PARATYPHOID B OSTEOMYELITIS OF FRONTAL BONE

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Although paratyphoid osteomyelitis has been described repeatedly in the literature we could find no case in which the skull bones were involved. Cases of typhoid infection of the skull, however, have been recorded by Winslow (1923), and by Lever and Barker (1945). Typhoid osteomyelitis occurs in almost 1% of typhoid infections, while the incidence of this complication is much lower in cases of paratyphoid fever (Winslow, 1923; Keith and Keith, 1926; Rozansky, Ehrenfeld, and Matoth, 1948).

Our observation of paratyphoid B osteomyelitis of the frontal bone deals with a hitherto unreported localization of paratyphoid osteomyelitis and is added to the 23 cases of bone involvement collected by Rozansky, Ehrenfeld, and Matoth (1948), and the cases of Ecker, Kuehn, and Recroft (1942), Ancherson (1947), Abrams and Gaenslen (1948), and Ludwig (1949).

Case Report

A man aged 54 was seen at a surgical out-patient clinic on April 27, 1951, because of a soft swelling at the right side of his forehead. There was no fever, pain, or any other subjective complaints. As x-ray examination of the skull showed foci of bone destruction in the right frontal area, he was referred to the neurosurgical department. Thirty-three years previously he had been ill with a "typhoid-like intestinal infection," after which he had not been well for almost a year. Since then he had been complaining of a dull pain in both legs which had been diagnosed as a rheumatoid condition. Seven years before admission he had had a swelling on the forehead similar to the present one; it had ruptured after a blow on the frontal region; a few millilitres of pus was expelled and the abscess healed.

He was a fairly well nourished man. His temperature and pulse were normal. A small scar in the middle of the forehead pointed to the site of the previous abscess. At the right frontal area a soft fluctuating swelling, the size of a plum, was attached to the underlying bone as well as to the skin above. There was painless thickening of the lower third of the left tibia, the adjacent soft tissues being normal. General physical examination revealed no other abnormality. The sedimentation rate was 4 hours 10 minutes (Linzenmeier). A blood count showed: red cells, 5,140,000; haemoglobin, 80%; white cells, 9,600 (polymorphonuclear neutrophils 75%, eosinophils 1%, lymphocytes 16%, monocytes 8%). Wassermann and Kahn reactions in blood were negative.

Radiographs showed several areas of bone destruction in the right frontal region, with irregular borders without signs of bone reaction. X-ray examination of the chest showed thickening and increased density in the posterior part of the left seventh rib, and thickening of bone in the lower thirds of both tibiae, with scattered areas of rarefaction and increased radiolucency. The cerebrospinal fluid, which was clear and under normal pressure (110 mm.), contained 72 mg. of glucose per 100 ml., 12 mg. of protein per 100 ml., and no cells; Wassermann and Kahn reactions negative; gold-

sol test, 000000. Aspiration of the frontal swelling yielded greyish-yellow pus; no micro-organisms were found in direct smear or culture. Osteomyelitis of unknown origin, probably specific, with formation of subcutaneous abscess, was diagnosed.

On May 7, 1951, the diseased bone was freed by a curved incision at the right frontal region. A small amount of pus was found under the skin over an area of destruction of the outer lamina of the bone. The diseased bone was removed as a whole, and its borders were nibbled away with rongeurs until healthy bone tissue was encountered. On the inner lamina there were several holes of different sizes due to bone destruction. A fair amount of pus was found in the epidural space and evacuated. The dura mater was thickened with adherent granulation tissue, which was scraped off. Nowhere was this membrane destroyed. After removal of all affected bone a defect of 6 by 8 cm. resulted. Galea and skin were sutured individually. The operative wound healed by first intention, and the patient made an uneventful recovery, leaving hospital on the sixth post-operative day. He has been well since.

Microscopical examination of the removed bone showed chronic and acute osteomyelitis. Gram-negative bacilli were found in a direct smear of the pus evacuated during operation. By culture and biochemical reactions they were identified as *Salmonella typhi* B. Agglutination tests performed with the patient's serum gave the following results: *Salm. typhi* 1:100±; *Salm. paratyphi* B 1:100±. Agglutination of the strain cultured from the patient's pus by the patient's serum 1:200++. Repeated cultures of the urine, stools, and duodenal juice were negative for salmonella organisms.

Comment.—This patient had recurrent osteomyelitis of the frontal bone 26 and 33 years after an enteric infection. Although no bacteriological and clinical data of this are available, there is no doubt that the micro-organisms cultured from the osteomyelitic pus—*Salm. paratyphi* B—were of the same type responsible for the primary disease.

We must further assume that the x-ray changes found in both tibiae and in one rib were also due to an invasion by the same micro-organism.

Discussion

Paratyphoid bacilli are known to stay in the bone marrow long after they have disappeared from blood, urine, and faeces (Ling *et al.*, 1940; Macdonald, 1941). Obviously they also can remain in the diploe of the skull.

Several cases have been reported in which the bone infection became manifest many years after the original intestinal disease (Jensen and Kock, 1913, 20 years; Ecker, Kuehn, and Recroft, 1942, 24 years; Ancherson, 1947, 18 years; Rozansky, Ehrenfeld, and Matoth, 1948, 32 years). Most authors agree that trauma, physical strain, or an intercurrent disease may lead to activation of the clinically latent micro-organisms and produce the condition of acute osteitis and osteomyelitis. No such factors could be elicited in our own case.

