LETTERS

Radiological changes of short and flat bones in primary hypertrophic osteoarthropathy

Hypertrophic osteoarthropathy (HO) is defined as a syndrome characterised by finger clubbing and periostosis of long bones.¹ It is classified into primary (PHO) and secondary hypertrophic osteoarthropathy (SHO).²

To establish the HO diagnosis, finger clubbing and periostosis of long bones must be present.²

Cases of PHO have not been frequently reported in the medical literature.³ HO cases have recently been more described, since HO was classified into the bone and cartilage group of diseases according to the international classification of rheumatic diseases.⁴

We formed a large group of patients in whom changes of flat bones were found. The analysis was based on a group of patients with PHO from 1970 to 1996. Over 26 years, a total of 76 patients was enrolled in the study. There were five female (6.5%) and 71 male (93.5%) patients. Their age ranged from 18 to 64 years (mean age 43).

Figure 1 Periostosis of calvaria.

Figure 2 Periostosis of calvaria (laterolateral view).

Figure 3 Periostosis in ilium to the left, foramen opturatum contralaterally, tuber ossis ischii and small trochanter.

Figure 4 Periostosis visible in the foramen opturatum to the right, and more pronounced osteoporosis is evident parallel with acetabulum and femur head.

Radiological examination of short and flat bones (pelvis, vertebral column, ribs, sternum, clavicle, skull, hand, and foot) was performed in all the patients. A more or less pronounced periostosis of long bones was found in all the patients.

Periostitis was mainly evident in diaphysis, metaphysis and epiphysis with the irregular configuration of bones and extremities.

The localisation of periostosis was in calvaria, where the periosteal reaction was present across the whole surface (figs 1 and 2).

It was evident in the clavicular ribs (pelvis, os ilium, os ischii, foramen opturatum, pubic bone) (figs 3 and 4), radially in os navicularis carpi (fig 5), patella (fig 6), carpal bones (fig 6), and metatarsal bones (fig 7).

In the terminal phalanges of fingers and toes, a clear acroosteolysis was found in 52 patients (78.4%) (fig 8).

Figure 5 Periostal layers in the distal part of radius, ulna, and metacarpal bones, and os naviculare.

Figure 6 Periostosis of tibia, fibula and patella (more pronounced in the left leg).

Figure 7 Periostal layers in the metacarpal bones.

Figure 8 Distal phalanges are rumpled. Periostosis is developed across the whole surface, and it is most pronounced in the basis of the distal phalange that tends to be connected to the one on the top. Acroosteolysis in the distal part of I, II, and IV phalange of the left foot and first four toes of the right foot.

PHO is defined as a syndrome characterised by finger clubbing and periostosis of long bones.¹ Data and this definition indicate that periosteal reaction manifests very rarely in short and flat bones.^{1-3,5} In our study, the skeleton of 76 patients was radiologically examined and periosteal reaction in short and flat bones was found in 14 patients (18.5%). It was developed in many flat bones (calvaria, clavicle, scapula, pelvis - os ilium, os ischii, pubic bone, patella, small bones of the hand, foot and vertebral column skeleton).

Nevertheless, periosteal reaction is not so pronounced in short and flat bones, as it is in long bones, and thus it should be "searched for". It has been reported by only a few authors,³ while many others have not recorded it.⁶

Acroosteolysis is almost a regular phenomenon in terminal phalanges of the fingers and thumbs of hands and feet in cases when PHO was present for more than 10 years. Some authors have already described this, but in individual patients.⁷

In conclusion, it should be pointed out that periosteal reaction can develop not only in long bones, but also in short and flat bones more often than it has been presumed.

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1 Martinez-Lavin M, Matucci-Cerinic M, Jajić I, Pinedo C. Hypertrophic osteoarthropathy: consensus on its definition, classification, assessment and diagnostic criteria. J Rheumatol 1993;20:1386-7.

2 Matucci-Cerinic M, Lotti T, Jajić I, et al. The clinical spectrum of pachydermoperiostosis (primary hypertrophic osteoarthropathy). Medicine 1991;70:308-14.

