which may invaginate the bowel wall and become a compound intussusception.

It is, however, most unlikely that the lumen of the bowel, in the case of an ileo-ileal intussusception, is sufficiently large to permit the passage of such a large mass to pass inside it in a prograde direction. It is much more likely that the proximal bowel is pushed over the mass by peristaltic action. This is borne out by the frequency of compound intussusceptions consisting of a retrograde and a prograde intussusception reported in the literature. Retrograde intussusception is a misleading term, since it suggests that the intussusceptum is driven in an opposite direction to peristaltic action. I suggest that the intussusceptum remains stationary and the proximal bowel slides over the mass driven forwards by peristalsis.

There remains the problem of why a double retrograde intussusception was formed in this case. The Meckel's diverticulum was rather longer than usual, and was adherent to the bowel wall, the mouth of the diverticulum lying proximally. This had then formed a mass which was incapable of invaginating the bowel wall and passing along the lumen in a prograde or downward direction. The proximal bowel was then pushed over the mass (Fig. 1) leading to a single retrograde intussusception containing a Meckel's diverticulum (Fig. 2). This state of affairs probably existed following the attack of intestinal obstruction a year before admission. Adhesions then formed between the diverticulum, now an integral part of the intussusceptum, and the intussuscipiens. Immediately before admission a second intussusception began to form (Fig. 3), leading to intestinal obstruction and the appearance of the resected specimen (Fig. 4).

I would like to thank Mr. R. V. Fiddian for permission to publish details of this patient's history, the patient being admitted under his care. I would also like to thank Mr. F. E. Weale for helpful advice and criticism, and Peter Cull, medical artist to St. Bartholomew's Hospital, for the illustrations.

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Rat-bite Fever Due to *Streptobacillus moniliformis*

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Rat-bite fever can be caused by two different organisms, *Streptobacillus moniliformis* and *Spirillum minus*. Both organisms are transmitted to man by the bite of an infected rat or other rodent. It may also be acquired from contaminated food or milk.

Before the introduction of antibiotics the illness was protracted, and a case has been described lasting 17 years (Surveyor, 1913). It is a rare disease in Great Britain, most published reports coming from North America. This paper describes two cases of rat-bite fever due to *Streptobacillus moniliformis* treated in this hospital within a period of eight months.

CASE 1

A 67-year-old retired miner was bitten on the right hand by a rat on 6 October 1964. The wound was washed thoroughly and it healed rapidly over the course of a few days. Twenty-four hours after this incident he developed rigors and he thought that he had influenza. The rigors occurred intermittently for two days, and on 9 October he developed pain and limitation of movement of his right knee and thigh. The frequent sweating and shivering attacks persisted throughout the next week. His family doctor treated him symptomatically as a case of influenza. No antibiotics were given. The patient had forgotten completely about the rat-bite incident, but towards the end of the week he volunteered the information, and was admitted to the Northern Hospital, Dunfermline, on 23 October.

Examination on admission revealed an ill-looking man, lying immobile in bed, and obviously experiencing severe pain in his left knee and thigh, both of which were flexed and extremely tender to touch. There was no discoloration or effusion in his knee. He was apyrexial, and there was no evidence of a rash, jaundice, lymphadenopathy, hepatomegaly, or splenomegaly. On closer questioning he complained of dysuria and had low backache, and for the past few days he had noticed that his urine was dark in colour. Urinalysis revealed albuminuria, microscopic haematuria, and sterile pyuria.

The results of investigations carried out on admission were as follows. Haemoglobin 12.3 g./100 ml., white blood count 9,300/c.mm. (neutrophils 54%, lymphocytes 40%), E.S.R. 120 mm. in one hour (Westergren). Serum protein, 6 g./100 ml. (albumin 2 g., globulin 4 g.). Serum bilirubin 0.4 mg./100 ml., alkaline phosphatase 13 K.-A. units. Thymol turbidity 1 unit. Thymol flocculation nil. S.G.P.T. 15 international units. Blood cultures on admission sterile. Leptospiral agglutinations and guinea-pig inoculation negative. W.R. negative. Radiographs of the lumbosacral spine and left knee showed no abnormality. That of the chest was also negative.

