Spinal surgery and severe vitamin D deficiency

Clinical records

Case 1: A 46-year-old Indian man required lumbosacral (Steffee) fusion[1] for chronic low-back pain after a successful laminectomy for disc herniation sciatica. Good pain relief was obtained for four weeks after the operation, and then his back pain recurred. After a brief period of symptomatic treatment, he was re-operated on at six weeks, and the metal screws and plates, which were found to be loose in the bone, were removed. The bone was noted to be softer than normal and the fusion was not sound. Swabs for infection were sterile.

The patient gave a history of long-term adherence to a vegetarian diet, as well as continuous night-shift work for 10 years with minimal sunlight exposure. He had never smoked. His 25-hydroxyvitamin D (25OHD) concentration was < 12 nmol/L (reference range, 35–155 nmol/L). The results of laboratory tests were calcium, 2.27 mmol/L; albumin, 38 g/L; phosphate, 1.33 mmol/L; alkaline phosphatase, 179 U/L (reference, < 120 U/L); and γ-glutamyltransferase, 195 U/L (< 65 U/L). The patient was taking carbamazepine, which may account for the liver function abnormalities.

Vitamin D supplementation (ergocalciferol, 2000 units daily) was commenced. Dietary calcium intake was assessed as at least 800 mg/day. Physiotherapy, initiated because of his back problems, was continued and his work was transferred to day-time shifts only. After eight weeks of treatment and sunlight exposure, the results of laboratory tests were calcium, 2.44 mmol/L; albumin, 45 g/L; alkaline phosphatase, 117 U/L; γ-glutamyltransferase, 102 U/L; and 25OHD, 125 nmol/L. Plain x-ray and computed tomography scan showed his fusion to be sound at 12 months and his symptoms to be much improved.

Case 2: A 49-year-old white woman was admitted for elective removal of metal plates (Steffee) from L2–L3 fusion two years previously. Before x-ray films were obtained, it was hoped that her fusion was sound. However, imaging had suggested the pedicle screws had loosened, contributing to ongoing low-back pain and sciatica (Figure 1). Furthermore, x-ray of the lumbar spine in flexion showed narrowing of the angle between the end-plates of L2 and L3, indicating movement posteriorly (Figure 2). At operation, all screws were grossly loose, and a bone graft placed posterolaterally had not joined to the vertebral bodies. Further bone chips were inserted, and a bone biopsy was taken. Swabs for infection were sterile. Areas of woven bone were present and the appearance of the osteoid suggested a mineralisation defect, although undecalcified sections were not available.

This patient had a four-year history of insulin-requiring diabetes mellitus, for which she also took metformin, glibenclamide and pioglitazone. Other medical problems included obesity (body mass index, 44 kg/m^2), hypertension and bronchospasm, treated without long-term inhaled or systemic corticosteroids. She had never smoked. Her 25OHD concentration was < 12 nmol/L. Ergocalciferol 3000 units daily was commenced. The serum alkaline phosphatase level was normal (79 U/L). Dietary calcium intake was considered adequate. Specific questioning revealed a history of "hating the sun", and avoiding exposure to sunlight throughout her adult life. One month later, her 25OHD concentration was 27 nmol/L and the dose of ergocalciferol was increased to 4000 units daily. Six months after operation, imaging suggested that the new bone graft had become incorporated into the spine.

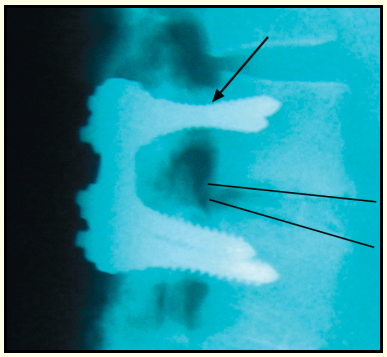

Figure 1: Lateral x-ray of the lumbar spine in extension. Arrow denotes a radiolucent zone at the margin of the upper screw, indicating loosening.

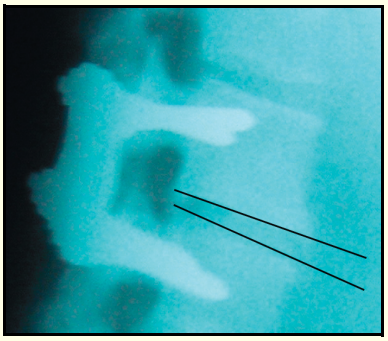

Figure 2: Lateral x-ray of the lumbar spine in flexion, showing significant narrowing of the angle between the endplates of lumbar vertebrae 2 and 3, compared with Figure 1.

Recently, attention has again been drawn to the high prevalence of severe vitamin D deficiency in certain population groups in Australia, including veiled and dark-skinned women and their children.[2,3] Another group at high risk are the elderly: 67% of older patients admitted to a short-stay geriatric rehabilitation unit were found to have vitamin D deficiency.[4] A similar prevalence has been found in residents of nursing homes.[5] However, it is not widely appreciated that moderate to severe vitamin D deficiency may also occur in middle-aged people.