- 3 Rimion DZ. Pachydermoperiostosis (idiopathic clubbing) and periostosis. Genetic and physiologic consideration. *N Engl J Med* 1965;272:923-31.
- 4 Schumacher HR. Classification of rheumatic diseases. In: Klippel JH, Dieppe PA. *Rheumatology*. St Louis: Mosby, 1994:1.7.1-4.
- 5 Jajić I, Jajić Z. *Syndrome of hypertrophic osteoarthropathy*. Zagreb: Medicinska knjiga, 1997:1-170.
- 6 Fisher DS, Singer DH, Feldman SM. Clubbing, a review emphasis on hereditary acropachy. *Medicine* 1964;63:1974.
- 7 Schwartzman S, Spiera H. Idiopathic hypertrophic osteoarthropathy without pachiderma and with acrolysis. *Mount Sinai J Med* 1982;49:335-7.

Chondrocalcinosis and Gitelman's syndrome. A new association?

Calcium pyrophosphate dihydrate (CPPD) deposition disease is in most cases idiopathic, but there are familial forms and others in connection with metabolic disease.¹ One of the metabolic associations is with hypomagnesaemia, frequently because of Bartter's syndrome (BS).² Another cause of hypomagnesaemia is Gitelman's syndrome (GS),³ a hypocalciuric variant of BS. Recent studies confirm that these are different processes, with diverse molecular bases that explain the clinical and analytical findings characteristic of each one of them.⁴ We present the case of a patient with chondrocalcinosis (CC) and pseudogout in whose study hypomagnesaemia secondary to GS is detected. In our search of the Medline database, we have not found reports of a similar association. However, reading of some of the CC and BS cases leads us to think that we are actually dealing with patients whose GS had not been diagnosed.⁵⁻⁷

The patient is a 43 year old woman without pathological antecedents who consulted because of a four year history of bilateral mechanic knee pain with episodes of acute arthritis that remitted days after treatment with non-steroidal anti-inflammatory drugs. During four years of follow up she presented with three acute episodes of arthritis affecting the right knee, the left knee, and the right ankle respectively. On one occasion, knee arthrocentesis was carried out and intracellular CPPD crystals were observed in the synovial fluid. Radiographs showed chondrocalcinosis in the knees (fig 1) and pubic symphysis. Table 1 shows the most significant laboratory

parameters. The complete blood cell count, hepatic function, serum iron, ferritin, total iron binding capacity, thyroid and parathyroid hormones, and urine analysis were normal.

The patient was normotensive, she denied intake of alcohol, diuretics or any other drugs, she did not have an inadequate diet, and she did not present with any history of vomiting, diarrhoea, tetany, cramps or weakness. Other causes of hypomagnesaemia were excluded and her mother, brother and two children underwent analytic studies. The 83 year old mother had mild hypomagnesaemia and metabolic alkalosis. The 55 year old brother had mild hypokalaemia with serum magnesium in the lower limit of normality. The 18 year old son had normal values and the 24 year old daughter had mild hypokalaemia with serum magnesium in the lower limit of normality. The rest of the parameters and radiographs of knees, pelvis, and wrist were normal. With these data, the patient was diagnosed GS with autosomal dominant transmission and variable phenotypic expression. Oral magnesium lactate treatment (6 mmol/day of elemental magnesium) was started with acceptable gastrointestinal tolerance and mild rise of serum magnesium (0.65 mmol/l) and potassium (3.6 mmol/l) concentrations. After magnesium treatment the values of plasmatic aldosterone and renin were normal. After the start magnesium supplementation the patient has not had any new episodes of acute arthritis.

Arthropathy because of CPPD deposition disease has been described in patients with hypomagnesaemia of renal origin^{2,8,9} or secondary to BS.^{10,11} Clinical findings in these cases include CC and recurrent episodes of pseudogout,¹ but chronic pyrophosphate arthropathy has been also reported.¹² GS is a primary renal tubular disorder and it is considered to be a hereditary variant of BS, its diagnosis being based on the following criteria: (a) hypomagnesaemia of renal origin, (b) hypokalaemia of renal origin, and (c) hypocalciuria.¹³ Other findings in these patients are normotension or hypotension, metabolic alkalosis, hyper-reninaemic hyperaldosteronism and tendency to reduced sodium and plasmatic chloride with inappropriate excretion of sodium and chloride.¹⁴

Data in favour of GS diagnosis instead of the classic BS are the age of presentation (adults), lack of frank polyuria, hypocalciuria, and hypomagnesaemia.¹⁵ Classic BS and GS

Figure 1 Plain radiograph of the knees showing images of chondrocalcinosis.

