Lymphology 18 (1985) 86-89

PRIMARY LYMPHEDEMA OF THE LEG: RELATIONSHIP BETWEEN SUBCUTANEOUS TISSUE PRESSURE, INTRAMUSCULAR PRESSURE AND VENOUS FUNCTION

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ABSTRACT

In eight patients with unilateral primary lymphedema, subcutaneous tissue and intramuscular pressure were measured in both legs using the slit-catheter technique. Venous function was assessed by venography, or Doppler or photoplethysmography. Both at rest and during exercise, subcutaneous tissue pressure was elevated in the lymphedematous leg (17.9±7.6 and 33.0±10.8 mmHg respectively) compared to healthy contralateral leg (-0.4±2.6 and −0.6±3.6 mmHg; $p < 0.001$). The intramuscular pressure in the anterior tibial compartment was also increased at rest and during exercise in the edematous leg (24.9±4.4 mmHg and 43.6±11.2 mmHg respectively) compared to control leg (9.6±5.6 and 25.8±10.00 mmHg). These findings suggest that derangements in both the superficial and deep lymphatic systems as well as venous dysfunction contribute to the clinical appearance of "primary lymphedema."

Lymphatic occlusion increases tissue concentration of protein and colloid osmotic pressure thereby favoring a rise in volume of interstitial fluid (1). Qvarfordt et al have shown that intramuscular pressure is elevated in patients with secondary lymphedema (2). Both the venous and lymphatic system work together to reduce accumulation of tissue fluid (3) and it is possible that derangements in both systems contribute to lymphedema (4).

The present investigation was done to evaluate pressure relationships within the superficial subcutaneous and intramuscular compartments and to correlate the findings to venous function in patients with unilateral primary lymphedema of the lower extremity.

MATERIALS AND METHODS

Eight patients, (five males and three females), aged 29-60 (mean 43) years, with unilateral primary lymphedema of the leg were studied. The duration of lymphedema was 9.7±4.2 years. Leg swelling was pronounced with an increase in calf circumference of 9.2±5.7 cm in the diseased leg compared to the uninvolved contralateral leg. Each patient had primary lymphedema with a predominance of left-sided involvement (6 patients). The diagnosis was established by patient history, physical examination and confirmed by radioactive isotope lymphangiography according to Seki (5). The latter test revealed lymphatic obstruction in six and impaired lymphatic flow in two patients.

A. *Venography:* Standard ascending venograms were done in six patients prior to subcutaneous and intramuscular pressure measurements.

B. *Non-invasive vascular assessment:* Doppler studies were performed to assess patency and competency of peripheral veins while photoplethysmography was done to evaluate venous refilling time. Arterial inflow was assessed by pedal pulse palpation and the ankle/ brachial pressure index.

C. *Subcutaneous tissue and intramuscular*

pressure measurements: The slit-catheter technique, previously described by Rorabeck et al (6) was used to measure both subcutaneous tissue and intramuscular pressures bilaterally. A slit-catheter was introduced through a sterile saline-filled 16-gauge epidural needle initially into the subcutaneous compartment after intracutaneous infiltration of local anesthesia, with the tip of the catheter 8 cm distal to the site of skin penetration. The epidural needle was then removed leaving the slit-catheter in place. Thereafter, no fluid was instilled. To avoid tissue fluid leakage the catheter was fixed to the skin with surgical tape covering its dermal entrance. Pressures were recorded with an electromagnetic transducer and recorder. The zero level was taken on a plane with the tip of the catheter both in the supine and erect position at rest.

Recording was performed until a steady pressure was reached. Thereafter, the subcutaneous tissue pressure was measured during heel-raising (60/min) for five minutes or until leg pain forced the patient to stop. After heel-raising the subcutaneous tissue pressure was followed for ten minutes. The slit-catheters were then removed and using the same technique new catheters were introduced through the same skin puncture, but this time into the anterior tibial muscle compartment. Intramuscular pressures were obtained using the same protocol outlined above for measuring subcutaneous tissue pressure.

RESULTS

A. *Venographic findings:* Ascending venography in four patients showed dilated, tortuous, patent veins without postphlebitic changes. In the other two patients the venograms were unremarkable.

