Obesity-induced Lymphedema
Nonreversible following Massive Weight Loss

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Obesity-induced Lymphedema Nonreversible following Massive Weight Loss

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Summary: Lymphedema is the progressive enlargement of tissue due to inadequate lymphatic function. Obesity-induced lymphedema of the lower extremities can occur once a patient’s body mass index (BMI) exceeds 50. We report our first patient with obesity-induced lower extremity lymphedema who was followed prospectively before and after weight loss. A 46-year-old woman with a BMI of 80 presented to our Lymphedema Program complaining of bilateral lower extremity swelling. Lymphoscintigraphy showed impaired lymphatic drainage of both lower extremities consistent with lymphedema. She was referred to a bariatric surgical weight-loss center and underwent a sleeve gastrectomy. After reaching her new steady-state BMI of 36 eighteen months following her procedure, lymphoscintigraphy showed no improvement in lower extremity lymphatic function. Patients at risk for obesity-induced lymphedema should be counseled that they should seek weight-loss interventions before their BMI reaches 50, a threshold at which point lower extremity lymphedema may occur. Unlike other comorbidities that reverse following massive weight loss, obesity-induced lymphedema may not resolve.

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CASE REPORT

A 46-year-old woman presented to our Lymphedema Program complaining of bilateral lower extremity swelling. Her height and weight were 153 cm and 187 kg (BMI = 80). The patient reported that at age 34 she weighed 68 kg (BMI = 29), her legs were asymptomatic, and she subsequently began to steadily gain weight. Other than her obesity, she had no risk factors for lymphedema (eg, family history of lymphedema, primary lymphedema, travel to areas endemic for filariasis, and inguinal radiation/lymphadenectomy/operations). The patient’s lower extremity lymphatic function was evaluated by lymphoscintigraphy, which is 100% specific and 92% sensitive for lymphedema.³ Lymphoscintigraphy at her initial presentation showed impaired lymphatic drainage of both lower extremities consistent with lymphedema (Fig. 1). She was referred to a bariatric surgical weight-loss center.

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and underwent a sleeve gastrectomy. After reaching her new steady-state BMI of 36 eighteen months following her procedure, lymphoscintigraphy showed no improvement in lower extremity lymphatic function.

**DISCUSSION**

Lymphedema is a disease caused by the anomalous development of the lymphatic system or injury to lymphatic vasculature. The condition involves the extremities, although the genitalia can be affected as well. Lymphedema is progressive, and the area enlarges over time. Patients suffer from psychosocial morbidity, infection, difficulty using the extremity, and rarely malignant degeneration. This report documents the first patient with obesity-induced lower extremity lymphedema who had serial evaluation

![BMI 71](image)

![BMI 36](image)

**Fig. 1.** Obesity-induced lymphedema not improved following massive weight loss. A, Forty-six-year-old woman with a BMI of 71 before undergoing a weight-loss operation. Lymphoscintigram shows delayed transit of Tc-99m filtered sulfur colloid to the inguinal nodes 45 minutes following intradermal injection into the feet consistent with lymphatic dysfunction and lymphedema (*). Lymph nodes are visualized 2 hours following administration of radiolabeled tracer (^). B, Eighteen months following sleeve gastrectomy the patient’s BMI decreased to 36. Lymphoscintigram image of inguinal nodes at 45 minutes (*) and 120 minutes (^). Despite significant reduction in the size of her lower extremities, she continues to have delayed transit of radiolabeled tracer to her inguinal nodes and no improvement in her lymphatic function.
The finding that lymphedema did not improve in this individual despite massive weight loss has broad implications. Obesity, which affects one-third of the US population, is an increasing public healthcare problem. Patients at risk for obesity-induced lymphedema should be counseled that they need to seek weight-loss interventions before their BMI reaches 50, a threshold where irreversible lower extremity lymphedema may occur. Unlike other comorbidities that reverse following massive weight loss (eg, diabetes, hyperlipidemia, hypertension, and sleep apnea), obesity-induced lymphedema may not resolve. As a result, after losing weight, obese patients may continue to suffer from a progressive, incurable condition over the course of their lives.

The mechanism by which obesity causes lymphatic dysfunction is unclear. If obesity collapsed lymphatics or increased lymph production overloaded transport capacity, then lymphatic flow would be expected to improve following weight loss. Because lymphatic dysfunction remains despite massive weight loss, obesity likely damages lymphatic structures through inflammation and/or other mechanisms. Further study is necessary to determine if variables such as severity of obesity, duration of lymphatic dysfunction, and comorbidities affect the reversibility of obesity-induced lymphedema.

CONCLUSIONS

Obesity-induced lymphedema may develop once a patient’s BMI exceeds 50. We report our first patient with obesity-induced lower extremity lymphedema who was followed prospectively before and after weight loss. Despite lowering her BMI from 80 to 36, her lymphatic function did not improve. Consequently, obesity-induced lymphedema may cause irreversible lymphatic dysfunction. Individuals at risk for the condition should be referred to a bariatric surgical weight loss center.

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REFERENCES