On 25 October his temperature was 99° F. (37.2° C.) and the following day it rose to 101° F. (38.3° C.). Further blood cultures were taken, and from these *Streptobacillus moniliformis* was isolated on 2 November.

On 29 October the pain and tenderness in his left knee and thigh had disappeared completely but the next morning he had severe limitation of movement and pain in his right shoulder and an obvious effusion was present. A radiograph of this joint was normal. On 2 November E.S.R. was 129 mm. in one hour, white blood count 10,000/c.mm. (neutrophils 77%, lymphocytes 17%). His haemoglobin had fallen to 11.3 g./100 ml. and bromsulphalein test showed 24% retention of dye after 30 minutes. Serum fibrinogen was 1,360 mg./100 ml. Serum protein electrophoresis: albumin 36%, α_1-globulin 10.5%, α_2-globulin 18%, β-globulin 15%, γ-globulin 20%.

On 2 November, when *Streptobacillus moniliformis* had been isolated, the patient was given streptomycin, 1 g. twice daily, to be followed four days later by the addition of benzyl penicillin, 500,000 units six-hourly. The institution of this latter therapy was delayed because the patient was suspected of being allergic to penicillin. The temperature remained raised over the course of the next two weeks. His haemoglobin continued to fall, and on 11 November it was 9.7 g./100 ml.; 2 litres of blood were transfused. With these measures the patient progressively recovered, and, apart from some discomfort in his right shoulder, became asymptomatic. His E.S.R. remained high, and when he was discharged on 12 December it was still 66 mm. in one hour.

At follow-up on 25 January 1965 he still had considerable limitation of movement of his right shoulder, but no pain. His E.S.R. was 17 mm. in one hour, liver-function tests were normal, and haemoglobin was 13.2 g./100 ml.

CASE 2

A 67-year-old farmer was bitten on his right hand by a rat on 10 May 1965. The wound was immediately cleaned with an antiseptic lotion and it healed in a few days. He remained well until 24 May, when he developed anorexia and headache with sweating and shivering attacks. He thought he was suffering from influenza. Two days later, on the 26th, he developed severe aching muscular discomfort in both shoulders, radiating down his arms, involving his elbows and wrists. His left knee became painful and tender to touch but there was no effusion or discoloration. He was admitted to the Northern Hospital, Dunfermline, on 3 June.

On admission he was an ill-looking man, pyrexial (100° F.; 37.8° C.), lying immobile in bed with obvious large effusions of both shoulder joints and both knees. There was no evidence of lymphadenopathy, hepatosplenomegaly, or rash. Specific inquiry with respect to rat bite was made, and for the first time the story of having been bitten was obtained.

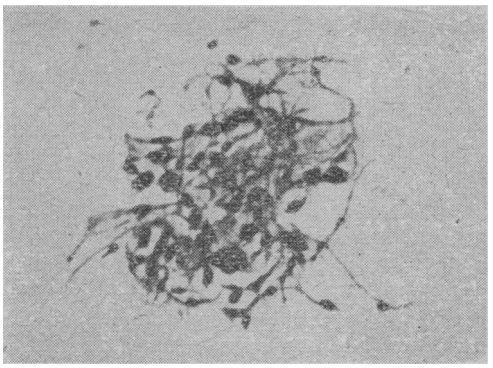
The results of investigations carried out on admission were as follows. Haemoglobin 15.3 g./100 ml., white blood count 13,000/c.mm. (neutrophils 71%, lymphocytes 23%), E.S.R. 70 mm. in one hour (Westergren). Alkaline phosphatase 22.5 K.-A. units. Serum bilirubin 1.4 mg./100 ml. Thymol turbidity 1 unit. Zinc sulphate turbidity 2.5 units. Serum protein 5.8 g./100 ml. (albumin 2.9 g., globulin 2.9 g.). Serum protein electrophoresis: albumin 50%, α_1-globulin 5%, α_2-globulin 19%, β-globulin 12%, γ-globulin 15%. Bromsulphalein excretion 15% retention of dye after 30 minutes. Blood urea 48 mg./100 ml. Urine—no albuminuria on admission, but sterile pyuria and microscopic haematuria present. Leptospiral *Salmonella* and *Brucella* agglutinations negative. Paul-Bunnell test negative. A.S.O. titre normal. W.R. negative. Radiograph of the chest negative.

