whole of his left upper chest wall anteriorly and posteriorly and radiating down the ulnar side of his arm as far as the wrist. So severe was the pain that he cried out, and 100 mg. of pethidine given intravenously gave only partial relief. No sensory defect could be demonstrated in the area of the pain, which lasted about 30 minutes and was then replaced by a sensation of numbness, soreness, and tingling. No change in size of the supraclavicular and left axillary lymph nodes (the latter was also examined by biopsy later and did not show carcinomatous invasion) was noted after the onset of the pain.

On a second occasion the patient was given 50 mg. of diphenhydramine (Benadryl) orally 30 minutes before the administration of alcohol. He was then asked to take initially 10 ml. of brandy and when this had produced no symptoms within half an hour he was asked to drink a further 25 ml. This again had no effect within the next 10 minutes, and a further 15 ml. was given, making a total of 50 ml. of brandy. About 20 minutes after the last dose the patient reported only a mild discomfort in the usual area of distribution. Again no increase in size or tenderness of his palpable lymph glands was noticeable.

On a third occasion 30 ml. of brandy was given without antihistamine being administered beforehand, and half-hourly white cell counts and differential counts were done, but these showed no significant variations compared with a count taken before alcohol was given.

Vasodilators such as trinitrin gr. 1/130 sublingually and tolazoline (Priscol) 50 mg. orally, as well as the higher alcohol derivative methylpentynol 400 mg. orally, failed to reproduce the pain.

Shortly after the start of radiotherapy to the tumour site the patient lost all spontaneous and alcohol-induced pain. Further investigations at that time showed a normal 5-hydroxyindolacetic acid excretion, the serum protein electrophoresis showed a raised alpha-2-globulin, and a bone-marrow examination showed a few atypical blast cells, no excessive plasma cells, but was otherwise normal.

COMMENT

James (1960) estimated that when alcohol-induced pain is specifically asked or tested for in patients suffering from Hodgkin's disease it is found in 17% of cases, and it occurs more frequently in those under the age of 40 and in those patients whose nodes show an excess of eosinophils and fibrosis on biopsy.

Since its first description in 1950 alcohol-induced pain has been found in isolated instances in a wide variety of conditions. James *et al.* (1957) report its occurrence in a patient with carcinomatous invasion of the thymus and in a patient with pulmonary metastases from a presumably primary pancreatic carcinoma. Conn (1957), who tested for alcohol-induced pain in 98 patients, describes its occurrence associated with osteomyelitis, with traumatic fracture of the acetabulum, and in a patient with pyogenic lymphadenitis who also had sarcoidosis. In

addition alcohol-induced pain has been described in association with Brodie's abscess of bone (Alexander, 1953), eosinophilic granuloma of the skull (Lewes and Valentine, 1962), carcinoma of the cervix (Healey, 1959), as well as in a normal healthy physician who experienced this symptom for 15 years (Conn, 1957).

The symptoms can be produced by oral as well as by intravenous ethanol usually by both routes in the same patient, though sometimes only by one or the other. It has been reported in patients with and without bone involvement and it has been known to antedate clinical and radiological evidence of disease at the sites of pain. Quite minute quantities of alcohol such as a portion of sherry-containing jelly (Portman, 1950) can produce the symptoms and it has been reported with all forms of alcoholic beverage. Gros, Veillon, and Waeckel (1953) first noticed that alcohol-induced pain could be prevented by the administration of an antihistamine beforehand, and, as in the patient described here, Bichel and Bastrup-Madsen (1953) failed to reproduce the symptom with a vasodilator.

Though 14 years have elapsed since it was first described, there is still no explanation of the pathogenesis of the symptom. Most of the case descriptions have been of patients suffering from Hodgkin's disease, and as dilute alcohol is often used as a nutrient either in the form of a drink or intravenously in carcinomatosis the occurrence of the symptom in the latter condition must be very rare indeed.