B. *Non-invasive venous assessment:* Doppler studies revealed patent peripheral veins in both diseased and healthy (control) legs. In the diseased leg an incompetent popliteal vein was detectable in half the patients. Photoplethsmography disclosed a shortened refilling time (9±4 sec) after exercise compared with the normal side (20±8 sec) compatible with some venous incompetency. Distal pulses were consistently palpated, and the ankle/brachial pressure index was normal with no differences between sides.

C. *Tissue pressure measurements:*

1. Subcutaneous tissue pressure — After an initial insertion artifact, subcutaneous tissue pressure stabilized after 1-5 minutes. Patency of the catheter was checked by slight leg external compression and decompression. At rest with the patient in a supine position, the subcutaneous tissue pressure of the edematous leg was 14.8±6.8 mmHg, and was not changed with pressure while standing at rest (17.9±7.6 mm Hg). These resting values in the diseased leg, however, were considerably higher than corresponding values in the healthy contralateral leg (-0.6±1.8 and -0.4±2.6 mm Hg respectively; $p < 0.001$). Moreover, during exercise, the subcutaneous tissue pressure rose sharply in the diseased leg (33.0±10.8 mm Hg) but not in the control leg (-0.6±3.6 mm Hg; $p < 0.001$).The pressure return time ($T\frac{1}{2}$) was 1.2±1.4 seconds in the control leg but considerably prolonged in the diseased leg (55.6±30.0 seconds; $p < 0.001$) (Fig. 1).

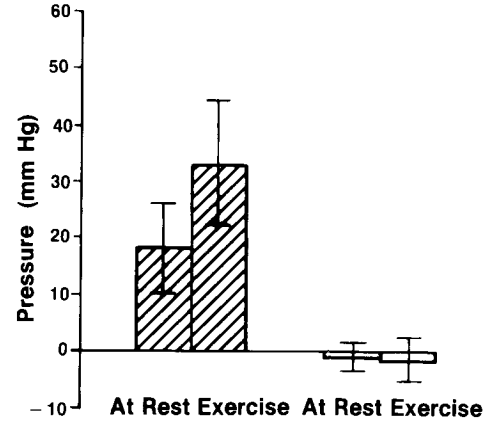

Fig. 1: Bilateral leg subcutaneous tissue pressure with primary unilateral lymphedema in eight patients while standing at rest and during exercise (mean±SD).Striped bars at left are diseased legs and clear bars at right are control legs.

2. Intramuscular pressure — At rest in the edematous leg, the anterior tibial compartment pressure was 20.8±5.6 mm Hg in the supine position and 24.9±4.4 mm Hg while erect. During exercise the level rose sharply to a maximum value of 43.6±11.2 mm Hg ($p < 0.001$).

In the healthy (control) leg the pressure rose from 9.6±5.6 mm Hg at rest with the patient standing to 25.8±10.0 mm Hg during exercise ($p < 0.01$). Intramuscular pressures were significantly ($p < 0.001$) higher in the diseased compared with the healthy leg both at rest and during exercise (Fig. 2). The return time (T½) to preexercise pressure values was significantly prolonged in the diseased extremity (187.5±73.1 sec.) compared with the control leg (8.3± 7.0 sec.; $p < 0.001$).

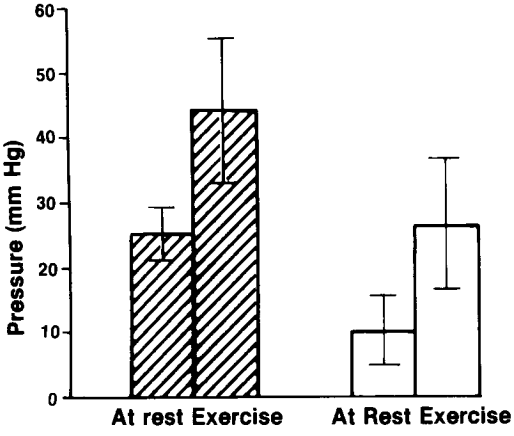

Fig. 2: Bilateral anterior tibial compartment intramuscular pressures in eight patients with primary unilateral lymphedema while standing at rest and during exercise (mean±SD).Striped bars at left are diseased legs and clear bars at right are control legs.

