Morbid Anatomy and Histology.—As already mentioned, the naked-eye appearances of the tumour were those of a subserous haematoma. On opening the caecum several small worms were noted lying free in the lumen; these were afterwards identified as whip-worms (*Trichocephalus dispar*). Pieces of tissue were taken for microscopical examination from the centre of the tumour mass, and, after embedding and section, stained with haematoxylin and eosin. The sections of the appendix and intestine showed acute and chronic inflammation, oedema, and haemorrhage. The bleeding seemed to have infiltrated both muscular layers of the intestines and was also seen in the sub-peritoneal part of the appendix. The exact site of origin of the haemorrhage cannot be determined, and there was no evidence of tumour formation or of any specific granuloma in the sections examined (J. T. Quinlan).

Commentary

The condition described appears to be unusual, as a search of the literature failed to reveal an account of any similar case. The predominant feature was the large circumscribed haemorrhage in the wall of the caecum. Except for the inflamed appendix there were no other signs of infection, and no evidence of spread of infection along the base of the appendix to the caecum. Following upon the operation, investigations were made of the blood count, the bleeding, and the clotting time, but neither these nor other clinical manifestations gave any evidence of an underlying haemorrhagic diathesis. Moreover, the whip-worms, which were lying free in the lumen of the caecum, were clearly playing no part in the aetiology. These are common enough inhabitants of that organ. Three possibilities offer themselves as likely explanations, and these may be considered in turn.

Trauma.—There was no history of a blow on the abdomen or similar violent injury as one would have expected had the haematoma been caused by direct violence. Further, owing to the mobility of the caecum a lesser degree of injury, unnoticed by the patient, could scarcely have caused the haemorrhage.

Torsion of Caecum with Venous Congestion.—Embolism or thrombosis of the ileo-caecal artery could not be demonstrated at operation, and the naked-eye appearances did not resemble those of the case reported by Harris. If infarction was the cause it must have been in the terminal branch of the posterior division of the ileo-caecal artery. It is possible, however, that the acute inflammation of the appendix may have been associated pre-operatively with an incomplete torsion of the caecum, leading to venous congestion, capillary rupture, and haematoma formation, the condition of torsion then reducing itself before the abdomen was opened.

Infection.—The absence of any signs of an infective lesion involving either the caecum or the ileum has already been mentioned. I am aware, however, that haemorrhage into the bowel wall may occur in terminal ileitis, or "Crohn's disease," and it is possible that the findings in the above case might have some bearing on the causal factors involved in that condition. Jackman has cited instances of intramural haemorrhage in the disease. In these the terminal ileum was involved, and

the haemorrhages were invariably small, scattered diffusely in the submucous and interstitial layers of the bowel wall, and distributed over a considerable length of gut, their cause and spread apparently taking the lines of least resistance. Regional and terminal ileitis may also involve the caecum, and some authorities advocate removal of the latter along with the terminal ileum. The preponderance of opinion favours conservative measures. The caecum may become so thickened and fibrosed, however, that mechanical obstruction results, making excision necessary. Had the serous coat of the bowel not ruptured and no operation been performed in the above case the haemorrhage might gradually have been absorbed and been replaced by fibrous tissue, the resulting appearance being similar to that of long-standing ileitis. Although in regional ileitis the appearance suggests a chronic infection and the distribution and size of the haemorrhages differ from those seen in the present case, no causal organisms have been found, and it is possible that the infective element may be secondary, the primary lesion arising in some such way as in this case, through mechanical causes such as incomplete torsion of the bowel and haematoma formation.

Summary

An unusual case of haematoma of the caecum, arising without trauma and probably due to incomplete torsion of the bowel, with venous congestion, is described. The possibility of such an accident giving rise to the changes seen in terminal ileitis is discussed.

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CHRONIC LEG ULCER IN MYELOGENOUS RETICULOSIS

REPORT OF A CASE

BY

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The coincidence of chronic ulceration of the leg and splenomegaly was discussed by Witts (*Journal*, 1942, **2**, 309) when he described two instances of chronic leg ulcer associated with purpura haemorrhagica. From his report it appears that chronic leg ulcer has not been described in association with myelogenous reticulosis. Furthermore, Witts said that "mere size of the spleen is not significant, inasmuch as ulceration is absent in the gross splenomegaly of leukaemia, and present with much less enlargement." An instance of chronic leg ulcer and considerable splenomegaly in a patient suffering from myelogenous reticulosis is therefore here put on record.

Case History

The patient was a woman aged 72 who had been "off colour" for three months, and had had a purpuric eruption beneath the skin of the legs and upper part of the chest. She also complained of painful swellings of the joints of the hands, loss of weight, poor appetite, and severe constipation. When examined in Oct., 1940, petechial haemorrhages were numerous beneath the skin of the arms, hips, left shoulder, and back; some of the haemorrhages were ecchymoses. Splenomegaly was detected, the lower pole of the spleen being about two fingerbreadths below the left costal margin; there was slight enlargement of the liver.

A series of seven blood examinations made during Oct. and Nov., 1940, showed that the haemoglobin percentage lay between 60 and 70; the red cells averaged three millions per

c.mm.; the colour index was 0.87; and the mean diameter of the red cells was $7\ \mu$. The leucocytes at no time exceeded 8,400 per c.mm., and on one occasion were as few as 3,000, 48% of the cells being neutrophil polymorphs and 52% lymphocytes; no abnormal white cells were seen. The Rehfuß test meal demonstrated free acid in the gastric juice, with a maximum level of 40 units.

