

PRIMARY SKIN ACTINOMYCOSIS OF THE CHEST WALL

D. K. KAPUR, J. D. A. ROBB, P. FERGUSON, D. DOWNING

Surgical Unit, Route Hospital, Ballymoney, Co. Antrim, Northern Ireland

ALTHOUGH actinomycosis is believed to be a rare disease, an increasing number of case reports suggest that this may not be so. Many cases may not be recognised, because of their abortion by the widespread use of antibiotics. Another pitfall in the diagnosis is the failure to obtain anaerobic cultures in suspected cases (Shah 1971).

A presentation with some unusual features is described and some points emerging from recent literature are discussed.

CASE REPORT

The patient, an eleven-year-old schoolboy, was admitted on 8th March 1973 to the Route Hospital, Ballymoney, with a large painful swelling on the right lower chest posterolaterally. Previously, on 15th September 1972, he had had an appendicectomy for acute appendicitis. The appendix was reported as normal on microscopic examination.

On examination, he looked unwell, but his temperature was normal. There was a tender swelling over the ninth and tenth ribs; this was red and fluctuant. Auscultation of the chest was clear and both abdominal and rectal examination revealed nothing abnormal. A provisional diagnosis of pointing sub-diaphragmatic abscess or empyema necessitans was made. An x-ray of the chest and ribs, together with screening of the diaphragm, was carried out. This showed a soft tissue swelling on the right lateral chest wall. There was periosteal reaction on the inner aspect of the right eighth and ninth ribs in the axilla. There was slight elevation of the right diaphragm, which showed very little movement on inspiration. There was no sub-diaphragmatic fluid level. The blood picture revealed leukocytosis (haemoglobin 11.5 g/100 ml.), white cell count 11,500 (neutrophils 66, lymphocytes 24, monocytes 10). The ESR was 110 mm/hr. His weight was 4 st. 5 lb.

On 9th March 1973, the track was explored from the anterior end of the ninth rib to the posterior angle of the same rib on the right side. It was found to run deep to the external oblique muscle but did not penetrate the chest wall. At the posterior end of the wound pus extruded. This was greenish-yellow and contained beads.

The laboratory was informed of the suspicion of actinomycosis and a specimen of pus was submitted. This confirmed the presence of actinomycosis. The following day the patient commenced a course of Dalacin C 150 mg three times daily orally and procaine penicillin 0.25 mega twice daily intramuscularly.

There was a steady regression of signs and symptoms over the next week. On 15th March 1973 the haemoglobin was 10.8 g/100 ml., white cell count 9,800 and ESR 63 mm/hour. The patient was discharged on 19th March 1973, by which time he had been afebrile for six days. He continued to improve. On 29th March 1973 he was afebrile, his weight was 4 st. 12 lb., white cell count 7,800, haemoglobin 12.7 g/100 ml., ESR 43 mm/hour. At follow-up on the 13th April 1973 the treatment schedule was changed because the patient complained of headaches, dizziness, nausea and some vomiting: it was thought that this might have been a side effect of Dalacin C. Treatment was continued with procaine penicillin 0.25 mega twice daily intramuscularly, and the symptoms settled. By 17th May 1973 his weight was 5 st. 5 lb., haemoglobin 13.6 g/100 ml., white cell count 5,100, ESR 8 mm/hour and temperature 98.6°F. The wound had healed. Treatment was then stopped.

DISCUSSION

Actinomycosis is a fungal, granulomatous disease, caused in humans by *Actinomyces israeli*, and in animals by *Actinomyces bovis*. It is a chronic suppurative infection. It is characterised by its disregard for anatomical boundaries (Foley et al., 1971). The disease may occur at any age, but most frequently between 30 and 40 years. In most series, it affects males more than females (Glahn 1959). It may affect any race, and occurs in persons of all occupations (Sochocky 1972).

The epidemiology of actinomycosis is that of an endogenous infection. This was shown by Wolff and Israel (1891) and by Wright (1905). *A. israelis* is a normal commensal of the mouth and is found particularly in carious teeth, gums and tonsils. It is not clear which local conditions must be fulfilled before the organisms invade tissues and set up the characteristic diffuse, progressive inflammatory reaction (Shah 1971). The organisms have a tendency to duplicate in tissue their in vitro colonial growth by the formation of granules (Shah 1971).

The actinomycotic lesion starts as an acute suppurative inflammation with a marked tendency to form abscesses and sinus tracts. The condition progresses to intractable chronicity. The spread of actinomycotic infection is by direct extension, involving all adjacent tissue, including bone (Shah 1971). Lymphatic spread has not been reported. Blood borne spread is important, and may set up secondary actinomycotic abscesses anywhere in the body, the most common sites involved being the liver and brain (Shah 1971). Death in actinomycosis may be due to involvement of vital structures, to generalised pyaemia, or to amyloid disease (Illingworth and Dick 1968).

Diagnosis is relatively simple in advanced cases with multiple discharging sinuses; examination of the pus should yield the typical sulphur granules. When Actinomyces infection is suspected, the laboratory must be notified, because most clinical laboratories do not routinely look for anaerobic actinomycetes (Foley et al., 1971). In the acute inflammatory stage, when an abscess has formed, the diagnosis can be made reasonably easily on microscopy and culture. When a chronic inflammatory response follows without abscess formation, the diagnosis may be difficult (Shah 1971). In obscure swellings of the neck, actinomycosis must be considered in the differential diagnosis (Shah 1971).

The pathological reaction in response to infection by Actinomyces demonstrates features which emphasise two general principles of treatment – (1) intensive and prolonged antimicrobial treatment, and (2) surgical excision of the involved tissue.

