ALGODYSTROPHY AND OSTEOPOROSIS AFTER TIBIAL FRACTURES

P. P. SARANGI, A. J. WARD, E. J. SMITH, G. E. STADDON, R. M. ATKINS

From Bristol Royal Infirmary, England

We made a prospective study of the incidence and natural history of algodystrophy and associated changes in bone mineral density in the ankles and feet of 60 consecutive patients who had suffered unilateral fractures of the tibial shaft. At bone union, 18 patients showed signs of algodystrophy. Its development was independent of the type of fracture management and of the severity of injury. Patients with algodystrophy lost significantly more bone mineral than did those without but the degree of this loss was independent of the type of treatment and of the time to fracture union.

In most cases the symptoms resolved within six months of fracture union but in four patients they were still present at one year and two of these had not returned to work.

Received 25 August 1992; Accepted 29 October 1992

Algodystrophy (reflex sympathetic dystrophy or Sudeck's atrophy) is a disorder characterised in its early stages by pain and tenderness with vasomotor instability, abnormal sweating, swelling, and joint stiffness. As the syndrome progresses, the swelling and vasomotor instability disappear but in severe cases contractures and joint stiffness result (Kozin et al 1976; Doury, Dirheimer and Pattin 1981). In chronic cases severe osteoporosis develops.

Until recently the syndrome was considered to be a rare complication, occurring in less than 2% of fractures (Plewes 1956; Pool 1973; Poplawski, Wiley and Murray 1983). An incidence of up to 30% of a mild form of the dystrophy, however, has been reported after Colles' fracture (Hoffman 1953; Atkins, Duckworth and Kanis 1990). Although the symptoms are transient in most cases, they nevertheless cause considerable morbidity and some patients continue to suffer for more than a year (Bickerstaff 1990).

Joint stiffness is a common complication of tibial fractures. Ellis (1958) and Nicoll (1964) both reported that stiffness was more common after severe fractures and in those with associated vascular injuries but stiffness also occurs after simple injuries and in the absence of vascular damage. In these cases it may be the result of algodystrophy.

Post-traumatic loss of bone mineral content is a well-known phenomenon (Nilsson 1966) and losses of up to 50% have been reported after tibial shaft fractures (Uliviéri et al 1990). The cause remains speculative, and the relationship between post-traumatic and dystrophic osteoporosis is not clear. Osteoporosis after fractures of the lower limb may cause refracture from minimal trauma (Sarangi, Ward and Atkins 1992).

We have documented the incidence of algodystrophy of the foot and ankle after tibial shaft fractures and have investigated its relationship to post-traumatic osteoporosis.

PATIENTS AND METHODS

Between January 1990 and March 1991, we treated 64 patients at the Bristol Royal Infirmary for isolated fractures of the tibial shaft. Four patients refused to participate in the study, leaving 60 who were prospectively enrolled. Their mean age was 32.4 years (17 to 68). Inclusion in the study did not affect the clinical management of the patient or the routine of the admitting consultant.

Thirty-one fractures were treated in plaster casts, 26 of them with manipulation before application, 15 by intramedullary nailing, 7 by open reduction and internal fixation using AO plates and screws, and 7 by external fixation. The time to weight-bearing was recorded.

Not more than two weeks after union of the fracture the patients were examined for signs of algodystrophy such as pain and tenderness, swelling, joint stiffness and vasomotor instability. At the same time estimations of bone mineral density were made by dual-energy X-ray absorptiometry (DEXA). All patients who showed the features of algodystrophy were re-examined at intervals to establish the natural history of the condition.

Pain and tenderness. Pain in the ankle and foot (but not at the fracture site) was assessed by direct questioning. Tenderness of the foot was measured by dolorimetry.
which has been described elsewhere (Sarangi et al. 1991). The summed dolorimetric ratio (SDR) is highly sensitive in differentiating patients with algodystrophy from normal control patients, and is highly reproducible, with a coefficient of variation of 6% and a normal range of 0.78 to 1.21 (Sarangi et al. 1991).