The patient left the hospital on Nov. 18, 1940, and was not seen again until Dec. 16, 1941, when she was readmitted on account of ulceration, thought malignant, on the inner side of the leg. It was of uncertain duration, but was probably several weeks old. She was now pale and anaemic. The lower pole of the spleen was felt about 5 in. below the left costal margin, and its anterior border was almost in the abdominal midline. There was a purpuric eruption on the limbs and the anterior wall of the chest. An oval ulcer, $2\frac{1}{2}$ by $2\frac{1}{2}$ in., was present on the inner aspect of the middle third of the left leg. It had a circumvallate appearance, particularly in the lower half of the ulcer (see Fig.). The margin was regular around

the greater part of the ulcer's periphery; it was approximately $1\frac{1}{10}$ in. broad, raised by $1\frac{1}{10}$ in., and covered by thinned epidermis; there was no appreciable induration of the margin. The ulcerated area was occupied by dark-red vascular tissue, which bled readily when handled. This tissue was somewhat nodular but not indurated; it was covered in most areas by a thin layer of opaque greyish slough. The initial impression of malignancy was countered, on consideration, by the peculiar margin, its regularity, and the general lack of induration. Moreover, radiological examination excluded bone involvement. Biopsy was undertaken, and is responsible for the dark irregular areas at the left centre and left upper portion of the ulcer margin, shown in the photograph. Squamous carcinoma was at once excluded. The outstanding change was a massive infiltration of the ulcer and the adjacent corium by mature and immature white cells of the granular series. The appearances were those of a myelogenous deposit. Blood examination confirmed the presence of myelogenous reticulosis and severe secondary anaemia. The patient now had 43,000 white cells per c.mm., of which 64% were polymorphs, 16% myelocytes, 11% myeloblasts, 5% smear cells, and 4% lymphocytes. Normoblasts were present in the ratio of 24 per 300 nucleated blood cells. No platelet count was made.

The patient was gravely ill when admitted, and died a fortnight later. Treatment was without influence on her general condition; the ulcer remained almost stationary during this final period of observation.

Post-mortem Examination.—In addition to the ulcer of the leg described above, the other pathological changes at necropsy on Dec. 31, 1942, were as follows: Spleen 1,350 g., 9 in. from pole to pole, maximum thickness 3 in.; the pulp was somewhat soft and greyish red in colour, and the Malpighian bodies were obscure. The kidneys were both pale, except that the middle third of the right was destroyed by a congested infarct. Small, friable calculi, the largest $1\frac{1}{4}$ in. in diameter, were present in both kidneys, the largest lying over the pelvic orifice of the

right ureter. There was a solitary cholesterol stone, and the gall-bladder showed slight diffuse cholesterosis. No abnormality of note was found elsewhere; the brain and bone marrow were not examined. Histological examination of the tissues demonstrated the usual appearances of myelogenous reticulosis in the spleen, the liver—where the changes were slight—in the skin, and the kidney, where they were accompanied by acute infarction.

Comment

At the time of her readmission to hospital the ulcer was established and she was unable to give a satisfactory account of its earlier phases; its genesis is therefore a matter of speculation. It may have originated in a subcutaneous haemorrhage. There was no evidence of old haemorrhage in the biopsy specimen, and, although slight recent haemorrhage, possibly caused by biopsy, was present in the corium, the abundance of white cells, and their arrangement, are not ascribable to haemorrhage alone. The interpretation preferred is that the ulcer was a result of devitalization of the skin by a subcutaneous myelogenous deposit.

I thank Dr. John Dick, medical superintendent of St. James's Hospital, Leeds, for permission to quote from the clinical records.

Medical Memoranda

A Filtration Method of Sterilizing Sulphanilamide Powder

After experiments with several methods of sterilization the following technique has been evolved. It is simple in operation and quickly carried out.

METHOD

Sterilize by dry heat at 140°C . for one hour an 8-oz. insufflator, the tubes of which have their outer ends plugged with cotton-wool. The jar must contain about a dozen heavy glass beads or marbles. A Seitz positive-pressure filter of 100 c.cm. capacity and fitted with an E.K. disk is sterilized in the autoclave at 15 lb. for 30 minutes.

The filter is washed out with about 20 c.cm. of acetone by pumping it through the disk. This is to remove any water of condensation. Dissolve 20 g. of sulphanilamide in 100 c.cm. of acetone and pour the solution into the filter. Pump this through direct into the sterile insufflator, the cap of which is then immediately replaced and securely clamped down. To the longer tube of the insufflator is connected a short length of rubber tubing, which is closed off with a Hoffman's screw clamp, without removing the wool plug. The outlet tube is then connected to a water-vacuum pump and the acetone evaporated under negative pressure. A slow current of carbon dioxide is passed over the solution during evaporation (which may be accelerated by standing in a water bath at 80°C). When the contents have assumed the consistency of a thick paste the jar is agitated frequently in order that the beads may prevent the formation of large clumps of crystals. On drying, disconnect the pump. By further agitating the jar by a circular motion for 10 minutes or so the crystals will be broken down to a fine powder. To remove the last traces of acetone reconnect to the pump and alternately evacuate and release the vacuum by opening the clamp at intervals of 10 minutes for about an hour, shaking the jar occasionally. The powder will now be in a finely divided state, dry, and free-running, ready for use.

Samples of the powder obtained were subjected to sterility tests as follows: 0.5 g. of powder was introduced into each of three tubes of nutrient broth and three tubes of Robertson's cooked meat medium. The tubes remained sterile after 7 days' incubation. Further samples were sent for chemical analysis, which proved that the final powder showed no evidence of toxic oxidation products. We claim that this method is simpler than any other as yet described. It can be economically and practically carried out either on a commercial scale or in small cottage hospitals.

We wish to express our indebtedness to Mr. R. G. Minor, Ph.C., F.I.C., City Analyst's Laboratory, Cardiff, for his willing assistance.

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