thyroidism with metastatic calcification of the lungs should be entertained whenever sarcoidosis cannot be confirmed by tissue biopsy. The risks of hypercalcaemia, etc., as a result of calciferol treatment of sarcoidosis would appear to support this contention.

The significant points of correspondence in hyperparathyroidism and sarcoidosis are best illustrated by reference to Table II. The value of serum-protein estimation as an aid to differentiation may be clearly seen.

TABLE II.—*Indicating the Close Correspondence that May Occur Between the Findings in Hyperparathyroidism and Sarcoidosis. For Details see Text*

	Hyperparathyroidism	Sarcoidosis
Bone Changes	Generalized decalcification, cystic areas, or nil	Localized decalcification, cyst-like areas in bones, of hand, feet, and occasionally spine, or nil
Serum calcium	Generally raised	May be raised
Serum phosphorus	Low—except with retention by damaged kidneys	Usually normal; may be raised by associated renal changes
Alkaline phosphatase	Raised, with bone changes	May be raised (liver, etc.)
Serum proteins	Normal or low. A./G., normal	Raised; hyperglobulinaemia. A./G., inverted in active phase
Urine	Hypercalcaemia and albuminuria may occur in both disorders	
Kidneys	Metastatic calcification or stones	Sarcoid lesions or associated stones
Heart	Similar cardiographic changes are described	
Lungs	Radiographic similarity may occur	
Eyes	Metastatic calcification and band keratopathy may occur	

Summary

A case of primary hyperparathyroidism due to adenoma is described.

The apparent similarity between the findings in this case and those occasionally encountered in sarcoidosis is emphasized from the point of view of diagnosis.

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According to a report in the *New York Times* (December 3) the Automobile Club of New York stated recently that each year about two million American youths reach the driving age and only about one in every eight is properly taught to drive. In an effort to reduce the death rate on the roads many high schools and civic organizations have set up free driving clinics for students. They are taught both how to drive and the laws of the highway, together with the reasoning behind them. Unfortunately, however, says the article, there are far too few of these schools, and the great majority of these young drivers depend upon the guidance of friends or parents, who teach them just enough to gain a driving licence and no more. If, on the other hand, they can be persuaded to participate in a group plan that is really conducive to road safety, then a great step towards lessening the number of avoidable accidents would be achieved.

MONOCYTIC LEUKAEMIA AND PULMONARY TUBERCULOSIS COMPLICATING PREGNANCY

BY

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In view of its rarity the following case is considered worthy of record. Williams (1948) first reported a case of acute monocytic leukaemia and pregnancy, the patient (aged 35) dying within 12 days of caesarean section. Two cases of monocytic leukaemia and active tuberculosis have been reported (Heller and Hiles, 1946). In one of their cases, that of a man aged 70, there was no evidence that the leukaemic infiltration had been associated with reactivation of the tuberculosis, which involved the pleurae, pericardium, lymph nodes, and the lumbar region.

Case History

The patient was a married woman aged 25. Her father died of tuberculosis in 1933. Symptoms of pulmonary tuberculosis developed in January, 1947, and after the birth of her first child in April she became acutely ill and febrile, extensive active bilateral cavitated disease being shown by x-ray examination. In May the sputum was positive. She improved steadily on streptomycin, which was discontinued in September (244 g. in 122 days). The disease remained quiescent.

She became pregnant again in 1948, and was admitted to the Grove Park Hospital antenatal unit on September 14, when slight hypertrophy of the gums was noted. These became markedly swollen and spongy by September 25, and the haemoglobin had by then decreased from 12.5 to 8.4 g.%. There were 29,800 white cells per c.mm., with 11,600 monocytes, and monocytic leukaemia was diagnosed. A blood transfusion of 6 pints (3.4 litres) of packed cells was given. On November 6 she was delivered normally of a healthy female child, with a blood loss of 15 oz. (425 ml.). The gums then became almost normal and all the white cells returned to a normal figure, except the monocytes, which remained at 2,000–3,000 per c.mm. Her condition remained good, but within five weeks of delivery sputum culture showed tubercle bacilli, although there was no radiological change.

