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# Severe Pretibial Myxedema

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## Abstract

Although pretibial myxedema (PTM) occurs in 4.3% of patients with Graves's disease, the most severe variant, elephantiasis nostras verrucosa, is found in less than 1% of cases. The most frequent location of infiltration is the lower extremities, especially the pretibial areas and on the dorsum of the foot. The authors report one of the most severe cases of elephantiasis nostras verrucosa, following radioactive iodine therapy.

## Keywords

thyroid, pretibial myxedema, Graves's disease

## Case Presentation

A 51-year-old female initially presented with Graves's disease 6 years ago. At that time she had exophthalmos but no pretibial myxedema (PTM) of the legs or dorsum of the feet. She was then treated with radioactive iodine on 2 occasions. However, over the following 6 years, PTM developed in both legs, getting progressively worse. Though bilateral, the hyperkeratotic, myxedematous lesions on her left leg spread circumferentially to involve most of the skin up to mid calf. Additionally, there was distal involvement of the left foot with deep folds in the thickened skin, causing the appearance of the foot to resemble that of elephantiasis (Figures 1 and 2). Moreover, the skin and subcutaneous tissue were so hard and unyielding that dystrophic calcification was suspected. Plain x-ray confirmed that there was no calcification and that the disease was confined to the skin and subcutaneous tissue (Figure 3). The marked deformity and the hard, unyielding tissue do not permit the use of normal footwear. The striking abnormality also makes it cosmetically unacceptable.

## Discussion

Although pretibial myxedema (PTM) occurs in 4.3% of patients with Graves's disease,<sup>1</sup> the most severe variant, elephantiasis nostras verrucosa (ENV), is found in less than 1% of cases.<sup>2</sup> The most frequent location of infiltration is the lower extremities, especially the pretibial areas and on the dorsum of the foot.<sup>3</sup> Pretibial myxedema is a misnomer, as the word *myxedema* describes the features of hypothyroidism and edema clinically implies excess fluid in the interstitial spaces. However, PTM is caused by mucinous infiltration of the deeper parts of the dermis, followed by a reactive fibroblastic proliferation. The mucin separates the



**Figure 1.** Severe pretibial myxedema of the left leg and foot and early myxedematous changes of the dorsal aspect of the right foot

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**Figure 2.** Lateral view of pretibial myxedema of the left leg and foot



**Figure 3.** X-ray of the left foot showing that the myxedema is limited to soft tissue

collagen fibers of the dermis and makes the skin very thick, as was noted in this patient.

Kriss et al<sup>4</sup> postulated that the destruction of the thyroid gland by disease (hyperthyroidism or thyroiditis) or by therapeutic agents (especially iodine-131) results in the release of an antigen of thyroid cell origin that then stimulates the production of the antibody, LATS (long acting thyroid stimulator). The antibody is then fixed to the tissues of the legs. The resulting immunological reaction then causes a local inflammatory tissue reaction that manifests as PTM. Incidentally, though this patient had radioactive iodine twice, her leg swelling continued to worsen over 6 years in spite of her euthyroid state.

In a Mayo clinic review of 150 patients with PTM, 58% had nonpitting edema (brawny), 20% nodular variant, 21% plaque type, and less than 1% had either polypoid or elephantiasic morphologies.<sup>2</sup> Treatment for ENV is generally unsuccessful. However, Iwao et al<sup>5</sup> used a motor-powered grinder to abrade the affected area after shaving off the verrucous lesions with a freehand knife; re-epithelialization occurred in 2 weeks. When ENV is very severe as in our patient, there is pressing need for surgical correction, but this is likely to offer only limited success.

#### Declaration of Conflicting Interests

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