Our two patients were in their late 40s when severe vitamin D deficiency became apparent. In the first patient, recurrent pain a short time after surgery was of such severity that internal fixation plates had to be removed. The screws were loose, and, in the absence of infection, the profound vitamin D deficiency can be assumed to have resulted in failure of mineralisation of osteoid and hence failure of the surgical procedure, with resulting increased morbidity.

This patient had multiple risk factors for vitamin D deficiency (pigmented skin, habitual night-shift work and a vegetarian diet). Although he now exercises regularly, including during daylight hours, and works day-time shifts, he has required ongoing vitamin D supplementation to maintain satisfactory 25-hydroxyvitamin D (25OHD) concentrations.

The second patient's bone had an abnormal histological appearance, and, despite an omnivorous diet, her choice to avoid direct sunlight for many years had resulted in profound vitamin D deficiency.

Vitamin D deficiency of the severity reported here is extremely uncommon in younger Australian adults. However, patients with long-standing orthopaedic or spinal

Lessons from practice

- Persistence or recurrence of low back pain and sciatica after spinal fusion surgery may indicate failure of the operation.
- Risk factors include infection, smoking and vitamin D deficiency.
- Patients at any age who actively avoid exposure to sunlight are at risk of vitamin D deficiency.
- Vitamin D deficiency is readily identified and corrected and should be considered in patients who may require spinal fusion surgery.

disorders who become housebound may be particularly at risk, irrespective of their age.

The functions of vitamin D in bone metabolism are well known and its deficiency may be reconciled with the failure of spinal fusion, as described in these patients. This report highlights the need for attending surgeons and physicians to be aware of the potential for vitamin D deficiency in their patients, as failure to recognise this easily reversible problem may result in complications of treatment, including failure of spinal fusion surgery, additional morbidity and the substantial costs of further surgery and hospitalisation.

Walter E Plehwe,* Roy P L Carey†

*Consultant Endocrinologist, †Orthopaedic Surgeon
Epworth Medical Centre, Richmond, VIC

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OBITUARY

Henry Oliver Lancaster
AO, FAA, DSc, MD, PhD, MB BS, BA

HENRY OLIVER LANCASTER was born in Sydney on 1 February 1913, but spent his early years in Kempsey, NSW, where his father had a medical practice. His father died when Oliver was almost nine, and he boarded in Kempsey through his primary and West Kempsey Intermediate High School years. After a year studying economics/arts at Sydney University, he enrolled in medicine in 1931 and graduated in 1937. In that year he worked as a Resident Medical Officer at Sydney Hospital, and in 1938 as a pathologist and Senior Medical Officer.

Lancaster joined the Australian Imperial Force as a Medical Officer in 1940, and later served as a pathologist in the Middle East and New Guinea. His first two papers (jointly with T E Lowe), on worm infestations in troops, were published in the *Medical Journal of Australia* in 1944. The same year, a secondment to the Australian New Guinea Administrative Unit awakened his interest in demography and sparked his return to the serious study of mathematics.

From 1946 to 1948, while on a temporary appointment to the School of Public Health and Tropical Medicine (SPHTM) (affiliated with Sydney University), he took the opportunity to study advanced mathematics and read the work of the English and American medical statisticians/epidemiologists. In 1948, he left to spend a year as Rockefeller Fellow in Medicine at the London School of Hygiene.

After returning to work at the SPHTM, Lancaster was involved with statistical and epidemiological studies of disease in Australia, including diabetes, cancer (notably melanoma and its association with latitude) and tuberculosis. In 1941, the ophthalmologist Norman Gregg had observed that many cases of cataract were the result of maternal rubella in the first month of pregnancy. This was to lead to Lancaster's landmark discovery that "ordinary" rubella infection of pregnant women (rather than a new, highly virulent strain, as Gregg had thought) was linked with congenital deafness of offspring.[1]

In 1959, Lancaster was appointed to the Foundation Chair of Mathematical Statistics at the University of Sydney, a position he held until his retirement in 1978. He was elected a Fellow of the Australian Academy of Science in 1961, and awarded its Thomas Ranken Lyle Medal for Physics and Mathematics in the same year. He was awarded an MD in 1967, and in his retirement returned to intensive writing in medical statistics. His publications included *Expectations of life* (1990), a massive study of world mortality, and *Quantitative methods in biological and medical sciences. A historical essay* (1994).

Oliver was made an Officer of the Order of Australia in 1992 for services to science. An account of his career, *Some recollections of Henry Oliver Lancaster* (1996), edited by his brother Richard, is in the Sydney University archives. He died in Sydney on 2 December 2001 of coronary heart disease.

Eugene Seneta

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