are thought to represent distinct tubular disorders: specifically a Henle loop defect in the former and a distal tubule defect in the latter.¹⁴ Two types of genetic transmission have been suggested in GS, one with autosomal recessive inheritance presenting with frequent tetanic episodes and lower plasma potassium and magnesium concentrations, and one with autosomal dominant inheritance with high phenotypic variability, without tetany and with not so low concentrations of potassium and magnesium.¹³ The treatment includes potassium and magnesium supplementation, but the use of spironolactone has also been suggested.¹⁴ In some patients with CC secondary to hypomagnesaemia, a CC decrease has been observed after continuous treatment with magnesium.⁷⁻⁹ In the present case report good results have been obtained with oral magnesium supplementation.

Since its first description by Bauer in 1979,¹⁰ 30 patients with BS and CC have been reported. On our review of the Medline literature we did not find any reference to association between CC and GS. It is probable that many of the patients reported as BS actually have GS, as authors do not usually report the data of calciuria.⁴ The patients reported with both CC and BS have hypomagnesaemia. Some of them also present with hypokalaemia, hypocalciuria, and hypermagnesiuria,⁵⁻⁷ which are the hallmarks of GS.¹³ In many other cases the values of calciuria are not reported and therefore GS cannot be excluded.

We consider that it is useful to determine plasma bicarbonate, blood and urine ions, urinary magnesium and calcium (urinary calcium/creatinine molar ratio) in all patients with CC and hypomagnesaemia to exclude GS. Also, a screening of family members may be indicated.⁹ Reports of new cases will lead to a better knowledge of the pathophysiology of both processes.

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1 Jones AC, Chuck AJ, Arie EA, Green DJ, Doherty M. Diseases associated with calcium pyrophosphate deposition disease. *Semin Arthritis Rheum* 1992;22:188-202.

Table 1 Biochemical data in patient with GS at diagnosis, and normal values

Parameters	Patient	Normal
Plasma renin activity (ng/ml/h)	3.23	0.2-2.8
Plasma aldosterone (pg/ml)	209.2	7.5-150
Serum bicarbonate (mmol/l)	33	22-28
Serum creatinine (μmol/l)	62	35-106
Serum sodium (mmol/l)	138	137-148
Serum chloride (mmol/l)	98	95-109
Serum potassium (mmol/l)	3.3	3.8-5
Serum magnesium (mmol/l)	0.54	0.74-1.07
Serum calcium (mmol/l)	2.5	2.15-2.75
Serum phosphate (mmol/l)	1.2	0.87-1.45
FENa ⁺ (%) [*]	1.49	0.5-1.45
Urine sodium (mmol/24 h)	369	<250
FECl ⁻ (%) [*]	1.67	0.7-1.7
Urine chloride (mmol/24 h)	306	110-250
FEK ⁺ (%) [*]	18.7	<12
Urine potassium (mmol/24 h)	94	25-100
FEMg ⁺⁺ (%) [*]	13.6	<4
Urine magnesium (mmol/24 h)	16.2	<1.2
Urine calcium (mmol/24 h)	0.63	2.5-7.5
Molar urinary calcium/creatinine	0.02	0.16-0.6

^{*}FE: fractional excretion. FE = urine electrolyte concentrations×plasma creatinine concentrations/plasma electrolyte concentrations×urine creatinine concentrations.

