(1953); here, however, the fibrosis was more pronounced and there was no leukaemic phase comparable to that seen in the present instance.

We have been unable to find a record of any similar case treated with steroids. Gray and Taylor (1953) have, however, reported a patient with an acute systemic reticuloendotheliosis who developed a frank monocytic leukaemia while on A.C.T.H. therapy. That steroid therapy might be harmful in at least some cases of acute myeloblastic leukaemia, in contrast to the remarkable though temporary remissions often obtained in lymphoblastic leukaemia, has been suggested by several authors (Rosenthal *et al.*, 1951; Fessas *et al.*, 1954; Björkman, 1956). Very recently Joseph and Levin (1956) have described a very interesting case of monocytic leukaemia in which cortisone made the leukaemia worse. One of us (R.B.T.) has further unpublished evidence pointing to the same conclusion. On the other hand, steroid therapy is recommended for both lymphoblastic and myeloblastic leukaemias by Scott (1955), and by Hayhoe and Whitby (1955), although it is clear that the responses in myeloblastic leukaemia are poorer. It seems likely, therefore, that not all myeloblastic cases show acceleration of the leukaemic process under the influence of steroids. At present there is no method of distinguishing one type of case from the other except by clinical trial of steroids. It is, admittedly, often difficult to distinguish acute lymphoblastic from acute granulocytic leukaemia. With care, however, such a distinction is usually possible with the exception of occasional very acute cases showing no evidence of differentiation. In these a diagnosis of stem-cell leukaemia must be made; most of them seem to react as lymphoblastic leukaemia. In view of the disastrous effect of steroids in the case described above it does seem important to make as accurate a diagnosis of the type of leukaemia as possible and to bear in mind the possibility that an adverse effect may be obtained in the myeloblastic type.

Summary

An unusual case of aleukaemic myeloid leukaemia with fibroblastic proliferation in the marrow is presented which, on treatment with cortisone, became frankly leukaemic and ended fatally in spite of a haematological remission with 6-mercaptopurine therapy.

Acknowledgments are due to the Departments of Pathology and of Photography, King's College, Newcastle upon Tyne, for sections and photographs respectively.

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The Central Council for Health Education has published a report of a conference held on January 24 at B.M.A. House on the "Education of the Public regarding Cancer." The report (price 1s.) includes sections on "Prevention and Treatment of Cancer," "Problems of Cancer Education," and "Cancer Education in Practice." Other recent publications of the Central Council for Health Education include "Look Inside," an illustrated leaflet for children on the care of teeth (price 2s. 6d. per dozen); "Safe Storage," a guide to food storage without refrigeration (price 2s. per dozen); "Poliomyelitis" (price 1s. per dozen); "Weaning and Mixed Feeding" (price 6s. per dozen). These publications may be obtained from the Central Council for Health Education, Tavistock House North, Tavistock Square, London, W.C.1.

BLEEDING HAEMANGIOMATOUS HAMARTOMA OF THE SMALL BOWEL

BY

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[WITH SPECIAL PLATE]

Haemangioma of the gastro-intestinal tract is a very uncommon condition, rarely diagnosed without laparotomy. Like Guy and Cooper (1950), we believe that this warrants the reporting of all cases. The case we now describe is instructive in that the diagnosis was not suspected before operation, and the result of exploratory laparotomy and excision of the lesion was dramatically favourable.

Case Report

A man aged 46 was admitted to hospital in the early hours of April 29, 1955. On the previous evening, while driving his car, he suddenly felt faint and had a desire to defaecate. He managed to drive home—a few miles—and then passed a stool mixed with bright red blood. Six days previously he had had a brief spell of nausea, vomiting, and diarrhoea which he attributed to something he had eaten, but he had felt perfectly well on the day of onset of the rectal bleeding. He had always been a healthy man, though five years previously there had been a little rectal bleeding, said to be due to haemorrhoids. There had been no weight loss and no dyspepsia.

On admission he was not shocked, though his own doctor had found him to be in a state of shock about an hour before. Abdominal and rectal examination revealed no abnormality except the presence of fresh blood in the rectum. Next morning he was passing dark fluid blood; his haemoglobin was 74%. There was no evidence of any abnormal bleeding tendency.