At the end of February, 1949, the leucocytes began to increase rapidly, and by the middle of April they numbered 26,000. In early May, after a mild "coryza," she became much worse. The gums swelled again and developed purplish "granulomatous" masses. The left tonsillar gland was enlarged and tender. The spleen could not be palpated. By the middle of May the total leucocyte count was 96,000, and the monocytes and polymorphs each numbered about 30,000 per c.mm., with many immature forms of both series present. Her temperature was 100° F. (37.8° C.), and there were marked coarse rales over the left upper chest. Urethane (1 g. thrice daily), a blood transfusion of 3 pints (1.7 litres), and penicillin were given. There was a temporary remission in mid-June, when the leucocyte count fell to 23,000, but petechiae and ecchymoses reappeared. Streptomycin (1 g. daily)—the organisms being still sensitive—and a third transfusion of 4 pints (2.3 litres) were given, but the leucocyte count rose to a maximum of 111,000 and the haemorrhages increased. The patient became rapidly weaker and died suddenly from a haemorrhage into the medulla oblongata on July 16.

Investigations.—Radiographs of the chest indicated progressive healing of a diffuse bilateral fibrocaseous tuberculosis.

The sputum was tubercle-negative in the last two months of life. On November 11, 1948, a report on the sternal marrow described it as "a very active marrow in which almost half the cells are members of the monocyte series." On June 10, 1949, a further report on the sternal marrow stated: "The most numerous cells are large abnormal myelocytes. Erythropoiesis shows a megaloblastic picture simulating pernicious anaemia. The process suggests a monocytic leukaemia of Naegeli type." Bleeding time: ear, 20 minutes; finger, 5 minutes. On June 10, 1949, the coagulation time was 2 minutes and the platelets numbered 20,000 per c.mm.

Necropsy

"Generalized purpuric and petechial haemorrhages throughout the body. Left pleural effusion (blood-stained). Extensive pleural adhesions. *Lymph nodes*: Tonsils and paratracheal glands necrotic. Abdominal glands enlarged and pale greenish in colour. *Lungs*: Extensive but healing fibrocaceous tuberculosis. A ragged necrotic cavity $\frac{1}{2}$ in. (1.25 cm.) in diameter at apex of left lower lobe. Spleen and suprarenals very soft. *Brain*: Small haemorrhages in right parietal lobe, in right occipital lobe, and in upper medulla, with bleeding into fourth ventricle."

Histology.—Lungs showed extensive leukaemic infiltration. The tuberculous areas gave no evidence of reactivation even where a walled-off calcifying caseous focus was infiltrated by monocytes. Leukaemic infiltration was present in the liver, spleen, brain, and lymph nodes, with a subretinal deposit in the left eye. The sternal marrow was almost grey and showed displacement by mononuclear cells; foci of erythropoietic tissue were not seen. (There had never been any evidence of tuberculous infection of the bone marrow.)

The infant was healthy at birth and had a normal blood count on October 6, 1949, when 11 months old.

Discussion

This case was considered to be a true leukaemia rather than a tuberculous leukaemoid reaction, particularly in view of (a) the high white-cell count; (b) the widespread leukaemic infiltration in the lymph nodes and the viscera; which were free from tuberculosis; and (c) the massive cellularity and anaplasia of the bone marrow.

