

A CASE OF PRIMARY CARCINOMA OF THE FALLOPIAN TUBE

BY

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The incidence of primary carcinoma of the Fallopian tube is given by various authors as 0.2–0.4% of all cases of pelvic cancer, and so far fewer than 600 have been reported in the literature. Rhu (1957) records 525 in addition to two of his own. The literature has also been reviewed and cases reported by Bancroft-Livingston (1946), Hu, Taymor, and Hertig (1950), and others. The rarity of the condition prompted the publication of this single case, with photographs of the specimen.

Case Report

A widow aged 72 had noticed a lump in the lower abdomen for several weeks. She consulted her general practitioner on account of vaginal bleeding of moderate amount. Examination revealed a tumour just palpable

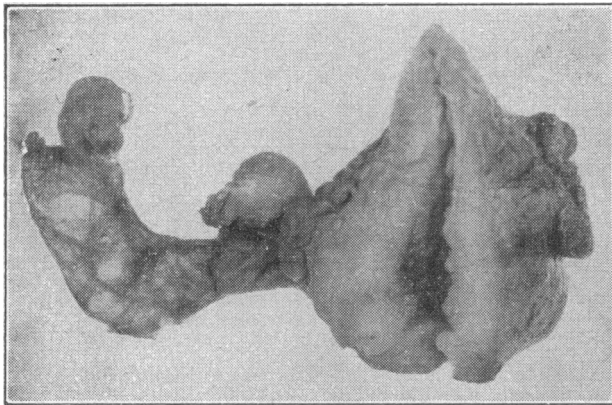


FIG. 1.—Uterus with left tube opened to show the dilatation caused by the bleeding, with the tumour at the fimbrial end.

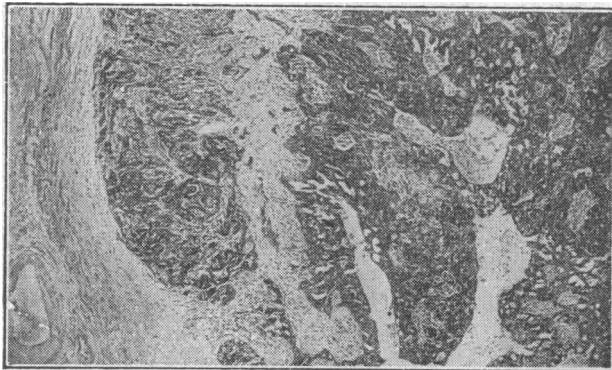


FIG. 2.—Microscopic appearance of carcinoma of the Fallopian tube. ($\times 22.5$)

above the symphysis pubis, but vaginal examination was negative. After this examination there was fairly severe vaginal bleeding accompanied by a watery discharge and disappearance of the "lump."

Three days later I saw the patient. Abdominal examination was negative except for a low mid-line scar. Vaginal examination revealed a small uterus with, on speculum inspection, an apparently healthy cervix. There was no bleeding or discharge.

There was a past history of "failed forceps" followed by caesarean section and no further pregnancies. Periods had stopped 12 years previously, at the age of 60, and there

had been no bleeding or discharge until the present occurrence. A diagnosis of carcinoma of the body of the uterus was made.

Operation.—The abdomen was opened by a low transverse incision. The uterus, ovaries, and right Fallopian tube appeared normal. The left tube was dark purple in colour and was distended in its outer two-thirds to a diameter of $\frac{3}{4}$ in (2 cm.). The proximal one-third was not distended. Hysterectomy and bilateral salpingo-oophorectomy was performed. Owing to technical difficulties the uterus was removed supravaginally and the cervix excised separately.

Specimen.—This consisted of a uterus and adnexa removed by supravaginal hysterectomy and a cervix with a cuff of vagina. The uterus was 8 cm. in length and 4.5 cm. in width across the fundus. The endometrium was brown in colour and slightly roughened. The right tube and ovary were normal. On the left side, 2.5 cm. from its commencement, the tube became widened and thin-walled, with a maximum diameter of 2.5 cm. Attached to this, at the fimbriated end, there was a solid round buff-coloured tumour measuring 2 cm. in diameter (Fig. 1). The ovary on this side was normal.