**Vasomotor instability.** Assessment was by questioning and examination. Skin coloration, excessive sweating and subjective skin temperature variations were noted.

**Joint stiffness.** The range of movement of the injured ankle was expressed as a percentage of that of the other ankle. Reduction of the arc of movement by more than 50% was classified as severe stiffness.

**Swelling.** Swelling was expressed as the difference between the bimalleolar circumferences of the injured and the uninjured ankle.

The reproducibility of these measurements was assessed for one observer who measured 20 patients, each of whom was examined twice with an interval of 90 minutes between each examination. The coefficient of variation was 10% for swelling and 8% for stiffness.

**Bone mineral density.** Bone mineral density (BMD) was measured in the distal metaphysis and the calcaneum of both legs using DEXA. The BMD of the fractured leg was expressed as a percentage of that of the uninjured leg. Previous examination of 30 normal subjects had shown that although there was much variation in density between subjects at a given site, there was no significant difference between similar sites in both legs of the same individual. Longitudinal studies on ten patients with fractures of the tibial shaft showed no significant changes in the BMD of the uninjured leg from the time of injury to the time of union of the fracture.

**RESULTS**

Of the 60 fractures, 59 united in a mean time of 16.1 weeks (11 to 23). One closed mid-shaft fracture in a 24-year-old woman treated in a plaster cast did not unite until 45 weeks after injury. This patient was excluded from the following analyses.

The mean time between injury and assessment was 17.2 weeks (13 to 25).

**Pain and tenderness.** At the time of assessment 19 patients complained of pain or discomfort in the ankle or foot. The dolorimetric ratios varied from 0.45 to 1.1. 20 patients having a ratio below the normal range, indicating considerable tenderness. There was a significant correlation between pain and a reduced dolorimetric ratio; 19 patients had both, 39 had neither and one had a reduced dolorimetric ratio but no complaint of pain (chi-squared test = 25, p < 0.001).

**Vasomotor instability.** Vasomotor instability was found in 22 patients: 15 had excessive sweating, 21 had blue or red skin discoloration, 20 experienced excessive warmth or cold in the affected limb and 15 had all these features.

**Joint stiffness.** Slight loss of ankle movement was a common finding; severe stiffness was found in 22 patients. **Association of features.** Pain, dolorimetric tenderness, vasomotor instability and gross ankle stiffness were all present in 18 patients; 33 had none of these features. Using log-linear analysis (Armitage and Berry 1987) the association between dolorimetric tenderness, severe stiffness and vasomotor instability was found to be highly significant. We therefore deemed the 18 patients with all four features to have algodystrophy and treated the other 33 as a control group.

Eight patients had one or two of the above features. Four had only red discoloration of the skin, two had severe ankle stiffness after high-energy injuries, one had a painful pin-track infection and dolorimetric tenderness, and one had dolorimetric tenderness but none of the other features of algodystrophy.

**Swelling.** Nearly all the injured ankles were swollen, but swelling was significantly greater (p < 0.001) in the patients with the signs of algodystrophy (mean = 29.5 mm ± 8.5, range 15 to 45) than in the control group (mean = 14.3 mm ± 11.5, range 0 to 50). Furthermore, 75% of patients with swelling greater than 30 mm had algodystrophy.

There was no significant association between algodystrophy and the type of treatment. Algodystrophy occurred in 9 of the 30 patients treated in plaster casts, in 5 of the 15 treated by intramedullary nailing, in 2 of the 7 treated with plates and screws, and in 2 of the 7 treated by external fixation. Nor was there any association with the severity of the injury or the quality of reduction of the fracture. There was no difference in the time to union between the dystrophic patients (mean 16.2 weeks, range 12 to 23) and the control group (mean 16.1 weeks, range 11 to 22).

**Bone mineral density.** The BMD was measured an average 18 weeks after injury (13 to 25). Five patients, all in the control group, declined DEXA scans.

