



■ HISTORICAL NOTE

A history of osteomyelitis from the *Journal of Bone and Joint Surgery*

1948 TO 2006

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Osteomyelitis is one of the oldest diseases known. It took many years before the acute infection could be brought under control with antibiotics and chronic osteomyelitis remains difficult to manage. The modern history of the disease is reflected in the pages of the *Journal of Bone and Joint Surgery*.

This paper describes the history of acute haematogeneous osteomyelitis. It is based on a review of articles in the British Volume of the *Journal of Bone and Joint Surgery* from the start of the Journal in 1948 to 2006. The British Volume began when knowledge of the effects of penicillin was becoming increasingly common and the morbidity of osteomyelitis more controlled. The oldest known evidence of osteomyelitis lies in the fractured spine of a dimetrodon Permian reptile, which was in existence 291 to 250 million years ago.¹ There is clear evidence of inflammation where the bone was roughened and swollen above the fracture, showing the fracture to have become infected. There is also histological evidence of the condition.² Dating back to Hippocrates (460-370 BC), infection after bony fracture has been recognised, although the clinical picture of acute haematogeneous osteomyelitis was only recognised much later. It was described by Bromfield³ in 1773 as bone that “may become carious, first in its internal parts and that from external injury, as well as from a vitiated state of the animal fluids.” He termed the disease, “Abcessus in medulla”, and advised that “we cannot be too early in letting it out when we are assured of matter being under the perosteum”. In 1831, Smith,⁴ the first Professor of Surgery at Yale University, observed that “a very great majority of patients survive the attack, albeit with long confinement, protracted suffering and great emaciation. In a few cases, however, the disease proves fatal.” He called the disease necrosis, to indicate the death of bone. Nelaton⁵ is credited with introducing the term “osteomyelitis” in 1844.

Before Ogston⁶ (1844 - 1929) had identified and named staphylococci, Pasteur⁷ had implicated microbes in furuncles, osteomyelitis and

puerperal fever. He had described osteomyelitis as “a boil of the bone marrow”. Ogston, Professor of Surgery in Aberdeen, was one of the first to grasp the importance of the antiseptic system and germs as a cause of sepsis. He examined pus, carefully removed with antiseptic precautions, from a series of 82 abscesses. There were 13 from typical cold abscesses in which he found no organisms. In 65, from acute abscesses of a few days, there were micrococci. Four were chronic abscesses whose duration could be measured in weeks and were similar, but not identical to, the 65. He distinguished two kinds of micrococci; those forming chains and those forming groups, “like the roe of a fish in clusters”.⁶ He named the organism that grew in masses like bunches of grapes, Staphylococcus, at the suggestion of Geddes, the Professor of Greek in Aberdeen. For those which grew in chains, he accepted the name, Streptococci, introduced by Billroth.⁸

Prior to the introduction of penicillin in 1940 by Chain et al,⁹ the management of acute osteomyelitis was based on the principles of the American surgeon, Winnett Orr¹⁰ (1877 - 1956). These were a combination of both radical and conservative measures. He aimed to undertake one primary operation at which an attempt was made to completely remove all diseased tissue. This could be done at any stage of the illness and no distinction was made between acute and chronic cases. He advocated incisions which were large enough to expose the whole diseased areas of the bone. All necrotic bone was to be removed with “saucerisation” of the resulting cavity. The wound was left open and vaseline gauze used for packing. The limb was then immobilised in plaster in a neutral position. Dressings and

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plasters were changed as infrequently as possible in an effort to reduce secondary infection. Pain and fever provided the main indication for inspection of the wound. No antiseptics were used, as he relied on the natural defence mechanisms of the tissues. Braces were required in the later stages.¹⁰

The first paper on osteomyelitis in the *Journal* in February 1948, was from the Royal Hospital for Sick Children in Glasgow,¹¹ and dramatically illustrates the benefits of penicillin which had gradually become available in the 1940s. It noted that there had been little change in the incidence in Glasgow since the beginning of the century, and, before the introduction of chemotherapy in the form of sulphonamides, the mortality has remained high. Most cases were admitted with toxæmia and treatment of the local bony focus had little effect in arresting the disease. Surgical treatment included incision of soft-tissue abscesses, immobilisation, drilling and guttering, and even amputation. At post-mortem examination, empyema, lung abscesses, purulent pericarditis and widespread evidence of pyæmia were seen. Between 1936 and 1940, there were 69 patients with 23 deaths, giving a mortality of 33%, but between 1941 and 1945, there were 70 patients, with only seven deaths, a mortality of 10%. In the series described in the paper, there were 30 patients of the septicaemic type treated by penicillin. Of these, 28 (93%) were well and their radiographs showed no evidence of bone disease.