Microscopical Examination (Fig. 2).—Sections show that the solid mass at the fimbrial end of the tube is a well-differentiated adenocarcinoma which has the appearances of a primary tumour of the tube itself. The growth is circumscribed, but it is actively growing and many mitoses are present.

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RETINAL HAEMORRHAGES IN SEVERE ANAEMIAS

THEIR DIAGNOSTIC SIGNIFICANCE

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Since the pioneer days of ophthalmoscopy it has been appreciated that retinal haemorrhage may occur in a variety of severe anaemias and blood dyscrasias. Early opinions on the significance of this physical sign were handicapped by lack of correlation with precise haematological diagnosis. These opinions have been perpetuated in some modern writings. A textbook published in 1888 states that "... when the corpuscular richness of the blood falls below 50 per cent., whatever the cause of the anaemia, the tendency to retinal haemorrhage is present" (Swanzy, 1888). In a more modern textbook it is written that "retinal haemorrhages, often of a characteristic type, may occur in many blood diseases, varying as a general rule with the degree of anaemia. It seems probable that in such diseases deficient oxygenation may lead to an increase of capillary permeability and consequent increased diapedesis" (Duke-Elder, 1954). Thiel's (1948) atlas contains an illustration of the retinal changes in

pernicious anaemia, but the author states that similar changes occur in "secondary anaemias." Most authors concur, though most of them suggest that pernicious anaemia and leukaemia are particularly apt to cause retinal changes.

Severe megaloblastic and iron-deficiency anaemias are common among Natal Indians. Our experience with these patients has led us to believe that in certain severe anaemias retinal haemorrhages have greater diagnostic significance than the literature suggests (Cosnett, 1957). We have studied 74 cases of severe anaemia in Indian patients to test the validity of this impression.

Material and Methods

Twenty-three of our patients were consecutive unselected cases of megaloblastic anaemia admitted to a medical unit of King Edward VIII Hospital. The remaining 51 were patients with anaemias of various types who were selected for comparison only on the grounds of severity of the anaemia. Our object was to determine the relationship between the nature and degree of the anaemia and the presence or absence of retinal changes.

The haematological investigations were done independently by the routine pathological service of the hospital. Haemoglobin was estimated as oxyhaemoglobin in a photoelectric colorimeter. Diagnosis of the type of anaemia was based on examination of the peripheral blood, haematological indices, and, in doubtful cases, on bone-marrow examination. The megaloblastic anaemias were diagnosed on the presence of megaloblasts in the peripheral blood or marrow. Final retrospective proof of diagnosis was obtained by response to specific therapy.

Findings

Tables I and II show the nature of the anaemias in the megaloblastic and normoblastic groups respectively. Figs. 1 and 2 illustrate the Hb levels on admission to hospital and the presence of retinal changes in the two groups. We have not attempted to grade the severity of the retinal haemorrhages. Any change from the mildest to the most severe is depicted similarly.

Among the patients with megaloblastic anaemias only one had no retinal changes. This patient's Hb was the highest in the group (8.4 g./100 ml.). In most of our cases the haemorrhages were more extensive than those illustrated by Thiel (1948), Duke-Elder (1954), or

Wintrobe (1956). The haemorrhages were usually flame-shaped or coalescent, being largest and most numerous around the optic disks. Some of the haemorrhages resembled those found characteristically in leukaemia in that they had pale yellow centres. Soft yellowish or whitish exudates were seen in some cases.

In the group of 53 cases with normoblastic anaemias only four showed retinal changes. The numerals in Fig. 2 refer to these cases.

