at the Mayo Clinic. Yet Kleckner *et al.* (1952) subsequently reported cirrhosis in 18.7% of biopsy material from this same clinic.

Our series is comparable in size to those previously reported, the largest of which is composed of 93 cases. The average duration of symptoms, too, was not significantly shorter in our patients. We consider, therefore, that our figures lend strong support to the view that the incidence of cirrhosis in ulcerative colitis is not materially greater than its incidence in the population as a whole. Many of the reported cases are probably examples of post-hepatic cirrhosis, as the clinical histories and necropsy findings not infrequently suggest.

Summary

The liver lesions in 73 cases of ulcerative colitis coming to necropsy in the Bernhard Baron Institute of the London Hospital in the years 1914 to 1953 have been studied.

Severe fatty change was present in 9 of the 39 cases examined microscopically; in one of these, however, it was attributable to exogenous poisoning. Apart from recent pregnancy in three cases, and possibly anorexia and excessive vomiting in others, no clinical factors predisposing to the lesion were found.

Inflammatory cell infiltration was minimal, and, except in one case with biliary obstruction, cirrhosis was not present. It is concluded that there is little if any increased liability to the development of cirrhosis in patients with ulcerative colitis as compared with the population as a whole.

We wish to thank Professor Dorothy S. Russell and Dr. W. W. Bridgen for their helpful criticism.

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Medical Memoranda

Sensitivity to Penicillin Causing an Acute Abdominal Emergency

Reactions to penicillin occur in 1.5 to 5% of cases (Keefer and Anderson, 1950). Many are trivial, the most frequent being an urticarial rash. Other reactions described include arthralgia, lymphadenopathy, fever, and oedema (Humphreys, 1951; Wilkes, 1951). More recently fatal anaphylactoid reactions have been reported (*British Medical Journal*, 1951; Thompson, 1952; Pick and Patterson, 1953; and Mayer, 1953), and there has been an increasing correspondence about near-fatal anaphylactoid reactions.

The following case is reported, in which, after an injection of crystalline penicillin, a young woman developed severe abdominal pain and, later, urticaria, oedema, arthralgia, and lymphadenopathy.

CASE REPORT

A married woman aged 28, in the second day of the puerperium after a normal delivery, developed a small

whitlow. She was given a parenteral injection of 300,000 units of crystalline penicillin. There was no immediate reaction, but the next morning she complained of severe pain at the site of injection; the area was found to be red and swollen. It was then discovered that when she had received penicillin in 1948 her eyes and tongue had become swollen and large weals had arisen on her arms and legs.

Four hours later, 24 hours after the penicillin injection, she developed severe generalized abdominal pain, and her temperature rose to 100° F. (37.8° C.). Shortly afterwards intractable vomiting started. Tenderness and rigidity were generalized, but apparently were more marked in the right iliac fossa. A tentative diagnosis of acute appendicitis was made.

At laparotomy a normal appendix was found, but some 6 in. (15 cm.) of small bowel presented which was dark purple in colour, fading into normal-looking bowel at either side. The related omentum was swollen and haemorrhagic, and some turbid fluid was present in the peritoneal cavity. As the affected bowel appeared to be rapidly becoming necrotic, and the process seemed to be extending alarmingly, a resection was performed, including several inches of normal bowel at each side. An end-to-end anastomosis was made.

The post-operative period was stormy. Gross oedema of the periorbital tissues, face, hands, and feet developed almost immediately. Elbow, knee, wrist, and ankle joints became swollen and acutely painful. The tongue became swollen, and a generalized urticaria appeared; cervical and axillary lymph nodes became palpable, also the lymph nodes draining the site of the original penicillin injection. Under treatment with antihistamine drugs, adrenaline, and intravenous glucose-saline, the oedema slowly subsided. Four days later the joints became painless, and from that time she made a rapid recovery.

Approximately 19 cm. of small bowel, with frank purpuric haemorrhage into the mesentery and local subserous spread over the bowel, was removed. The muscularis and mucosa appeared normal. Histological examination confirmed the capillary haemorrhage into the mesentery and subserous layer of the bowel. In addition, there was acute congestion of the submucosa layer, with oedema of all layers, including the muscle.

COMMENT

Both the naked-eye and microscopical appearances of the resected portion of the bowel were those of extensive capillary haemorrhage into the mesentery and subserosa. These findings are similar to those seen in Henoch-Schönlein purpura—one of the anaphylactoid purpuras. These appearances, combined with oedema, arthralgia, and urticaria, make it certain that the purpura in this case was due to penicillin sensitivity.

Anaphylactoid purpura has already been reported in a patient receiving penicillin (Anderson, 1947). In the present case no skin purpura was seen before or after the acute abdominal episode, the reaction being confined to a small area of intestine.

In retrospect, resection would seem to have been the correct treatment in this case, despite the normal appearance of the mucosa. As in other intestinal purpuras, necrosis may well have occurred, and it appeared imminent at laparotomy.

I am grateful to Mr. L. A. Cruttenden for permission to publish this case, and to Dr. R. T. Cooke for providing an opportunity of studying the specimen of bowel.

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