- 2 Zarco P, Jaller JJ, Rapado A, Herrero-Beaumont G, Traba ML, Grant C, *et al*. Estudios metabólicos en 2 casos de condrocalcinosis con hipomagnesemia. *Rev Clin Esp* 1989;185:77-81.
- 3 Gitelman HJ, Graham JB, Welt LG. A new familial disorder characterized by hypokalemia and hypomagnesemia. *Trans Assoc Am Phys* 1966;79:221-3.
- 4 Simon DB, Lifton RP. The molecular basis of inherited hypokalemic alkalosis. Bartter's and Gitelman syndromes. *Am J Physiol* 1996;271:F961-6.
- 5 Mayoux-Benhamou MA, Clerc D, Ganeval D, Pertuiset N, Massias P. Chondrocalcinosis articulaire et hypomagnésémie d'origine rénale. A propos de 2 cas. *Rev Rhum Mal Osteoart* 1985;52:545-8.
- 6 Dupond JL, Humbert P, De Wazières B, Wolf JP. Syndrome de Bartter, chondrocalcinosis articulaire diffuse et hypomagnésémie néphrogénique chez un adulte. *Rev Med Interne* 1989;10:261-4.
- 7 Smilde TJ, Haverman JF, Schipper P, Hermus AR, van Liebergen FJ, Jansen JL *et al*. Familial hypokalemia/hypomagnesemia and chondrocalcinosis. *J Rheumatol* 1994;21:1515-19.
- 8 Runeber L, Collan Y, Jokinen EJ, Lahdevirta J, Aro A. Hypomagnesemia due to renal disease of unknown etiology. *Am J Med* 1975;9:873-81.
- 9 Wright GD, Doherty M. Calcium pyrophosphate crystal deposition is not always "wear and tear" or aging. *Ann Rheum Dis* 1997;56:586-8.
- 10 Bauer FM, Glasson P, Vallotton MB, Courvoisier B. Syndrome de Bartter, chondrocalcinosis et hypomagnésémie. *Schweiz Med Wochenschr* 1979;109:1251-6.
- 11 Salvarini C, Rossi F, Macchioni PL, Baricchi R, Capozzoli N, Castellani S, *et al*. Bartter's syndrome and chondrocalcinosis: a possible role for hypomagnesemia in the deposition of calcium pyrophosphate dihydrate (CPPD) crystals. *Clin Exp Rheumatol* 1989;7:415-20.
- 12 Erickson AR, Yeun JY, Enzenauer GJ. Chronic calcium pyrophosphate crystal deposition disease arthropathy associated with hypomagnesemia. *J Clin Rheumatol* 1997;3:51-4.
- 13 Betinelli A, Bianchetti MG, Borella P, Volpini E, Metta MG, Basilio E, *et al*. Genetic heterogeneity in tubular hypomagnesemia-hypokalemia with hypocalciuria (Gitelman's syndrome). *Kidney Int* 1995;47:547-51.
- 14 Colussi G, Rombola G, De Ferrari ME, Malcaluso M, Minetti L. Correction of hypokalemia with antialdosterone therapy in Gitelman's syndrome. *Am J Nephrol* 1994;14:127-35.
- 15 Betinelli A, Bianchetti MG, Girardin E, Caringella A, Cecconi M, Appiani AC, *et al*. Use of calcium excretion values to distinguish two forms of primary renal tubular hypokalemic alkalosis: Bartter and Gitelman syndrome. *J Pediatr* 1992;120:38-43.

Parotid lipomatosis in HIV positive patients: a new clinical disorder associated with protease inhibitors

Protease inhibitors (PI) are a major advance in the treatment of HIV-1 infection. However, side effects of these drugs occur frequently.¹ Abnormal fat accumulation in patients infected with HIV-1 has been described after starting treatment with PI.² There might be several clinical disorders associated with fat deposition, namely increasing abdominal girth, abnormal dorsal cervical fat ("buffalo hump"), and hypertrophy of the breast.²⁻⁴ We report two cases of parotid fatty deposition associated with with retroviral treatment.

CASE 1

A 54 year old heterosexual man with asymptomatic HIV infection of seven years duration was referred with a two year history of painless growing bilateral parotid swelling. He complained of dry mouth but not dry eyes. In June 1995 he was taking zidovudine (1200 mg twice daily). From January 1996 he took zidovudine monotherapy (600 mg twice daily). Four months later he noticed bilateral parotid enlargement. After 12 months of zidovudine, he was switched to lamivudine (150

Figure 1 Adipose cells among parotid acini. No acinar atrophy is seen. Lymphocytic infiltration is scarce (haematoxylin and eosin, original magnification $\times 450$).

mg twice daily), stavudine (40 mg twice daily), and saquinavir (600 mg twice daily). The parotid swelling disfigured his face. On examination both parotid glands were enlarged measuring 6×4 cm. The glands were painless when palpated and had a soft consistency. Neither abdominal girth enlargement nor "buffalo hump" were present. His CD4 cell count was 8% (97 cells/ μ l). His HIV-1 RNA was 14 000 copies/ml (Amplicor, Roche Molecular System, Madrid, Spain). Glucose, cholesterol, and triglyceride were in the normal range. ANA were negative. Schirmer test was less than 5 mm in both eyes. A parotid biopsy was performed and yielded a parotid lipomatosis with no acinar atrophy (fig 1). Currently after four months of having stopped PI both parotid glands are normal.