Case 1.—A 32-year-old woman had bled for two days following an abortion. On admission to hospital three weeks later her Hb was 2.7 g./100 ml. and M.C.H.C. 30%. Platelets were reported to be normal in a film of peripheral

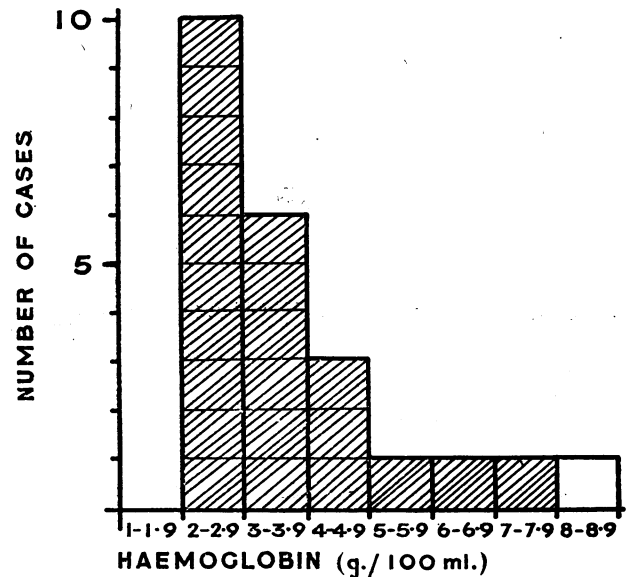


FIG. 1.—Megaloblastic anaemias. Cross-hatched squares = cases with retinal changes. White squares = cases without retinal changes.

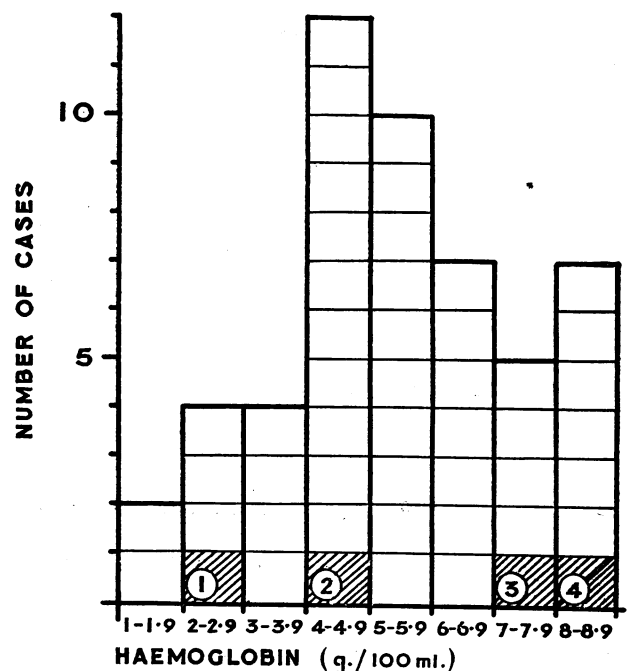


FIG. 2.—Normoblastic anaemias. Cross-hatched squares = cases with retinal changes. White squares = cases without retinal changes.

TABLE I.—Megaloblastic Anaemias

Type of Anaemia	No. of Cases
Megaloblastic anaemia of pregnancy and the puerperium	17
Pernicious anaemia	3
Nutritional megaloblastic anaemia (vegetarians)	2
Undetermined, possibly nutritional	1
Total	23

TABLE II.—Non-megaloblastic Anaemias

Type of Anaemia	No. of Cases	
Iron-deficiency anaemias	Hookworm	17
	Other chronic blood loss	8
	Undetermined	18
Acute blood loss	4	
Normocytic normochromic anaemia (chronic infections, etc.)	3	
Aplastic anaemia	1	
Total	51	

blood. There was a single small haemorrhage adjacent to the optic disk in each fundus. The urine and the blood pressure were normal.

Case 2.—A 25-year-old woman was found to have an iron-deficiency anaemia nine days after a normal confinement at home. On admission her Hb was 4.2 g./100 ml. and M.C.H.C. 25%. Platelets were reported to be normal in the film. She had a single small haemorrhage in the right fundus.

Case 3.—This was a case of idiopathic thrombocytopenic purpura with iron-deficiency anaemia in a woman aged 40. Her Hb level on admission was 7.3 g./100 ml. and M.C.H.C. 29%. Platelets varied from 9,000 to 21,000 per c.mm. Bone marrow examinations showed an iron-deficiency anaemia with "deficient platelet formation." She had multiple large coalescing flame-shaped haemorrhages adjacent to the disks in both eyes, with numerous smaller haemorrhages in the peripheral areas of the retinae.