At that time, the duration of treatment and the required dosage of penicillin required were ill-established. The most economical method of administration was by drip infusion into a muscle, which must have been painful. Continuous infusion was then abandoned in favour of intermittent intramuscular injections. The veins of children were not regarded as suitable for continuous intravenous infusions. In an editorial three years later, Wilkinson¹² summed up the position at the start of the 1950s. He stated that "acute haematogenous osteomyelitis need not, and should not, be a fatal or crippling disease. Early and adequate treatment with penicillin aborts the disease with minimal bone changes and lowers the mortality to almost zero". The main issue was early diagnosis so that adequate treatment could be started immediately. Osteomyelitis, like tuberculosis, was arrested, but not eradicated; sequestration, sinus formation, and early flare-ups were far less common than they had been, and involvement of more than one bone was comparatively rare.¹² This cautious prognosis was to develop positively over the years until Nade¹³ in 1983 stated that acute osteomyelitis was "a curable disease."

In 1959, Trueta¹⁴ made an important contribution to our understanding of acute haematogenous osteomyelitis. He recognised three types based on the blood supply of the bone. In the infant, the inflammation caused severe, and often permanent, damage to the epiphysis and metaphysis with a large involucrum, but only transient damage to the shaft. In the child, there was extensive cortical damage with the formation of an involucrum, but it was rare for there to

be permanent damage to the growth plate apart from some stimulation of growth.

In the adult, acute osteomyelitis of the long bones is rare. The cortex is absorbed instead of sequestering. The whole bone may be invaded and frequently remains chronically infected. Specific antibiotic treatment started as soon as possible in adequate doses formed the basis of successful treatment.

In 1962, Gilmour¹⁵ analysed a series of 328 cases. In the first group of 27, between 1944 and 1950, penicillin gave excellent results. After 1950, the disease took on new characteristics in relation to frequency, severity and age incidence, with the appearance of a penicillin-resistant *Staphylococcus*. Between 1951 and 1960, 251 cases were recorded. With the advent of penicillin-resistant staphylococci, there was an increase of infection in neo-natal infants. Early operation did not prevent extensive sequestration. Nevertheless, it was considered that decompression had a beneficial influence on the course of osteomyelitis. Interruption of the progressive elevation of the periosteum would reduce the amount of cortical necrosis, which was determined by the amount of bone deprived of its blood supply, and the value of draining a tense collection of pus in a sick and distressed child was unquestionable.

Primary subacute pyogenic osteomyelitis

In 1965, Harris and Kirkaldy-Willis¹⁶ described primary subacute pyogenic osteomyelitis. The main clinical features were the long history with often weeks or months before diagnosis. Little systemic reaction to the infection and minimal physical signs were also typical. Osteomyelitis of the vertebral bodies in adults is included in this group.

The most useful aids for diagnosis at the time were the staphylococcal antibody titres and the erythrocyte sedimentation rate. A limited surgical exposure was usually necessary to isolate the causative organism and avoid empirical antibiotic therapy. Curettage and local antibiotics were usually sufficient to treat a localised bone abscess.¹⁶ These observations were re-inforced in a paper from Durban, South Africa, which described 21 children with primary subacute haematogenous osteomyelitis. The subacute form of the disease was considered to occur as a result of increased host resistance and decreased bacterial virulence.¹⁷

Blockey and Watson¹⁸ and Blockey and McAllister,¹⁹ in Glasgow, clarified the relative roles of antibiotics and surgery in two papers in the early 1970s. They advocated a policy of rest and antibiotics based on a study of 113 children. Surgery was only indicated if pus was clinically detected. They noted that the organisms causing the disease were less sensitive to penicillin than they had been ten years previously, and that a proportion were also likely to become resistant to methicillin and cloxacillin.

Further changes in the pattern and incidence of osteomyelitis are described in a paper from Glasgow in 2001, showing the decline of both acute and subacute haemato-

genous osteomyelitis in children under 13 years of age between 1990 and 1997.²⁰ In this period, there was a fall of 44% for both forms of osteomyelitis. This was similar to the decline of just over 50% which had previously been reported in the same population between 1970 and 1990.