Over the next seven days three attacks of melaena associated with shock occurred, and on each occasion blood transfusion was required. During this week the haemoglobin level was never above 60% in spite of the infusion of a total of 6 pints (3.4 l.) of whole blood and 2½ pints (1.4 l.) of packed red cells. On May 2, after the second attack of bleeding, he was seen by one of us (A. F. R.), and it was decided that any further bleeding would necessitate urgent operation. Thereafter his progress was observed jointly by the medical and surgical units concerned. Operation was finally performed on May 6 after the third attack of bleeding, which, like the others, led to a rapid deterioration in the patient's general condition. At no time had there been abdominal tenderness or any sign of a local lesion in the abdomen, and a definite pre-operative diagnosis was not made. The following possibilities were considered (in order of likelihood): ulcerated Meckel's diverticulum; proximal colonic neoplasm; duodenal ulceration.

Exploration through a right paramedian incision centred opposite the umbilicus revealed distension of the lowest 6 ft. (180 cm.) of the ileum and of most of the proximal colon by an accumulation of blood. Just below the upper limit of the distended length of ileum there was a dimpling of the antimesenteric border with an underlying hard,

globular, intramural nodule about $\frac{1}{2}$ in. (1 cm.) in diameter (see accompanying drawing). The stomach, duodenum, and jejunum appeared normal, and no Meckel's diverticulum was found; the nodule was therefore judged to be the source of the bleeding. Accordingly a generous resection of over 8 in. (20 cm.) of ileum and its mesentery was carried out, being planned to allow for possible malignancy. Repair was effected by end-to-end anastomosis.

There was no more bleeding after the operation and the patient went home on the 15th post-operative day after an uneventful convalescence. He was seen again four weeks later, when he felt in normal health and showed no evidence of anaemia.

Pathological Report

The specimen consists of a loop of ileum 20 cm. long, together with part of its mesentery. On the mucosal surface, on the antimesenteric border, is a firm, blue, sessile polyp 1 cm. in diameter, at the apex of which is a cup-shaped depression 3 mm. in diameter. The cut surface shows the bulk of the polyp to consist of a blood clot surrounded by grey tissue, the whole covered by mucosa except at the apex, where the clot protrudes through the central depression (see Fig.). The muscularis is intact deep to the lesion.

Drawing of tumour. Note central depressed area.

Microscopically, in the submucosa there is a large, solitary, saccular vascular dilatation which has ruptured through the surface mucosa; its wall consists of smooth muscle and fibrous tissue with a small amount of elastic tissue externally; its lumen is plugged with recent thrombus. In serial sections the dilatation appears to arise from the artery to the right (see Special Plate). The vascularity of the submucosa around the lesion is no greater than that seen elsewhere in the specimen.

The lesion is clearly a vascular hamartoma; there is no evidence of a progressive neoplasm.

Discussion

Life-saving emergency operations often have to be performed to stop bleeding from duodenal and gastric ulcers. Operations for bleeding Meckel's diverticulum and bleeding oesophageal varices are also familiar, but operations for bleeding haemangiomas of the intestine must be rare, either in emergency or otherwise.

Haemangiomas may be found either in the small or the large bowel, the former being the commoner site. According to Raiford (1932) they form only 0.3% of all gastrointestinal tumours and 3.4% of tumours of the small bowel—themselves rarities. They are almost always benign, there

being only two recorded instances of malignancy, both affecting the small bowel. Brown (1924) and Kaijser (1936) consider and classify the intestinal haemangiomas in detail, and Hansen (1948) gives a most useful review of the 85 cases he found described in the literature between 1860 and 1948. His figures show that in the small bowel the tumours are often multiple, though single lesions as in the case here described, generally cavernous and polypoid in nature, are found in about 40% of cases.

The clinical presentation may take one of the following forms: (1) chronic haemorrhage, causing an unexplained anaemia with occult blood in the stools; (2) acute haemorrhage, as in this instance; (3) obstruction, acute or chronic, and sometimes the result of intussusception; (4) inflammation of the lesion itself, simulating appendicitis. In presentations (3) and (4) laparotomy is likely to be carried out without delay and the nature of the lesion soon discovered. But in (1) the diagnosis is often not made until the anaemia has been unsuccessfully treated medically for many years. In (2) the diagnosis of haemangioma will seldom be made. Even if it is considered, the alternatives of duodenal or Meckel's diverticulum ulceration are certain to appear much more likely sources of haemorrhage. This could even apply to laparotomy, much as in an operation reported by Smith *et al.* (1944) in which a radiologically detected duodenal ulcer was thought to be the source of bleeding; in actual fact the bleeding arose in an undiscovered leiomyosarcoma of the jejunum.