Summary

A case of bacteriologically verified paratyphoid B osteomyelitis of the frontal bone is presented. Operation brought about complete recovery. Other bone lesions in the same patient (both tibiae, and one rib) are thought to be of the same origin. The frontal-bone lesion first appeared 26 years after the acute enteric infection, and recurred seven years later.

Incidence of bone complications in paratyphoid infection is rare, and this is the first case to be recorded in which the skull was involved.

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APPARATUS FOR DEMONSTRATING HAZARDS OF STATIC ELECTRICITY IN OPERATING THEATRES

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We were fortunate enough to be able to witness the remarkably effective demonstration of the explosive properties of mixtures of cyclopropane and oxygen given by Dr. H. J. V. Morton at the Royal Society of Medicine on December 1, 1950 (*Lancet*, 1950; *British Medical Journal*, 1951). The apparatus described here was devised as a result of this stimulus. It gives a startling demonstration of the ease with which static electricity may be generated and caused to produce an explosion with ordinary equipment used in operating theatres. The apparatus reproduces the condition that could arise in an explosive atmosphere when two bulky items, either equipment or personnel, are brought together or near enough to each other to produce a spark. Some such apparatus may possibly have been used already in laboratory studies (cf. Bulgin *et al.*, 1949), but, if so, it does not appear to have been described in the literature.

One form of the apparatus is shown in Fig. 1, and a photograph of an actual explosion involving cyclopropane and oxygen is shown in Fig. 2.

Description of Apparatus

The apparatus consists essentially of two electrodes sealed by non-conductive material inside a "cellophane" chamber. The points of the electrodes are set about $\frac{1}{4}$ in. (3 mm.)

FIG. 1.—One form of the explosion chamber. FIG. 2.—Photograph of an explosion involving cyclopropane and oxygen, using another form of the explosion chamber.

apart, and they project above a circular "perspex" block 2½ in. (5.7 cm.) in diameter by ½ in. (1.3 cm.) thick, sealed on the perspex base. To fill the chamber with gas an entry port is provided from the outer edge of the main base to the inlet port in the centre of the block. A bronze ring is made to fit firmly on to the block, which is slightly tapered to provide a firm seating. The cellophane explosion chamber is prepared by cutting out a circle about 5 in. (12.7 cm.) in diameter from a sheet of thin cellophane, and, after forming it into a roughly hemispherical shape, placing it over the edges of the block. The bronze ring is now pressed down firmly, trapping the cellophane between it and the tapered edge of the circular block, producing a gas-tight joint. A flexible explosion chamber is thus produced with a rupture pressure of 100 to 150 lb. per sq. in. (10.54 kg. per cm.²), which is also transparent and permits the spark to be observed in the event of a non-explosive mixture being present within the chamber. The flexibility of the cellophane allows the volumes of gases admitted to be varied within wide limits, and they can be estimated accurately enough for demonstration purposes.

A still simpler apparatus may be constructed from 2-in. (5-cm.) ebonite rod through which three holes are drilled, and the cellophane bag secured by adhesive tape.

Method of Demonstration

To introduce the explosive mixture the cellophane is first compressed over the electrodes to expel the air. The bag is then inflated by applying the outlet valve of a cyclopropane cylinder to the gas entry port of the apparatus. Acetylene, coal gas, or other inflammable gas may also be used. The cellophane bag is now half compressed, expelling half the cyclopropane. Oxygen is then used to fill the chamber to capacity. It now contains a 50% mixture of gas and oxygen. It is sealed by means of the screw plug provided for the gas entry port. The chamber retains the gas mixture for several minutes before leakage takes place.

An ether-oxygen mixture is readily introduced by passing oxygen through or over the ether in the bottle of an anaesthetic machine and thence to the gas entry port. A pre-formed gas mixture may also be introduced if desired.

One of the electrodes is now connected to earth and the other to an anaesthetist's trolley. Before making this connexion it is essential to neutralize any charges which may be present on the trolley by connecting it momentarily to the earth lead and thus eliminating any risk of pre-ignition. The safe minimum distance a person may be from the explosion is 10 ft. (3 m.) in a room or chamber of not less than 10,000 cu. ft. (283 m.³). Care should be taken to observe this point when demonstrating. Light-gauge fuse wire is suitable for the leads. They are clearly shown in Fig. 2.

The trolley should stand on a non-conductive floor, preferably in a warm dry room, and should be provided with a rubber sheet and a dry blanket. On removal of the blanket a loud explosion takes place. The flash is orange in tint with cyclopropane and white with acetylene, ether having an intermediate colour. The explosion is due to the static charge induced in the trolley by the movement of the blanket on the rubber sheet. Measurements taken with an electrometer show the potential to vary from 10,000 volts to 15,000 volts when the blanket is removed by slipping it sideways on the surface of the rubber. Lifting the blanket vertically induces a smaller potential of 2,500 volts. The minimum potential so far recorded as producing an explosion with this particular apparatus is 2,700 volts.

To prevent loss of charge between the point of induction and the discharge electrodes, no earthed body or any mass which would serve as a capacitor should be near the conductors on the live side of the chamber. Care should also be taken to prevent tracking of the current in the explosion chamber itself. This can well be caused by a film of moisture left by a previous explosion or residing on the surface of the perspex. This can largely be pre-