On 6 June he developed pain and tenderness in the small joints of his hands, and a marked erythematous rash with several pustules appeared over his legs and arms. From blood cultures *Streptobacillus moniliformis* was isolated, and streptomycin, 0.5 g. twice daily, and benzylpenicillin, 500,000 units six-hourly, were started on 8 June. It was noted at this time that despite his pyrexia his pulse rate remained in the region of 70 to 80/min., sinus rhythm. An E.C.G. showed the presence of right bundle branch block. There was no evidence of cardiac failure, pericardial friction rub, or cardiac murmurs. S.G.O.T. was 17 international units, serum X-hydroxy-butyrate dehydrogenase 70 international units, E.S.R. 112 mm. in one hour, serum fibrinogen 970 mg./100 ml. Haemoglobin 14.6 g./100 ml. Blood urea 59 mg./100 ml. (despite adequate hydration). Over the next few days he improved rapidly and the effusions subsided within 72 hours of starting antibiotic therapy. By 17 June his E.S.R. was 5 mm. in one hour and he was apyrexial. He still experienced slight stiffness in the affected joints and despite lack of pain was unable to abduct his arms.

At follow-up on 13 July 1965 he was well but still had considerable limitation of abduction of both arms. Haemoglobin was 14.6 g./100 ml., and liver-function tests were within normal limits.

BACTERIOLOGICAL TECHNIQUE

Double-phase blood culture media were used, consisting of a chocolate agar slope with 10 ml. liquid broth, and 5 ml. of patient's blood added to each bottle. Growth of the organisms was detected by the appearance of small transparent colonies on the agar slopes, which had been inoculated at intervals by tipping up the bottles. The colonies consisted of Gram-negative filaments in tangled masses and chains, with numerous moniliform swellings in the filaments (see Fig.). This appearance is characteristic of *Streptobacillus moniliformis*. Growth was obtained both under aerobic and anaerobic conditions and was maintained in subculture on serum or chocolate agar. The strain from Case 1 was sensitive to chloramphenicol, penicillin, streptomycin, and tetracycline, but resistant to erythromycin. Case 2's strain was sensitive to all five antibiotics.



Characteristic appearance of *Streptobacillus moniliformis* showing clump of filaments with moniliform swelling. (CO_2 blood culture slope. Gram. ×1,000.)

COMMENT

Both cases developed severe painful polyarthritis; rapid relief of pain occurred after antibiotic therapy, but, despite this, a degree of limitation of movement remained in the affected joints. This may be due to a form of adhesive capsulitis or peritendinitis secondary to effusion. Myositis has been described as a rare occurrence (Beaumont and Gill, 1935; Swyer, 1945) and both cases described here demonstrated a degree of myositis.

Other complications described are endocarditis (Pappenheimer and Satchwell, 1907), splenic and renal infarction (Blake, 1916), and anaemia (Kane, 1944). Reviewing the literature there is no record of hepatic involvement occurring in this disease. Both our cases showed evidence of temporary parenchymatous liver dysfunction.

Immediate wound toilet does not help to prevent infection (Lominski *et al.*, 1948). The onset of rat-bite fever is similar to influenza, and the early use of antibiotics may prevent development of the full clinical picture and cure the illness before a diagnosis of strepto-bacillary fever is made. Perhaps the condition is more prevalent than is realized.

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