I would like to thank Dr. A. St. J. Dixon for allowing me to report on this patient who was admitted to the Rheumatism Unit at St. Stephen's Hospital under his care, Mr. O. Tubbs for allowing me to follow-up and further investigate the patient at the Brompton Hospital, Professor D. W. Smithers for access to the relevant case notes at the Royal Marsden Hospital, and Dr. H. E. M. Kay for carrying out some of the investigations.

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Otogenic Tetanus: Report of a Case with Bacteriological Findings

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Tetanus associated with chronic ear infection has often been reported from India (Stonham, 1938; Shah, 1955; Hazra and Agnihotri, 1960; Apte, 1961), but only two cases in the British Isles are found reported in the literature to date (Corcoran, 1938; Wadsworth, 1962).

The following is a report of a case together with bacteriological findings.

CASE REPORT

A 49-year-old woman, who was employed in a factory manufacturing horse-hair upholstery filling, was admitted to hospital on 13 December 1963 with a three-day history of pain along the right side of the lower jaw and difficulty in opening her mouth. During the 24 hours before admission she had been able to take fluids only. She had no recollection of any recent puncture wound or abrasion, but did volunteer the information that she had had some blood-stained discharge from the right ear some three weeks previously, that both ears had felt "blocked up" for some time, and that there had been intermittent discharge from the left ear for several years. She had not attempted any more than superficial mopping of the ears.

Clinical examination revealed an apparently over-anxious little woman who was unable to open her jaws more than $\frac{1}{2}$ in. (1.3 cm.).

She was edentulous and without dentures, and it was rather difficult to be certain that there was true masseteric spasm. Her temperature was normal, but there was a polymorphonuclear leucocytosis (W.B.C. 12,600/c.mm.). There was no evidence of recent injury. Reflexes were brisk but symmetrical. A slight degree of neck stiffness was detected, but normal cerebrospinal fluid was obtained on lumbar puncture. Signs of mild pulmonary emphysema were noted.

Purulent and epithelial debris was mopped out of each external auditory meatus, specimens being taken for bacteriology. A granular area was seen in the floor of the inner third of the right external meatus, but the right tympanic membrane appeared intact and mobile. There was, however, a postero-superior marginal perforation of the left tympanic membrane with possible underlying cholesteatoma. The nose and oropharynx appeared healthy and there was no tenderness in the region of the ears, parotid glands, temporo-mandibular joints, mandible, or neck.

In the absence of any local cause for trismus a diagnosis of tetanus was entertained as a distinct possibility, with hysteria also being considered. A course of penicillin intramuscularly was begun and the patient was given a test dose of antitetanus serum, there being no history of previous administration of antiserum or tetanus toxoid.

Over the next 48 hours the trismus lessened, but the patient had some difficulty in swallowing and had some overspill into her respiratory tract. She did not maintain an adequate fluid intake, and so intravenous fluid and electrolyte replacement was started, a serum potassium level of 2.8 mEq/l. in particular requiring correction. Parenteral chlorpromazine was given to control her general restlessness.

On 16 December the presence of *Clostridium tetani* in both ear swabs was tentatively reported. Antitetanus serum 100,000 units was given intramuscularly without any side-effect. Early the next morning there was a major tetanic spasm. Later that day, though the patient was receiving 100 mg. of chlorpromazine three-hourly, a second tetanic spasm occurred while an attempt was being made to pass a gastric feeding-tube. It was accordingly decided to transfer the patient to the intensive-care unit. Under anaesthesia with thiopentone and suxamethonium, a feeding-tube was introduced and bronchial toilet, yielding copious thick secretions, carried out (Dr. Donald Campbell). As adequate spontaneous respiration was not maintained, the patient was curarized and intermittent positive-pressure respiration begun on a triggered Barnet ventilator. Tracheostomy was carried out and the patient was maintained fully curarized for a further 14 days on the ventilator. Some difficulty was experienced in maintaining good aeration of the left lower lobe and partial collapse occurred. Bronchial aspiration and vigorous physiotherapy were, however, successful in re-establishing satisfactory air entry. Apart from this the management of the patient was uneventful and she was completely weaned from the ventilator by 7 January 1964 without recurrence of tetanic spasm. The feeding-tube was removed on 8 January, as the patient was able to swallow well.