CASE 2

A 38 year old male drug abuser with asymptomatic HIV infection of seven years duration complained of bilateral and painless parotid swelling and breast hypertrophy of six months duration. He denied alcohol intake or sicca symptoms. In 1992 he was taking zidovudine (1200 mg twice daily) and didanosin. In November 1996 he was switched to saquinavir (600 mg thrice daily), stavudine (40 mg twice daily), and lamivudine (150 mg twice daily). In July 1997 he complained of bilateral and painless parotid and breast swelling. In November 1997 zidovudine (600 mg twice daily) was added. The patient noticed that despite continuing with the same therapeutic approach the parotid and breast enlargement became stable. On examination both parotid glands and breast were enlarged and painless. There was no evidence of increased abdominal girth or buffalo hump. Schirmer test was more than 20 mm. At that time his CD4+ cell count was 22% (961 cells/ μ l) and his HIV-1 RNA viral load 11 000 copies/ml (Amplicor, Roche Molecular System, Madrid, Spain). Glucose, triglycerides, and cholesterol were normal. ANA were negative. The patient agreed to continue the treatment but refused a parotid gland biopsy.

To our knowledge parotid fatty infiltration has not been described as a side effect of PI.¹ Fat deposition might not be limited to the

cervical-dorsal region and abdominal girth.²⁻⁴ Parotid fatty infiltration might present in isolation mimicking the diffuse infiltrative lymphocytosis syndrome or even a lymphoma as in case 1.⁵ The simultaneous development of breast hypertrophy and parotid swelling (case 2) helped us to make the diagnosis, however as a parotid biopsy was not performed the association is not proved. As it is not clear what is the best approach for treating the fat deposition syndrome we would like to highlight the rapid reversal observed in case one after stopping the PI. The pathogenesis remains obscure. However, one current hypothesis suggests that HIV-1 protease inhibitor induces peripheral lipodystrophy and it is caused by the inhibition of two proteins that regulate lipid metabolism. This results in reduced differentiation and an increase in apoptosis of peripheral adipocytes with impaired fat storage and lipid release.⁶

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- 1 Flexner CH. HIV-protease inhibitors. *N Engl J Med* 1998;338:1281-92.
- 2 Lipsky JJ. Abnormal fat accumulation in patients with HIV infection. *Lancet* 1998;351:847-8.
- 3 Miller KD, Jones E, Yanovsky JA, Shankar R, Feuerstein I, Falloon J. Visceral abdominal-fat accumulation associated with use of indinavir. *Lancet* 1998;351:871-5.
- 4 Lo JC, Mulligan K, Tai VW, Algren H, Schambelan M. "Buffalo hump" in men with HIV-1 infection. *Lancet* 1998;351:867-70.
- 5 Schrot RJ, Adelman HM, Linden CN, Wallach PM. Cystic parotid gland enlargement in HIV disease. The diffuse infiltrative lymphocytosis syndrome. *JAMA* 1997;278:166-7.
- 6 Carr A, Samaras K, Chisholm DJ, Cooper DA. Pathogenesis of HIV-1-protease inhibitor-associated peripheral lipodystrophy, hyperlipidaemia, and insulin resistance. *Lancet* 1998;351:1881-3.

MATTERS ARISING

Ultrasound guided injection of plantar fasciitis

Kane and colleagues rightly emphasise the increasing importance of diagnostic ultrasound in rheumatological practice.¹ New equipment with small transducers and greatly magnified images, albeit more expensive, make the case for its routine use in clinics stronger all the time. As Kane points out, plantar fasciitis is an excellent example of this, both for diagnosis and ensuring correct placement of injection therapy. We entirely agree that it is altogether better than using bone scintigraphy in this condition.

As Kane *et al* suggest, plantar fasciitis can be difficult to treat. They claim that clinical results are improved by using an ultrasound guided injection. Even accounting for the sample size, their results are difficult to interpret. For the majority of patients appropriate advice on footwear, weight reduction, stretching exercises, non-steroidal anti-inflammatory drugs, and insoles are successful. In addition night splints may offer 59 to 88% success without injection.^{2,3} It is not clear whether all or some of these were used before patients were termed "recalcitrant". Were they used at or after ultrasound guided injection? Finally the dose of triamcinolone used when guided by ultrasound was twice that used before and in twice the volume of lignocaine. These factors make it difficult to interpret the "improved response" suggested by this open study.