Staphylococcus aureus remained the most commonly isolated pathogen. Only 20% of the patients required surgery. There was a low rate of complications of 10% after the acute form, and none in the subacute form. It was concluded that haematogenous osteomyelitis was becoming a rare disease in children in Glasgow with an annual incidence of 2.9 per 100 000 of the population. There clearly has been a big change in the prevalence of the disease since 1948, which may be related to improvements in nutrition and the general standard of living.

The comprehensive overview by Nade in 1983¹³ gives an up-to-date account of the subject, and despite the fact that it is now over 20 years old, it still reflects the principles of modern clinical practice which had been established by the early 1970s.

Chronic osteomyelitis

With the help of antibiotics, chronic osteomyelitis is potentially curable,²¹ but surgery is needed to improve the blood supply to the affected bone. It is essential that the scarring involving the bone and soft tissues is thoroughly excised to ensure an adequate blood supply for delivery of the antibiotic to the chronically infected bone. The dead space left by the excision must be filled by well-vascularised unscarred living tissue. Muscle grafts are usually available in the thigh or leg. In areas where these are impractical, or where the skin is widely scarred, skin flaps are used. Rowling and Kirkcaldy²² showed in 1970, that the results were improved by the use of Fucidin which penetrates bone very well. The removal of sequestra and scar tissue is usually achieved easily, but elimination of the dead space is the more difficult procedure. A free skin or muscle flap may be technically impossible in some situations. De Oliveira²³ described the use of cancellous bone chips, which are resistant to infection, in 120 patients with osteomyelitis. The results were gratifying with only four relapses.

McNally et al²⁴ from Belfast, reported a series of 37 patients treated in two stages. The first was a radical debridement of all infected bone and soft tissue with the provision of soft-tissue cover, followed by a second, three to six weeks later, for grafting with bone chips from the iliac crest.

For infection of long bones treated by internal fixation, the Lautenbach procedure has been advocated.²⁵ This involves debridement, intramedullary reaming and insertion of tubes with double lumina in the marrow cavity. The latter provides a system of delivery of local antibiotics and allows for analysis of the fluid used for irrigation for volume and bacterial culture. Each tube, 6 mm in diameter, traverses the length of the marrow cavity. The outer tube is a large suction drain, perforated at intervals and used to

drain effluent and measure volumes in the cavity. The inner tube is a central venous line used to deliver antibiotics. The end-point of treatment is reached when the irrigation fluid produces three consecutive clear cultures and obliteration of the volume of the cavity. Success was achieved in 17 patients in a series where osteomyelitis had been present for a mean of 12.5 years.²⁵

Professor Simpson and his team in Edinburgh have produced useful information on the effect of excision of various amounts of bone.²⁶ They prospectively studied a consecutive series of 50 patients with chronic osteomyelitis. The patients were allocated to the following treatment groups at the time of surgery: 1) wide resection, with a margin of clearance of 5 mm or more; 2) marginal resection, with a clearance of less than 5 mm; and 3) intralesional biopsy, with debulking of the infected area. All patients had a course of antibiotics intravenously for six weeks, followed by oral administration for a further six weeks. No patient in group 1 had a recurrence. In patients treated by marginal resection, eight of the 29 (28%) had a recurrence. All patients who had a debulking had a recurrence within one year of surgery. Thorough excision is clearly beneficial.

Rarities

In 1951, Gallie described the recurrence of osteomyelitis 80 years after the original infection had occurred at the age of ten.²⁷ This presented in a 90-year-old woman as a Brodies' abscess at the lower end of her femur (the site of her previous infection), where the bone was thickened and tender, despite a lifetime without symptoms. A cavity 1.5 inches (3.25 cm) long was uncovered and partially saucerised and a sequestrum, which presumably had been there for 80 years, was removed. Its surface was much pitted and in the pits were masses of granulation tissue.

In 1963, a report of osteomyelitis variolosa appeared from Northern and Southern Rhodesia (now Zimbabwe and Zambia), to remind those who had not seen smallpox of its most important and easily recognised complications.²⁸ Bone infection can be late, silent and often unexpected. It is usually symmetrical and almost always multiple. It affects the hands, legs, and feet. It is destructive, unpreventable and untreatable. It ends with deformity but not loss of life. It is the only known example of a virus infection of bone, and fortunately the disease has now been eradicated.

Summary

The reports in the *Journal* show how acute haematogenous osteomyelitis can readily and successfully be managed in most parts of the Western world. With the improvement in the general standard of living, hygiene and nutrition, the incidence of acute haematogenous osteomyelitis has declined, in contrast to developing countries where it remains a dangerous hazard. The treatment of chronic osteomyelitis remains difficult everywhere, but there are clear principles of treatment.

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