The Development of a Charcot Foot after Significant Weight Loss in People with Diabetes

Three Cautionary Tales

Rachel Murchison, DipPod*
Catherine Gooday, BSc, PGDip*
Ketan Dhatariya, MBBS, MSc, MD, MS, FRCP

Medication to aid weight loss and weight loss surgery are becoming more commonly available for people with diabetes. As a result of profound weight loss, diabetes may go into remission and many biochemical and physical parameters improve. However, some of the end organ damage associated with diabetes may not improve, peripheral neuropathy being an example. We present three cases in people with diabetes and pre-existing peripheral neuropathy who had lost significant weight. They became more mobile and developed a Charcot foot despite their diabetes improving significantly. People who have lost significant weight should continue to monitor their feet because the risks of foot disease remain even if diabetes goes into remission. (J Am Podiatr Med Assoc 104(5): 522-525, 2014)

Weight gain is intimately related to the likelihood of developing prediabetes and type 2 diabetes,[1] and subsequent weight loss contributes to diabetes remission.[2] As the prevalence of obesity increases, health-care providers are seeking strategies to either treat those who are already obese, or come up with successful strategies to prevent weight gain. Bariatric surgery has been widely advocated as part of the strategy for treating the increasing burden of obesity and type 2 diabetes, and may be used earlier in the natural history of the disease over the next few years.[3]

The combination of peripheral vascular disease and distal neuropathy predisposes one to the development of foot ulcers.[4] In addition, a combination of shear stress and pressure as a result of trauma or changes in the biomechanics of the foot can result in the development of foot ulcers.[5] Pressure—the force applied to a unit area of surface—is therefore increased in people who are overweight or obese, which increases the risk of developing Charcot neuroarthropathy in neuropathic patients.[6] Because obesity limits one's physical activity, obese individuals may not frequently experience the high foot pressures that occur while walking. When obese individuals do walk, they may experience very high pressures on the feet, thereby increasing the risk of ulceration.[6]

Obese individuals may be unable to perform self care of the feet due to their inability to reach their feet, further increasing their potential risk of developing foot problems. Previous work has shown a relationship between a high body mass index and diabetes-associated foot pathology.[7] Furthermore, there is evidence to show that weight loss increases strength, balance, gait, and gait speed, as well as changes the pressures within the foot.[8] It is not yet known whether existing foot disease improves with substantial weight loss. We present three cases in which foot pathology deteriorated despite significant weight loss and improved glycemic control after bariatric surgery.

Case 1

A 49-year-old woman with a 12-year history of gestational diabetes followed by type 2 diabetes was referred for weight loss surgery. Over the subsequent 5 months, she lost 40 kg and her HbA_1c dropped from 7.6% (60 mmol/mol) preoperatively, to 6.4% (46 mmol/mol) postoperatively. As a result, she was able to stop her glucose-lowering medication.

*Diabetic Foot Clinic, Elsie Bertram Diabetes Centre, Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, Norfolk, England.

Corresponding author: Ketan Dhatariya, MBBS, MSc, MD, MS, FRCP, Elsie Bertram Diabetes Centre, Norfolk and Norwich University Hospitals NHS Foundation Trust, Colney Lane, Norwich, Norfolk, NR4 7UY, England. (E-mail: ketan.dhatariya@nnuh.nhs.uk)

Prior to bariatric surgery, she had undergone a surgical debridement and excision of a pyogenic granuloma on the left foot. Six months after her weight-loss surgery, she developed osteomyelitis of her left second toe that necessitated a ray amputation. Nine months after her weight-loss surgery, she developed an ulceration on her left first toe and underwent a local amputation 11 months after her initial weight-loss surgery due to osteomyelitis. Three months after this amputation (ie, 14 months after her bariatric surgery), she was readmitted with a swollen left foot with painless fracture of her third metatarsal and wound on her fourth toe. A Charcot foot was confirmed radiographically on magnetic resonance imaging. She elected to have a below-the-knee amputation on the left lower extremity.

Case 2

A 40-year-old male with a 10-year history of type 2 diabetes was referred for weight-loss surgery. As a result, he lost 45 kg over the next 13 months. Over the 20 months following his surgery, his HbA_1c had fallen from 9.6% (81 mmol/mol) to 6.6% (49 mmol/mol). As a result, he was able to discontinue his basal bolus insulin regimen, but he remained on metformin. Fourteen months after his bariatric surgery, he underwent surgical debridement of a right foot ulcer. One month later, a right Charcot joint was suspected, and after a magnetic resonance image of his foot, he went into a total contact cast. However, adherence with the cast was poor and he opted to use a removable walker instead. He also underwent an apronectomy during this time. His activity increased and 20 months after his initial bariatric surgery, he developed a plantar wound under his third metatarsophalangeal joint. He refused to consider another total contact cast and traveled abroad with this wound. Unsurprisingly, his foot deteriorated, and he eventually elected to have a below-the-knee amputation despite the advice from our specialist multidisciplinary foot clinic suggesting that his foot was salvageable with optimal offloading. Despite our protestations, he felt that amputation was the best solution.