Associated angiomatous lesions in the skin and visible mucous membranes may suggest the diagnosis to the clinician, as in the cases reported by Rickham (1952) and by Heycock and Dickinson (1951). This association emphasizes the fact that the vascular lesions are developmental anomalies and not true neoplasms. In the present case there were no visible lesions.

In these days there is no need to stress the value of surgical treatment in cases of recurrent acute gastrointestinal haemorrhage. In reporting this case we hope to show that the diagnosis haemangioma should be considered before operation and that a search should be made for haemangiomas at the time of operation, even in the presence of an obvious peptic ulcer. This search is even more necessary before embarking on the usual empirical gastrectomy in those cases where no peptic ulcer is found.

The surgeon should not have a difficult task at operation unless the lesions are multiple. Bentley (reported by Heycock and Dickinson) in a two-stage operation, had to make 23 separate mural incisions and resect 5 cm. of bowel in a patient with 30 haemangiomas of the small bowel. The more usual difficulty will be one of diagnosis.

Summary

A case of acute intestinal bleeding in which an urgent operation became necessary because the bleeding persisted in spite of repeated blood transfusion and other medical treatment is reported. Owing to the absence of any significant previous history and any abnormality on abdominal examination, no precise diagnosis could be made pre-operatively, although duodenal ulceration, ulceration in a Meckel's diverticulum, and colonic neoplasm were thought to be likely causes. The source of the bleeding proved to be an angiomatous lesion in the ileum, and resection of a short segment of ileum led to complete cure.

The literature is reviewed and the various syndromes produced by angiomata of the intestine are discussed. It is suggested that this type of lesion should be considered as a possible diagnosis in cases of chronic anaemia with occult blood in the stools as well as in cases of acute intestinal haemorrhage. It is also pointed out that if these latter come to operation it is as well to

bear in mind the possibility of angioma even when a peptic ulcer is actually found at laparotomy, and especially when no such ulcer can be found.

We are grateful to Dr. C. S. D. Don and to Mr. R. L. Holt for permission to publish this case. Miss D. Davidson, Medical Artist, Manchester Royal Infirmary, kindly provided the drawing of the specimen.

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Medical Memorandum

Death following Injection of Paraffin into the Breast

Up to 20 years ago in Hong Kong it was considered indecent for Chinese women to have even a suggestion of a figure, and it was common for them to wear tight clothing to flatten their busts. But the advance of modern ideas, particularly through the medium of Western films, has changed this attitude. One method of improving the figure is the injection of liquid paraffin into the mammary glands, based on the use of such injections into the tissues in plastic operations, such as for the correction of nasal deformity. This has been practised indiscriminately, in some cases causing severe pain and leading to infection and sloughing.

In the following case the injection of paraffin into the breast for cosmetic reasons resulted in death from fatty embolism in the brain and lungs.

CASE REPORT

A Chinese woman aged 32, the mother of four children, who had been in poor health for some time, had arranged with a nurse to have a total of eleven injections of paraffin into her breasts to improve her appearance. She received the first three injections into the right breast at 3 p.m. on May 5, 1954. During and soon after the injections she experienced extreme pain and discomfort. Four hours later she felt giddy and faint and complained of severe headache. Her husband applied some Chinese medicinal oil to her face, forehead, and temples, and this seemed to relieve her. By 10.30 that evening she appeared to be asleep. About midnight she cried out, apparently in great pain, and this continued at intervals throughout the night. Early next morning her condition was worse, and a doctor was called in. He found her semicomatose and restless, with closed eyes; she was afebrile, but had a pulse rate of 100 per minute, regular but weak. Pupils were slightly dilated and reacted to light, more sluggishly on the left. Knee-jerks were exaggerated, but there was no neck rigidity or paralysis of extremities. Nothing significant was found in heart and lungs. There was no sweating. After prescribing for her he left.

As her condition did not improve another doctor was summoned at about 12.30 p.m. She was still semicomatose and with dilated pupils, but her right breast had become swollen and tender. Immediate removal to hospital for observation and treatment was advised.

By midnight the patient's condition had not improved despite energetic care, and a third practitioner was called in for consultation. She was then unconscious, with a temperature of 102° F. (38.9° C.) and incontinence of urine and faeces. The pupils were dilated and reflexes still present. The right breast was hot to the touch and swollen, and three puncture marks were noted. A diagnosis of cerebral paraffin embolism was suspected; ice packs were applied and nikethamide and nicotinic acid injections prescribed.

The patient was transferred next day to Queen Mary Hospital under the care of Professor A. J. S. McFadzean.

