proteins trapped in the interstitium, activated hyaluronic-acid secreting mast cells, and deposition of collagen fibers (fibrosclerosis). Indeed, in earlier studies we demonstrated the close correlation between ultrasonographic findings and histopathology by light microscopy after true cut needle biopsies of the superficial and subfascial compartments (7). Thus, where ultrasonography depicted hyperechogenicity with "atypia" light microscopy verified fibrosclerosis (7) whereas with mild or no hyperechogenicity, or with little or no "atypia," fibrosclerosis typically was minimal or absent (8).

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