All patients measured had lost bone mineral from the distal tibial metaphysis (mean loss 39.1% ± 13%; range 15% to 69.3%) and the majority had lost bone from the calcaneum (mean 30.9% ± 21.2%; range -80.1% to +20%). The patients with algodystrophy all lost bone mineral and their loss was significantly greater at both sites than in the control group (Table I). The loss of bone mineral did not correlate with the type of treatment; fractures treated by internal fixation lost as much bone as those treated in plaster casts. There was no significant

<table>
<thead>
<tr>
<th>Site</th>
<th>Algodystrophy (n=18)</th>
<th>Control (n=36)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal tibia</td>
<td>49.5 ± 11.1</td>
<td>34.5 ± 10.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>(−25.2 to −69.3)</td>
<td>(−14.3 to −55.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcaneum</td>
<td>45.1 ± 22.0</td>
<td>24.4 ± 16.8</td>
<td>&lt;0.002</td>
</tr>
<tr>
<td>(−13.6 to −81.1)</td>
<td>(+21.5 to −52.3)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
association between loss of BMD and length of time to fracture or length of time to measurement.

The one patient in whom union was delayed until 45 weeks had the signs of algodystrophy: pain, tenderness, severe stiffness and vasomotor instability. There was no ankle swelling, but the skin was atrophic and shiny.

**Long-term outcome.** In most cases the algodystrophy had resolved by six months after fracture union. In four patients, however, the features were still present at this time. These patients became chronic cases and even at one year continued to suffer significantly, two of them being unable to return to work. All the other patients in our study had returned to their employment by this time. There was therefore a significant association between failure to return to work and continuing features of algodystrophy (chi-squared test $= 15.2$, $p < 0.001$).

**DISCUSSION**

The incidence of algodystrophy after tibial fractures in our series was 30% which is similar to that of algodystrophy of the hand after Colles' fracture (Hoffman 1953; Atkins et al 1990). We made the diagnosis of algodystrophy at fracture union, by which time some cases may have resolved, so it is likely that we have underestimated the true incidence.

We have shown that the development of algodystrophy is independent of the type of treatment and that internal fixation and early weight-bearing did not prevent it. The incidence of algodystrophy was similar whether the fracture was treated in a plaster cast or by internal fixation and it seems that the 'fracture disease' described by the AO group (Müller et al 1979) does not affect every leg treated in a cast. It is possible, however, that the early joint mobilisation which internal fixation allows may minimise the later joint stiffness.

Algodystrophy can significantly delay the patient's return to work if the symptoms persist for more than six months. It did not, however, increase the time to fracture union, despite the association between dystrophy and greater bone mineral loss.

Post-traumatic bone mineral loss is generally thought to result from decreased functional loading of the fractured limb (Rubin and Lanyon 1984). Finsen and Benum (1989), however, showed that early weight-bearing after ankle fracture made no difference. The increased bone mineral loss seen in our patients with algodystrophy may have been due to increased bone resorption as part of that condition, or it may have been the result of decreased load-bearing on a limb made more painful by the algodystrophy.

Recovery of lost bone mineral content is slow and a deficit of as much as 25% may persist for many years after fracture (Nilsson 1966). Finsen, Haave and Benum (1989) thought that post-traumatic osteoporosis could lead to permanent weakness of the bone, noting that patients who had had femoral or tibial fractures were likely to sustain a second fracture of the same bone. We have recently shown that persistent regional osteoporosis can predispose to a further fracture after a trivial injury (Sarangi et al 1992). It seems reasonable to suggest that, in those patients in whom continued symptoms of algodystrophy reduce the normal use of the leg, the syndrome may retard or even prevent the normal recovery of bone mineral content, leaving these patients vulnerable to second fractures.

We conclude that algodystrophy is a significant cause of morbidity after tibial fractures. Since most of the patients who sustain these injuries are young men in employment, the development of algodystrophy has long-term social and economic implications which merit its further investigation.

This work was supported by a grant from the South Western Regional Health Authority. No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

**REFERENCES**


