A 38 year old female owner of a fashion shop presented at the rheumatology outpatient clinic because of intense pain and nodular lesions in her hands (fig 1A). She had a 1 year history of pain and multiple small intradermal deposits, with drainage of white chalky material. She had been initially considered to have an infection owing to the presence of a pyogenic pustule. Because of this, she had been treated with several courses of antibiotics (oral cloxacillin for 2 weeks and then oral amoxicillin plus clavulanic acid for another 2 weeks) draining a thick material, but not frank pus. At the outpatient clinic a needle aspiration obtained a thick white matter. Cultures were sterile and the material contained numerous monosodium urate crystals. She was diagnosed as having intradermal tophaceous gout.

Tophaceous gout is an unusual feature in a young woman. In these cases a careful search for secondary conditions needs to be carried out. Our patient had a 3 year history of intense fear of gaining weight or becoming fat, subjective disturbance in her body shape “feeling fat”, and 1 year before diagnosis she had amenorrhoea and constipation. She also admitted excessive ingestion of laxatives and diuretic abuse. At diagnosis her body fat was undetectable, and the bones protruded through the skin. Her height was 163 cm and her weight 55 kg (body mass index was 20.7). Our patient had a medical history consistent with anorexia nervosa.

Laboratory determinations disclosed anaemia (haemoglobin 99 mg/l), leucocytosis (17×10⁹/l), and 402×10⁹ platelets/l. A routine blood biochemistry profile yielded glucose 5.9 mmol/l, creatinine 90 μmol/l, urea nitrogen 16.0 mmol/l, sodium 138 mmol/l, potassium 2.2 mmol/l, and uric acid 456 μmol/l. The 24 hour urate clearance was clearly low (3.2 ml/min) and the 24 hour uric acid excretion was 1.6 mmol/day. Thyroid stimulating hormone and thyroxine (T4) levels were normal. Follicle stimulating hormone (14.8 U/l), luteinising hormone (8.2 U/l), and 17 β-estradiol (150 pmol/l) were within normal ranges. Purine enzyme studies, including hypoxanthine guanine phosphoribosyl transferase, adenine phosphoribosyl transferase, and phosphoribosyl pyrophosphate synthetase, were normal. Evaluation showed glucose-6-phosphatase deficiency was also normal. There was no history of exposure to lead. Treatment with allopurinol was started and 9 months later total resolution of the intradermal tophi was achieved (fig 1B).

A number of unusual cutaneous manifestations of chronic gout have been reported. As observed in our patient, deposition of urate crystals in the skin can take the form of intradermal, superficial collections of white tophaceous material resembling pus. Aspiration of the nodular collection demonstrating monosodium urate is the preferred technique to achieve a precise diagnosis.

In cases such as our patient, once a diagnosis of chronic tophaceous gout is made, a secondary condition must be excluded. Premenopausal gout is commonly associated with obesity, renal insufficiency, and hypertension. However, Hayem et al report three cases of tophaceous gout affecting premenopausal women and the only precipitating factor was
the overuse of furosemide. In their cases, the authors highlighted the importance of a careful search for hidden diuretic abuse.⁴ Uric acid is primarily excreted by the kidney, and certain drugs including diuretic agents can reduce the excretion of uric acid. No specific tests exist for a diagnosis of anorexia nervosa. Cultural issues, occupation, and body image are important factors in this condition.⁵ In our case a dysmorphophobia led to inadequate nutrition and an excessive consumption of diuretics. With this case we would like to emphasise that in Western countries, where culture and body image play an important role in eating disorders,⁶ the diagnosis of tophaceous gout in a young woman should require a psychological evaluation to exclude a medical history consistent with anorexia nervosa.

A colour version of fig 1 can be found at http://www.annrheumdis.com/supplemental

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**Treatment of chronic plantar fasciitis with botulinum toxin A: an open case series with a 1 year follow up**

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**Plantar fasciitis**, a special type of a soft tissue rheumatic syndrome, is a common painful condition which often becomes chronic. Its aetiopathology is not completely understood.¹ Being overweight and standing work are regarded as predisposing factors.² Microtrauma, nerve entrapment (Baxter’s nerve), or limited ankle dorsiflexion are thought to be responsible.³–⁴

Most patients can be treated with physical therapy, local glucocorticoid injections, insoles, acupunture, and extra-corporeal shock wave therapy.⁵–⁶ However, this treatment cannot improve the pain in all cases and can require a lot of time on the part of both patient and therapist. Surgery is controversial and should be restricted to patients who do not respond to conservative treatment.

It is not yet clear whether treatment of chronic plantar fasciitis with botulinum toxin A (Btx-A) works by causing muscle paresis or by analgesic/anti-inflammatory effects, or both. A combined effect may occur—namely, (a) induction of paresis of the muscles originating at the medial calcaneal process and (b) occurrence of direct analgesia owing to its anti-noiceptive and anti-inflammatory properties.⁷–⁸

In this pilot study here the effect of a single injection of Btx-A was studied in an open case series.

Nine patients with chronic plantar fasciitis (five women, four men, mean (SD) age 55 (9.5) years) were treated with Btx-A. The average disease period was 14 months (range 2–36). Patients were selected according to the inclusion and exclusion criteria of an already planned multicentre study (table 1).

Btx-A (500 units; Dysport, IPSEN Pharma, Ettlingen, Germany) was dissolved in 5.0 ml injection solution (0.9% saline), and 2.0 ml (200 units) were injected subfasically in four different directions through one injection

*Pain progression stage using Gerbershagen’s score (Mainz Pain Staging System) at first visit (injection) and final examination (week 52).*

- Greatest pain during the past 48 hours using a visual analogue scale (VAS), 0–10
- Pain at rest during the past 48 hours using a VAS, 0–10
- Measurement of muscle force using Brunner’s method, scale 0–5, for
  - Extension and flexion of the great toe
  - Extension and flexion of the foot
  - Pronation and supination of the foot
- Pain progression stage using Gerbershagen’s score (Mainz Pain Staging System) at first visit (injection) and final examination (week 52).

Statistical analysis was performed using Wilcoxon’s test. Two weeks after injection a pronounced and statistically significant reduction of pain at rest during the past 48 hours was observed using a VAS (fig 1). The maximum pain during the past 48 hours was reduced to the same extent (p=0.015).

The recorded Mainz Pain Staging System (1–3) at injection and at week 52 showed a decrease from an average stage of 1.56 to 1.00. It is certainly not a specific tool for staging chronicity in plantar fasciitis, but we aimed at making a general statement on the course of the disease. Undesirable effects such as muscle weakness or systemic reactions were not seen. Our patients were satisfied not only by the pain

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Accepted 3 April 2005

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**REFERENCES**