The remission appears to have coincided with parturition. Remissions have, however, been observed to follow closely on transfusions of fresh blood (Whitby, 1949). These may therefore have contributed to the remission in this case, which lasted four months, although the monocytes increased first in the peripheral blood, but after a mild infection there was a rapid rise in the myelocytes. Urethane appeared to produce a decrease of leucocytes of both myelogenous and monocytic series, but this remission may be related to the second transfusion. A further course of urethane and a third transfusion had no effect. In spite of the positive sputum five weeks after delivery, at necropsy there was no evidence of reactivation of tuberculosis. It seems likely, therefore, that a small focus was reactivated at or soon after parturition and later became quiescent and remained so. This is interesting in view of the statement that tuberculosis is a not infrequent complication of myeloid and lymphatic leukaemias (Whitby and Britton, 1946).

My thanks are due to Dr. M. H. Logg for permission to publish this case; to Dr. M. M. Nagley (Grove Park Hospital) for much advice; to Mr. Alistair Gunn (obstetrician), to Dr. E. H. Bailey (pathologist) and his technician, Mr. E. J. White, for the pathological work; and to Dr. J. C. White and Dr. J. W. Crofton (British Postgraduate Hospital, Hammersmith), Dr. C. W. L. Jeanes (Greenwich Chest Clinic), and Dr. J. H. Simmons (Lewisham Hospital).

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Medical Memoranda

Perforated Lymphosarcoma of Small Intestine

Perforation of a sarcoma of the small intestine is a rare complication of this disease. Ullman and Abeshouse (1932) reported two cases with this complication out of a total of 126. Lewis (1939) reported six cases out of a total of 400, while Kenneth Frazer (1945) reported three perforations in nine cases of sarcoma. In view of these figures it was felt that the following case is worthy of publication.

Case Report

The patient, a man aged 47, was admitted to hospital on May 7, 1950. He gave a history of 14 days' pain in the right iliac fossa; the pain was colicky in nature, but he did not feel sick, and his appetite remained good.

On the day of admission he had eaten a good dinner, but vomited immediately afterwards. His abdominal pain started at this time. He was seen by his own doctor at 6 p.m. on the same day, when tenderness in the right iliac fossa was noted and his temperature was 99.4° F. (37.4° C.) and his pulse rate 90.

He had a long journey to hospital, coming from the Tweed Valley to Newcastle-upon-Tyne. He was seen shortly after admission, at which time he had generalized abdominal rigidity; his temperature was 102.1° F. (38.9° C.) and his pulse rate 110. A pre-operative diagnosis of peritonitis was made, probably due to a localized appendix abscess which had subsequently perforated. No mass could be palpated owing to the generalized rigidity.

At operation under general anaesthesia a muscle-cutting incision was made in the right iliac fossa. On opening the peritoneum, purulent fluid was found, especially in the pelvis; this was aspirated. The appendix was then inspected and found to be normal, except for injection of its peritoneal surface. On further exploration of the abdomen a mass was found in the lower ileum. It was delivered into the wound and found to be perforated. There were massive glands in the omentum, extending up to the porta hepatis.

The tumour was resected, together with a wedge of mesentery. It was noted at the time of section that there were numerous milium-sized nodules in the mucosa. In view of the lymphatic spread no attempt was made to excise the bowel wide of these nodules. An end-to-side anastomosis was performed and the wound was closed with drainage into the pelvis.

Post-operatively the patient was treated by aspiration and intravenous glucose saline infusion until flatus was passed. He was also given penicillin, 100,000 units six-hourly, and streptomycin, $\frac{1}{2}$ gr. (32 mg.) twice daily. These were continued for five days. He made a satisfactory recovery from the operation and was discharged to a convalescent home on May 20. He was subsequently given deep x-ray therapy.

The pathological report on the specimens by Dr. R. Schade is as follows: "(1) The specimen consists of a loop of small bowel approximately 15 cm. long. Over a length of 0.5 cm. it is almost completely obstructed by neoplasm which has extended through all coats of the bowel, with resultant perforation in one part. The proximal bowel is hypertrophied and the lumen throughout contains blood clot. A number of small subserosal nodules of tumour are seen. The mesenteric nodes draining the area are infiltrated by neoplasm. (2) A segment of bowel 1 cm.