Case 4.—A man aged 70 complained of bleeding gums. After investigations, which included two bone-marrow examinations, a diagnosis of aplastic anaemia was made. The Hb on admission was 8.4 g./100 ml., the white cell count 3,000/c.mm., and the platelet count 21,000/c.mm. The fundi showed several large haemorrhages near the disks with smaller peripheral haemorrhages.

Patients in the megaloblastic group tended to present with more severe anaemias than those in the normoblastic group. The two most severe anaemias of all were, however, of the iron-deficiency type associated with hookworm infestation. These patients had Hb levels of 1.5 and 1.6 g./100 ml. Neither showed any retinal change apart from pallor.

Discussion

Retinal changes in severe anaemias have usually been ascribed to deficient oxygenation consequent on low haemoglobin. Few authors have ventured to suggest that they might be significant in the differential diagnosis of anaemia. The report which is in closest accord with our findings is that of Foster Moore (1925), who wrote: "It is believed that there is a considerable reduction of the platelets in the blood in such diseases as pernicious anaemia and acute lymphatic leukaemia, conditions in which [retinal] haemorrhages are common; in chlorosis, on the other hand, there seems to be no diminution of the platelets and haemorrhages do not occur."

Our evidence suggests that retinal haemorrhages are much more frequent and extensive in megaloblastic anaemias and thrombocytopenic conditions than in anaemias due to blood loss or iron deficiency. It appears that retinal changes are not solely dependent on deficient oxygenation or low Hb levels. Among the factors which may contribute to these changes is thrombocytopenia. In two-thirds (15) of our cases of megaloblastic anaemia the pathologists gave an unsolicited report that "platelets were decreased in the peripheral blood," or words to that effect. Recent experience has, however, shown us that some patients with megaloblastic anaemias and retinal haemorrhages may have platelet counts of over 90,000 per c.mm. Reports of deficient platelets appear only twice in the other group of patients. In both these patients (Cases 3 and 4) the thrombocytopenia was definitely established and retinal changes were marked.

Some of the haemorrhages in megaloblastic anaemias have pale yellow centres very similar to those which occur characteristically in leukaemia. In leukaemia this is said to be due to central aggregation of leucocytes within the haemorrhagic area. It is difficult to see why

a similar change should occur in megaloblastic anaemias unless it depends on the proportion of white cells to red cells. In megaloblastic anaemia due to the low red-cell count this proportion is usually greater than normal, though not as great as in leukaemia.

Summary

During an analysis of 74 cases of severe anaemia among Indians the retinal appearances were studied in relation to the type and severity of the anaemia. It was found that retinal haemorrhages occur almost universally in severe megaloblastic anaemia and in anaemia associated with thrombocytopenia. Retinal changes occur rarely in cases of iron-deficiency anaemia or anaemia due to blood loss.

Though this sign has been mentioned previously, it is felt that its value has not been sufficiently emphasized. It is diagnostically useful in communities where severe anaemias of these types are prevalent.

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Preliminary Communications

A Sodium-excreting Steroid ?

As soon as the sodium-retaining properties of the highly potent steroid aldosterone were discovered in 1952 by Simpson and Tait, it at once became a possibility that there was also formed in the body a steroid with the reverse action—that is, with sodium-excreting properties. Indeed, the suggestion that such a substance might exist had already been made by Wilkins and Lewis (1948) to explain the excessive salt-losing condition seen in some patients with congenital adrenal hyperplasia.

Other early observations pointed to the existence of such a compound. Thus the patient with Addison's disease is much more sensitive to the sodium-retaining effects of D.C.A. than is the normal subject. This suggests the production in the normal of a substance antagonizing the sodium-retaining action of D.C.A. Then, again, difficulty has been experienced in carrying out a satisfactory bioassay of the sodium-retaining factor present in urine extracts. For instance, Luetscher, Demin, and Johnson (1952), Singer and Venning (1953), and Cope and Garcia-Llaurado (1954) all encountered urine samples in which the sodium-retaining activity was not increased by using a much larger dose of extract in the test animals, so that a proper dose-response curve could not be obtained. Indeed, it was for this reason that Cope and Garcia-Llaurado (1954) carefully refrained from drawing any quantitative conclusions from their assays. They stated that their results did not