In the treatment of respiratory and urinary infection and in accordance with bacterial sensitivities, penicillin, streptomycin, colistin, nitrofurantoin, ampicillin, cloxacillin, and erythromycin were all used. The ears were mopped out with a 2.5% solution of Noxyflex (noxythiolin).

The right external otitis settled quickly, but slight serous discharge was noted on 23 December, and an antero-inferior perforation of the right tympanic membrane occurred. A little discharge could be mopped out of both ears until the patient was weaned from the ventilator, when the right ear became dry. X-ray films showed sclerosis of both mastoids.

The tracheostome healed without trouble. Pulmonary function tests (Dr. Donald Campbell) confirmed the presence of chronic obstructive lung disease. The patient was discharged to a convalescent home on 7 February.

She has been followed-up as an out-patient and has been given two injections of tetanus toxoid. Surgical exploration of the left ear has been strongly recommended to the patient, but she will not consent to this.

Since the initial swabs, repeated swabs from both ears have failed to yield *Cl. tetani*.

BACTERIOLOGICAL DATA

Swabs from the discharge from both ears were inoculated into anaerobically prepared Robertson's cooked meat broth, which was

then incubated at 37° C. Films made from the broth 48 hours later and stained by Gram's method showed frequent rather long pinkish-mauve staining bacilli. A few were of "drumstick" form with terminal spores, though this appearance was not very clear cut. Some bubbles of gas were noted in the broth cultures, but the meat was neither blackened nor digested. A tentative report of the presence of *Cl. tetani* in both ear swabs was then made.

Confirmation was obtained from animal inoculation and protection tests. Four mice were used. Two were injected intraperitoneally with 0.5 ml. of antitetanus serum. Half an hour later 1 ml. of the broth culture from each ear was injected intraperitoneally into a protected and an unprotected mouse. The mice were observed at intervals. After six hours one of the unprotected mice showed rhythmic contractions of the tail and the other showed paralysis of the hind legs. Both unprotected mice succumbed, but the mice protected by antitetanus serum remained apparently well.

Further studies were carried out on the broth cultures. Each was heated to 60° C. for one hour and then inoculated on to two blood agar plates. One of these was incubated anaerobically and the other aerobically. A fine fimbriated swarming growth was obtained on the anaerobic plate. Gram-stained films from this showed long pinkish-mauve staining bacilli, some of them of "drumstick" appearance.

COMMENT

Petzal (1930) points out that two forms of otogenic tetanus are found. In that resulting from trauma, including foreign-body trauma, the convulsions are acute, severe, and generalized. Where tetanus supervenes in existing suppurative ear disease, or perhaps becomes overt from a latent state, the clinical picture is much less severe. The case reported above falls into this second category. It is of particular interest in that *Cl. tetani* was cultured from both ears. While there was chronic suppurative otitis media in the left ear, the initial picture in the right ear was of an otitis externa. The perforation of the right tympanic membrane occurred only after the patient had been curarized for several days. Mopping of the ears with contaminated cotton-wool might be postulated as a cause of bilateral infection, but this patient denied mopping within the meatus. Her occupation, working with horse-hair, must be suspected as a relevant factor, though the ubiquitous nature of the organism would render this difficult to prove.

Apte (1961) in his reported series of 98 cases in India draws attention to the high mortality of tetanus complicating mastoid surgery and also reports a case of recurrence of otogenic tetanus. This stresses the need for active immunization in these patients, particularly when mastoid surgery is contemplated.

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