Case 3

A 64-year-old man with an 18-year history of type 2 diabetes and evidence of significant peripheral neuropathy lost 18 kg over a 1-year period on a diet under supervision from a specialist weight loss clinic. His HbA_1c had remained effectively unchanged over the preceding 4 years, between 6.9% and 7.3% (52 and 55 mmol/mol). His weight loss was achieved using a milk-based diet, and a glucagon-like peptide 1 receptor agonist. He became progressively more mobile during this time. He had been effectively housebound prior to starting the diet, and with his weight loss, he was able to enjoy walking around his local supermarket and beyond. He presented to our foot clinic with a warm, swollen right foot with a $>2°C$ difference between his two feet. There was no evidence of an ulcer or infection and a diagnosis of a Charcot foot was confirmed radiographically. He was treated with a below-knee, full-contact plaster cast.

Discussion

Weight gain changes the pressure distribution within the legs and feet, and alters the biomechanics of gait, balance, and posture.[9] In diabetic individuals, the peripheral neuropathy leads to additional concerns because of the reduced proprioception and intrinsic muscle wasting, leading to abnormal pressure distributions within the foot.[10] These changes increase the risks of developing foot problems. However, with increasing weight comes decreased mobility, and the effects of the diabetes-related microvascular disease are likely to be offset as mobility decreases.

Weight loss is one of the mainstays of treatment advocated in people who have type 2 diabetes.[11] As with the patients we described, glycemic control often improves and for many goes into remission.[2] Other biochemical parameters of cardiovascular risk also improve with weight loss, thus reducing the overall risk of macrovascular disease.[2] Diabetic neuropathy is essentially an irreversible process. Although bariatric surgery may stop the progression of diabetic neuropathy, it has not been shown to substantially improve the condition. Thus patients with pre-existing peripheral neuropathy need to remain vigilant when caring for their feet, even if their diabetes goes into remission. This risk is increased because as the weight reduces, activity levels are likely to rise. This increased activity level increases the stress and pressure on the neuropathic feet and may result in a higher risk of developing foot pathology, although these data are not consistent.[12] In addition, the relative immobility of the patient may have led to a degree of osteopenia, raising the risk of low-impact fractures, and the subsequent complications, as described in Case 1.

Further issues arise due to the change in bone

mineral density that occurs at sites of load bearing following weight-loss surgery. Although this may reflect wider biochemical changes, the bone density does diminish, leading to an increased risk of fractures.[13,14]

There is debate in the diabetes community about how to label people whose biochemistry normalizes after weight-loss surgery, with no evidence of dysglycemia. In many people whose glycemic indices may normalize over time, dysglycemia and diabetes reappear in a proportion of people.[15] This suggests that even if glycemic parameters normalize, people with a diagnosis of diabetes should never be lost to follow-up and should undergo regular review for the development of complications. There is currently no READ code (the standard clinical terminology system used for coding a diagnosis) for "diabetes in remission." The coding classification used to include a code for "diabetes in remission" needs to change given the increasing numbers of people having weight-loss surgery and the increasing possibility of missing serious consequences. In particular, research is needed that examines activity levels before and after bariatric surgery, as well as changes in plantar pressures. Although there are data to show that greater activity is related to an increased risk of developing foot ulcers,[16] it is the risk of loss to follow-up created by the misplaced focus on blood glucose normalization that needs to be addressed.

The fact that the risk of some microvascular complications does not diminish despite glycemic normalization may be a manifestation of 'metabolic memory.' There is some recent evidence to show that the risk of developing detrimental effects of chronic hyperglycemia—in particular microvascular complications—remain despite achievement of good glycemic control.[17]

All three of the patients described herein had lost more than 30% of their initial excess body weight but still developed significant foot problems. We suggest that the weight loss allowed them to have greater mobility. Patients may not be aware that the benefits of weight loss do not extend to improvement in neuropathy. Thus continued vigilance and ongoing specialist foot care are essential. A collaboration between centers looking at prospective outcomes would help in particular correlating activity levels, and the risk of developing ulcers or neuroarthropathy.

In conclusion, we suggest that a combination of bone mineral loss and the increased mobility our patients enjoyed due to significant weight loss led to their new foot pathologies and ultimately contributed to their amputations. We use these cautionary tales to highlight this issue that we believe is likely to become more common as bariatric surgery becomes increasingly available.

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