As the authors conclude, a properly controlled randomised study is needed to clarify the patients that require injecting and when. It should be remembered that corticosteroid injection is not without risk (particularly triamcinolone). Significant side effects such as fat pad atrophy, plantar fascia rupture, and osteomyelitis of the calcaneus may occur. Furthermore a prospective randomised study found no significant difference between corticosteroid and lignocaine injection or lignocaine alone.⁴

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- 1 Kane D, Greaney T, Bresnihan B, Gibney R, FitzGerald O. Ultrasound guided injection of recalcitrant plantar fasciitis. *Ann Rheum Dis* 1998;57:383-4.
- 2 Powell M, Post WR, Keener J, Wearden S. Effective treatment of chronic plantar fasciitis with dorsiflexion night splints: A crossover prospective randomised outcome study. *Foot Ankle Int* 1998;19:10-18.

- 3 Mizel MS, Marymont JV, Trepman E. Treatment of plantar fasciitis with a night splint and shoe modification consisting of a steel shank and anterior rocker bottom. *Foot Ankle Int* 1996;17:732-5.
- 4 Blockley NJ. The painful heel. *BMJ* 1956;iii:1277-8.

Authors' reply

Drs Quinn and Gough make a number of valid points concerning ultrasound, corticosteroid injection of the heel and plantar fasciitis. The development of portable ultrasound machines with 7.5-12.5 MHz transducer frequency and improved image processing and quality is an exciting new development in rheumatology. Our report was intended to highlight a potential application of this new technology and to outline the advantages ultrasonography might offer over bone scintigraphy in guided injection of the heel and other regions. In our report we freely accepted the criticisms that the numbers are small and that the study was open. Over the past 18 months we have been trying to resolve this by prospectively randomising patients with plantar fasciitis to a trial of ultrasound guided injection as compared with palpation guided injection.

We agree that in the majority of patients conservative measures will lead to a satisfactory outcome.¹ Despite this many patients are still undergoing surgery for plantar fasciitis.^{2,3} Nightsplinting, corticosteroid injection, radiofrequency neurolysis, dexamethasone iontophoresis, open and endoscopic plantar fasciotomy, and surgical neurolysis have all been reported as having variable success in chronic plantar fasciitis. It is difficult to compare these reports as patient selection and measures of outcome vary. We agree that night splints are very effective but they require fitting and good patient compliance for at least 4 to 8 weeks. We believe that corticosteroid injection also has a role in the management of plantar fasciitis that is unresponsive

to conservative treatment and that it produces a more rapid amelioration of symptoms than splinting. The patients in our report fall into this category and were all recalcitrant to orthotics, non-steroidal anti-inflammatory drugs, stretching exercises, and palpation guided injection.

The prospective randomised trial of hydrocortisone acetate and lignocaine versus lignocaine alone, which is referred to, did show a greater rate of cure in patients who received hydrocortisone (10 of 13 v 5 of 9), though this did not reach statistical significance. In a retrospective study of 411 patients with plantar fasciitis corticosteroid injection was regarded by patients as a highly effective treatment.⁴ In addition scintigraphic guided injection produced an 80% cure rate in patients with recalcitrant plantar fasciitis.⁵ As we have stated in our report a properly controlled randomised study is required to establish the exact role of corticosteroid injection in plantar fasciitis.

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- 1 Wolgin M, Cook C, Graham C, Mauldin D. Conservative treatment of plantar heel pain: long-term follow-up. *Foot Ankle Int* 1994;15:97-102.
- 2 Tomczak RL, Haverstock BD. A retrospective comparison of endoscopic plantar fasciotomy to open plantar fasciotomy with heel spur resection for chronic plantar fasciitis/heel spur syndrome. *J Foot Ankle Surg* 1995;34:305-11.
- 3 Sammarco GJ, Helfrey RB. Surgical treatment of recalcitrant plantar fasciitis. *Foot Ankle Int* 1996;17:520-6.
- 4 Gill LH, Kiezbak GM. Outcome of nonsurgical treatment for plantar fasciitis. *Foot Ankle Int* 1996;17:527-32.
- 5 Dasgupta B, Bowles J. Scintigraphic localisation of steroid injection site in plantar fasciitis. *Lancet* 1995;346:1400-1.