She was still unconscious, but her temperature had fallen to 100.5° F. (38° C.). She reacted to painful stimuli, and both pupils were small and reacted sluggishly to light. On auscultation there was diminished air entry with rales over both bases. Her condition progressively deteriorated and the coma deepened in spite of treatment, and she died 24 hours after admission. Necropsy was performed 20 hours later.

Post-mortem Findings.—The lips and nail-beds were cyanotic. The right breast was swollen and had three puncture marks, at 2, 5, and 9 o'clock, each about 5 in. (12.7 cm.) from the nipple. On reflecting the breast from the surface of the pectoralis major muscle about 10 ml. of clear oily fluid exuded from the under surface of the mammary gland. Incisions into the breast just under the puncture marks, and particularly in the lower medial quadrant, revealed globules of clear oil, which exuded from the yellow, semi-solid fatty tissue. A small haematoma mixed with oily fluid was present in the gland under the puncture mark at 9 o'clock. In the heart a thick layer of fat was visible under the visceral pericardium. The myocardium was soft and flabby. Petechiae were present on the endocardial lining and papillary muscle of the left ventricle and on the left interventricular septum. The bronchi contained frothy sanguineous fluid. The lungs were red and soft, with numerous petechiae on the surface. The cut surface was intensely congested. On squeezing the lung, frothy fluid mixed with oil globules exuded. Section of the kidney revealed a pale cortex and a slightly redder medulla. In the brain nothing abnormal was found in the basal arteries and soft meninges, but the cortical vessels were congested. The cut surface showed numerous minute petechiae spread over the white matter of the cerebrum.

Staining frozen sections prepared from brain and lung tissues with Sudan III resulted in red droplets appearing in the capillaries. These were considered by Professor P. C. Hou to be droplets of neutral fat, on the basis of an experiment in which he stained with Sudan III a mixture of liquid paraffin with human (neutral) fat, the fat droplets taking the stain but not the paraffin droplets.

DISCUSSION

When adipose tissues are lacerated, fat globules may enter ruptured venules and veins at the site of injury and be carried to the lungs. Most of the globules which reach the lungs are held up initially in the small arterioles, but by a process of moulding many of them ultimately pass through the pulmonary capillaries into the systemic circulation, in which they are carried to all tissues of the body and become lodged in the arterioles of such organs as the brain and kidneys.

The way in which fat globules enter lacerated or ruptured veins has not yet been established. In the case here described a blood vessel was ruptured, resulting in haematoma; in addition the high force needed to inject the liquid paraffin into the breast caused some displacement of the patient's own fat globules into the blood stream.

In cases of bone fracture it has been claimed that haematoma formation and inflammatory reaction at the site of fracture create a positive pressure in the marrow cavity which forces damaged fat cells into the veins. It has also been claimed that there is a negative pressure in the marrow cavity and that this has the effect of "sucking" damaged fat cells into the veins.

It is of interest that at the inquest on this patient the jury returned a unanimous verdict of manslaughter, and a warrant for the arrest of the nurse was issued by the magistrate sitting as coroner.

I am grateful to Professors A. J. S. McFadzean and P. C. Hou, of the University of Hong Kong, for their help; to the Honourable Director of Medical Services, Hong Kong, for permission to publish this report; and to the Commissioner of Police, Hong Kong, for access to his files.

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M. G. P. STOKER: LATENT INFECTIONS WITH VIRUSES AND RICKETTSIAE

FIG. 1.—Culture of uninfected human carcinoma cells (strain HeLa). All preparations $\times 320$, stained haematoxylin.

FIG. 2.—Cytopathic change five days after infection with herpes virus. Note giant cell and cytoplasmic processes between cells.

FIG. 3.—Cytopathic change five days after infection with herpes virus; herpes antibody added to medium two hours after virus. Focal distribution of affected cells surrounded by apparently normal cells.

A. M. DESMOND AND B. F. SWYNNERTON: ADULT HYPERTROPHY OF PYLORUS

FIG. 1.—Elongation and narrowing of pyloric canal with surrounding soft-tissue shadow and duodenal bulbar impression. (Case 18.)

A. F. ROBINSON *ET AL.*:
HAMARTOMA OF BOWEL

FIG. 2.—Elongation and narrowing of pyloric canal with proximal and distal impressions. (Case 14.)

Photomicrograph of tumour. ($\times 5$.)

R. B. THOMPSON AND S. G. M. MacKAY: ALEUKAEMIC MYELOBLASTIC LEUKAEMIA

Sections of ribs to demonstrate (a) fibroblastic proliferation; (b) reticulin fibrils. ($\times 225$.)