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MEDICAL MEMORANDA

Arthropathy in Transfusional Siderosis

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Arthropathy as a complication of idiopathic haemochromatosis was described in 1964 (Schumacher). It has since been recognized with increasing frequency, and descriptions of the joint changes have been recorded (Hamilton, Williams, Barlow, and Smith, 1968; Wardle and Patten, 1969; Dymock, Hamilton, Laws, and Williams, 1970). The metacarpophalangeal joints are usually affected and some patients have involvement of larger joints.

There have been no reports of arthropathy occurring in transfusional siderosis.

Case Report

A 60-year-old woman presented with congestive cardiac failure in December 1962. She did not have arthritis and there were no features of haemochromatosis. Haemoglobin was 5.1 g/100 ml, the red cells being normochromic and normocytic. The peripheral white cell and platelet counts were normal. There was no evidence of haemolysis or of bleeding into the gastrointestinal or urinary tract. Bone biopsy showed sparse haemopoietic cells which were mostly of the myeloid series. The red cell precursors were mainly early and intermediate normoblasts with a scarcity of late forms.

A diagnosis of hypoplastic anaemia was made and it was subsequently necessary to readmit her for blood transfusion at intervals of between 2 and 10 weeks. Haemoglobin was kept in the range 6-12 g/100 ml. Treatment with various haematinics including iron supplements did not reduce the transfusion requirements. After one year of treatment the serum iron was 161 µg/100 ml and the total iron binding capacity 200 µg/100 ml. The iron supplements were therefore stopped. In December 1966 the serum iron had risen to 267 µg/100 ml and daily desferrioxamine was started. When she became anaemic congestive cardiac failure recurred. She was therefore treated with digoxin and diuretics.

In March 1967, by which time she had received a total of 166 units of blood, she developed acute polyarthritis. The knees, ankles and wrists, and the left shoulder were painful, swollen, and had limited movement. No abnormality was detected in the spine or in the small joints of the hands and feet. Rheumatoid nodules were not found.

Investigations were: M.C.H.C. 34.5%, E.S.R. 143 mm in one hour, uric acid 3.6 mg/100 ml, Rose-Waaler test negative, and lupus erythematosus cells not found. The joints were not x-rayed at that

time but a chest x-ray examination the following year showed opacification in the synovium of the left shoulder which was not seen on previous films.

Enteric-coated aspirin was effective in relieving the symptoms and this was continued until November 1967, when the arthritis subsided. She experienced no further joint symptoms until March

FIG. 1—Femoral bone marrow showing abundant megakaryocytes. (Haematoxylin and eosin. $\times 262$.)

FIG. 2—Synovium of right knee joint containing abundant pigment in surface and deep cells. (Perls' stain. $\times 163$.)

1969, when she complained of aching in all limb joints. By August this had become severe. At that time she had been transfused a total of 272 units of blood. She died suddenly of cerebral haemorrhage in September 1969.

Necropsy.—The body was that of a pale old woman with slight oedema of the lower legs and ankles. The left cerebral hemisphere contained a large blood clot. There was conspicuous brownish discoloration of the synovium of the right hip, knee, and ankle joints but not of the sternoclavicular joints. The shoulder and finger joints were not examined. The upper half of the femur contained

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red marrow. The spleen was slightly enlarged (300 g) but the lymph nodes in various regions showed nothing abnormal. The liver had a brown cut surface that was slightly enlarged, firm, and finely lobulate. The heart was enlarged (490 g) owing to moderate hypertrophy of the right and left ventricles. There was severe oedema and congestion of both lungs. Scanty atherosclerosis was seen in the patent coronary arteries.

Histological Findings.—The bone marrow contained a moderate excess of megakaryocytes and haemosiderin and a slight excess of myeloid precursors (Fig. 1). A section of synovium from the right knee had villous projections covered with swollen synovial cells. Haemosiderin was abundant both in synovial cells and in macrophages in the underlying stroma (Fig. 2). Massive depositions of

FIG. 3—Heart showing abundant haemosiderin. (× 233.)

Q Fever Endocarditis Treated with Trimethoprim and Sulphamethoxazole

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We here present a case of endocarditis due to *Coxiella burnetii* which appears to have responded to trimethoprim and sulphamethoxazole.

Case History

The patient, a woman aged 45, developed night cramps in both legs in August 1969. By January 1970 she had classical intermittent claudication and presented to this hospital complaining of pain in her right foot, even at rest, loss of weight, poor appetite, exhaustion, and recent breathlessness. There was no history of a rash or of a recent upper or lower respiratory tract infection.

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haemosiderin were found in the heart (Fig. 3), thyroid, lymph nodes, and suprarenals. There was a considerable amount of haemosiderin in the liver, which was fibrotic, and also in the pancreas, although there was no fibrosis in this organ. Haemosiderin was not seen in skin or skin appendages.

Discussion

Arthropathy is a well-recognized complication of idiopathic haemochromatosis though iron is not always found in the synovial membrane. The diagnosis depends largely on clinical and radiological features. The changes are found more often in the finger joints, large joints being less often involved. We have been unable to find any reference to arthropathy as a result of transfusional siderosis. In the present case the clinical features were mainly involvement of large joints such as knees, ankles, shoulder, and hips. Unfortunately no specific radiographic studies were made of the joints at the time that she complained of arthritis, but at necropsy siderosis was a conspicuous feature.

Absence of both pancreatic fibrosis and cutaneous siderosis was against a diagnosis of idiopathic haemochromatosis, and it seems likely that the arthropathy in this case was directly due to the repeated transfusions and the resultant high levels of serum iron.

We are grateful to Dr. A. P. Dick for permission to report this case.

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Her only previous history was of a subarachnoid haemorrhage in 1965. The peripheral pulses were noted to be normal at that time. Examination on the present admission showed a temperature of 103°F (39.4°C), pulse 90/min, regular, and blood pressure 160/100 mm Hg. The heart was of normal size and there were no murmurs. The peripheral pulses are shown in Table I. All other systems were

TABLE I—State of Peripheral Pulses as assessed clinically in January 1970 (on Admission) and June 1970 (after two Months of Treatment)

	Right		Left	
	January	June	January	June
Femoral	—	+	+	+
Popliteal .. .	—	*	—	+
Posterior tibial ..	—	*	—	—
Dorsalis pedis ..	—	+	—	+
Subclavicular ..	+	+	+	+
Axillary	+	+	+	+
Brachial	—	+	+	+
Radial	—	+	+	+

+ = Palpable.
— = Impalpable.
* = Right leg amputated before second assessment.

clinically normal except that she was thought to have a minimal degree of hepatosplenomegaly.

The right foot was gangrenous and an above-knee amputation was performed. The stump healed well but she remained pyrexial—temperatures over 100°F (37.8°C) being frequently recorded. Extensive investigations failed to find the cause of her illness but by the middle of March a pansystolic murmur was noted at the apex for the first time. Numerous blood cultures were sterile but anti-