Although this organism is usually sensitive to penicillin in vitro, it is often difficult for an antibiotic to reach it on account of the densely scarred avascular areas in which the organisms flourish. Wide surgical excision of the infected tissue is recommended (Eastridge et al. 1972). In lesions involving superficial structures as in this case, the wound should be packed open, and allowed to heal by secondary intention or by skin grafting at a later date (Eastridge et al. 1972). Eradication of the infection and healing of the wound will be satisfactory if excision of the involved tissue has been adequate.

Radiation, iodine, thymol, autogenous vaccines and copper sulphate were used before the advent of antibiotics (Shah 1971) which have superseded them. Yet somehow the idea persists that iodides are useful as an adjunct to penicillin or used alone in the penicillin-resistant cases (Bailey and Love 1968). There is no sound basis for the use of iodides. They were initially used because of the mistaken identity between *A. bovis* and actinobacillus of cattle and pigs, causing lumpy jaws. Suter and Vaughan (1955) have confirmed in in vitro studies that actinomycosis can grow luxuriantly in culture media containing two per cent iodides. Sulphonamides were found to be relatively inefficient and were largely replaced by penicillin, which remains the drug of choice to-day (Shah 1971).

The duration, severity, extension and involvement of various structures of the body by actinomyces should be guidelines to the amount and duration of treatment with penicillin. The value of such agents as tetracycline (Seligman 1954), lincomycin (Mohr et al., 1970) and erythromycin (Herrell et al., 1955) has been reported. Lincomycin may be advantageous, especially if there is bony involvement, because of the rapid appearance of this antibiotic in effective concentrations within bone. According to some authors (Sochocky 1972) penicillin should be given in large doses, between 10-20 million units daily for at least three months. However, several authors recommend longer treatment, lasting up to one year. Others recommend penicillin G, up to five mega units daily for four to eight weeks (Sochocky 1972). A feature of the treatment that cannot be overstated is the necessity to continue the therapy for long periods after clinical extirpation of the infection. In this case therapy was given for 68 days, that is, 30 days after the symptoms and signs and other observations had returned to normal.

It is of interest that primary skin actinomycosis is not generally recognised. Hildick-Smith et al (1964), in their modern textbook of mycotic disease, do not mention this form and take the skin symptoms to be secondary ones. Schwarz and Baum (1955) developed principal criteria for diagnosing primary skin mycoses, including actinomycosis. According to them, one should be able to distinguish clinically primary skin symptoms from secondary ones. The latter are usually multiple and at different stages of development. These secondary skin lesions have a greater tendency to progress. Lymphangitis and lymphadenitis are usually not present unless secondary infection takes place.

Most cases of primary skin actinomycosis described in the literature had, in their previous history, a traumatic incident (biting by man, fist blow in the mouth region), or else the patients were agricultural workers. Cope (1915), McWilliams (1917) and Burrows (1944), described independently three cases of primary skin actinomycosis of the hand, following a blow with the fist to the mouth region. In this case, no traumatic incident was reported to have preceded the first symptoms of the disease.

SUMMARY

A boy aged 11 years developed a superficial Actinomycotic infection of the chest wall. This has been regarded as a case of primary skin actinomycosis. The chronic history of ill health, investigation and treatment are discussed. In particular the removal of devitalised tissue and the necessity for a prolonged course of chemotherapy are stressed.

REFERENCES

- BAILEY, H., and LOVE, M. (1968). *A Short Textbook of Surgery*. ed. Capper, W., and Rains, A. J. H. H. K. Lewis, London.
- BURROWS, H. J. (1944-45). *Brit. J. Surg.*, **32**, 506.
- COPE, V. (1915-16). *Brit. J. Surg.*, **3**, 55.
- EASTBRIDGE, C. E., PRATHER, R. J., HUGHES, F. A., YOUNG, J. M., and MCCAUGHAN, J. J. (1972). *Southern med. J.*, **65**, 839.
- FOLEY, T. F., DINES, D. E., and DOLAN, C. T. (1971). *Minnesota Med.*, **54**, 593.
- GLAHN, M. (1959). *Acta chir. scand.*, **108**, 193.
- HERRELL, W. E., BALLOWES, A., and DAILEY, J. S. (1955). *Antibiotic Med.*, **2**, 507.
- HILDICK-SMITH, G., BLANK, H., and SARKANY, I. (1964). *Fungus Diseases and Their Treatment*. 2nd ed., p. 293. Little, Brown & Co., Boston.
- ILLINGWORTH, C. F. W., and DICK, B. M. (1968). *A Textbook of Surgical Pathology*, J. & A. Churchill, London.
- MCWILLIAMS, C. A. (1917). *Ann. Surg.*, **66**, 117.
- MOHR, J. A., RHOADES, E. R., and MUCHMORE, H. G. (1970). *J. Amer. med. Ass.*, **212**, 2260.
- SCHWARZ, J., and BAUM, G. L. (1955). *Arch. Derm.*, **71**, 143.
- SELIGMAN, S. A. (1954). *Brit. med. J.*, **1**, 1421.
- SHAH, J. K. R. (1971). *East African med. J.*, **48**, 498.
- SOCHOCKY, S. (1972). *South Dakota J. Med.*, **23**, 15.
- SUTER, L. S., and VAUGHAN, B. F. (1955). *Antibiot. and Chemother.*, **5**, 557.
- WOLFF, M., and ISRAEL, J. (1891). *Virchows Arch. path. Anat. Physiol.*, **126**, 11.
- WRIGHT, J. H. (1905). *J. med. Res.*, **3**, 349.