DISCUSSION

The slit-catheter technique is useful for measuring intramuscular and subcutaneous tissue pressures. This method not only enlarges the area of contact with tissues and thereby facilitates reaching pressure equilibrium, but it also consistently yields at rest a negative tissue pressure compared with a simple capillary tube (7). The method is also easily adapted to determining compartment pressures during exercise.

Normally, peripheral tissue fluid pressure is negative in accordance with lymphatic pump "suction" action as described by Guyton and Barber (8). It is therefore noteworthy that in control legs we corroborate a slightly negative pressure in the subcutaneous tissue (9), while in lymphedematous legs the pressure is positive consistent with tissue edema from obstructed or impaired lymphatic fluid transport.

Intramuscular pressure was also elevated in the anterior tibial compartment of the lymphedematous leg, which also was likely due to an increased tissue fluid volume (10). These findings corresponded to those previously reported by Qvarfordt et al (2) who suggested impaired venous function in patients with secondary lymphedema. Presumably venous emptying was delayed by tissue swelling and partial compression from increased intramuscular pressure (2). In our patients with primary lymphedema, however, venography (4 patients) showed chiefly dilated rather then occluded or compressed veins, and Doppler studies and photoplethysmography also favored incompetent but not occluded veins.

Taken together the findings suggest not only superficial and deep lymphatic system involvement in primary lymphedema, but also concomitant venous dysfunction. With both venous and lymphatic blockade (anatomical and/or functional) peripheral veno-lymphatic communications in extremity lymphedema is also plausible (4). These observations should be taken into account and perhaps intramuscular pressure measured in patients with lymphedema undergoing surgical intervention. Thus, with increased intramuscular pressure, operations designed to drain lymphedema fluid from the superficial into the muscular compartment (e.g. Thompson's operation) may be inappropriate because a) deep lymphatics may also be impaired; b) peripheral venous drainage may also be sluggish or incompetent and c) high intramuscular

pressure favors collapse of lymphatics at relatively low tissue pressure elevations (~ 2-12 mm Hg) (11).

REFERENCES

1. Kinmonth, JB: The lymphatics. Surgery, lymphangiography and diseases of the chyle and lymphatic system. Arnold Ltd, London, 1982.
2. Qvarfordt, P, JT Christenson, B Eklof, PE Jonsson, P Ohlin: Intramuscular pressure, venous function and muscle blood flow in patients with lymphedema of the leg. Lymphology 16 (1983), 139.
3. Pflug, JJ, JW Calnan: The relationship between the blood, lymphatic and interstitial circulation in the leg. VASA 2 (1973), 75.
4. Sobhi Zaki, M, OM Askar, TA Ata: A plan of management for lymphedema of the lower limb. Egypt. J. Plastic Reconstr. Surg. 6 (1982) 97.
5. Seki, K: Lymph flow in human leg. Lymphology 12 (1979), 2.
6. Rorabeck, CH, D Castle, R Hardie, J Logan: The slit-catheter a new technique for measuring intracompartmental pressure. Surg. Forum 31 (1980), 513.
7. Scolander, PF, AR Hargens, SL Miller: Negative pressure in the interstitial fluid of animals. Science 161 (1968), 321.
8. Guyton, AC, BJ Barber: The energetics of lymph formation. Lymphology 13 (1980), 173.
9. Stranden, E, HO Myhre: Pressure-volume recordings of human subcutaneous tissue. A study in patients with edema following arterial reconstruction for lower limb atherosclerosis. Microvasc. Res. 24 (1982), 241.
10. Mubarak, SJ, AR Hargens: Compartment syndromes and Volkmann's contracture. Saunders, Philadelphia, 1981.
11. Hargens, AR, WH Akeson, SJ Mubarak, CA Owen, KL Evans, LP Garetto, MR Gopnsalves, DA Schmidt: Fluid balance within the canine anterolateral compartment and its relationship to compartment syndromes. J. Bone Joint. Surg. 60-A (1978), 499.

ACKNOWLEDGEMENTS

The study was supported by Grant MS 006, Kuwait University. The authors thank Paul Phillips for excellent technical assistance.

Key words: Lymphedema, subcutaneous tissue pressure, intramuscular pressure, venous function

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