gression to acute infarction the treatment is surgical when this is practicable. About 100 cases of successful elective revascularization of the cœliac and/or mesenteric arteries have been reported (Williams 1971). Unfortunately, there is incomplete follow-up data but it appears that relief of pain occurs in 90% and malabsorption, if present, is improved in 75–80%. The post-operative complication and morbidity rate was estimated to be 15–20% with an overall operative mortality of 5%.

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Dr L J Findley (*Charing Cross Hospital*) asked whether patients presenting with symptoms of mesenteric angina such as abdominal pain and weight loss, without significant positive physical signs, and with essentially normal routine investigations including barium studies, were sometimes labelled as 'psychiatrically ill'.

Dr Josse replied that he could find no reference in the literature to patients with this syndrome being so diagnosed before the true diagnosis was reached.

Pernicious Anæmia Followed by Autoimmune Hæmolytic Anæmia

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S S, woman aged 56.
Italian of Ligurian extraction

History: Came of a healthy family. History of 6 pregnancies, 4 of which ended with abortions. Underwent surgery for gall-stones at 38. Last pregnancy, at 40, complicated by late parturition. July 1973, developed increasing weakness, shortness of breath, palpitations, anorexia, moderate fever (38°C).

On admission: She appeared pale, including the visible mucosæ. Tongue: normal mucosal pattern, moist and not painful. Liver, spleen and lymph nodes not enlarged. Reflexes enhanced; no signs of involvement of central or peripheral nervous system.

Investigations (July 1973): ESR 20 mm in 1 hour (Westergren); hæmatocrit 17%; Hb 5 g/100 ml; reticulocytes 3%. Peripheral blood characterized by anisocytosis, poikilocytosis and macrocytosis. WBC 5000/mm³ (neutros. 74%, eosinos. 2%, lymphos. 18%, monos. 6%). Platelets 100 000/mm³.

Plasma iron 320 μg/100 ml; bilirubinæmia 7 mg/100 ml; lactic dehydrogenase 4000 mu/ml. Coombs test, direct and indirect, negative. Total proteins: 7.32 g/100 ml: albumin 61.9%; globulins – α_1 4.6, α_2 9.6, β 10.4, γ 13.5% (normal range). Bone marrow: marked erythroblastic hyperplasia, with predominance of megaloblasts; large metamyelocytes also present; megakaryocytes present in normal number, but rather immature. Titre of autoantibodies against gastric mucosa was weakly positive. Gastric secretion of free hydrochloric acid, after maximal stimulation with histamine, was absent.

X-ray examinations of alimentary tract negative. Schilling test of vitamin B_{12} absorption was 2% of the dose (normal 7–22%, corrected to 28.9% by the addition of intrinsic factor). Treatment with vitamin B_{12} caused a high reticulocyte count (60% on the fifth day) and after two weeks there was a rise in hæmatocrit to 26%, hæmoglobin to 7.5 g/100 ml; plasma iron 70 μg/100 ml.

Iron therapy was begun intravenously (as suggested by Wintrobe (1967) in middle-aged women with several pregnancies); hæmatocrit rose to 33% and hæmoglobin to 10 g/100 ml. The patient was discharged with the prescription of vitamin B_{12} and iron therapy and was in good condition for over a month.

November 1973: She complained of fatigue and weakness. On examination she was pale, the scleræ showing a yellowish colour. No other physical signs were present.

Investigations (November 1973): Hæmatocrit 28%; hæmoglobin 8.8 g/100 ml; reticulos. 20%; WBC 12 800/mm³; platelets 200 000/mm³. Morphology of the blood showed anisocytosis, poikilocytosis with mild macrocytosis.

On admission: Bone marrow showed marked hyperplasia of the normoblastic series, with granuloblasts and megakaryocytes in the normal range both in number and morphology. Bilirubinæmia 2.2 mg/100 ml (1.1 of the indirect and 1.1 of the direct reacting types), plasma iron 120 μg/100 ml; urine urobilinogen increased; no blood detected in fæces. Direct Coombs test positive; by means of immunofluorescence with

specific antisera, the immunoglobulins coating the red cells identified as IgG. Antiglobulin test on whole serum positive against a red cell panel of the CDe/cdE type. ^{51}Cr-tagged red cells had a half-life of five days. Total proteins 6.5 g/100 ml; albumin 64%; globulins – α_1 3%, α_2 9%, β 13% (normal range), γ increased to 20%.

Immunoglobulins (by immunodiffusion technique): IgG 1310, IgA 275, Igm 140 mg/100 ml. Values for serum complement normal. Coombs test (antinuclear antibodies) positive. LE, mononucleosis and Rose-Waaler tests negative.

After 10 days' therapy with prednisone (60 mg daily) and azathioprine (150 mg daily) hæmatocrit increased to 36% and hæmoglobin to 11 g/100 ml; the reticulocyte count fell to 5%.

At present, the patient is well, hæmatocrit (41%) and hæmoglobin (12.5 g/100 ml) normal. Coombs test still positive. Prednisone has been reduced because of an increase in blood glucose level and the immunosuppressive drug has been reduced.

Comment

The diagnosis of pernicious anæmia was based on the presence of megaloblasts in the bone marrow, and macrocytes in the peripheral blood; on achlorhydria, even after maximal histamine stimulation; on the normalization of the erythropoietic bone marrow cells after vitamin B_{12} administration; and on a positive Schilling test.

Serum lactic dehydrogenase levels were increased and antibodies against gastric mucosa were present. The fact that at first there was only a partial remission of the pernicious anæmia can be related to the depletion of the body iron stores, which is not uncommon, according to Wintrobe, in middle-aged women with a past history of several pregnancies. In fact the administration of iron caused an increase in the hæmatocrit and hæmoglobin values.

The diagnosis of autoimmune hæmolytic anæmia was based on high reticulocyte counts, jaundice, high urobilinogen in the urine, positive Coombs test and shortened red cell lifespan; it is very uncommon in subjects with pernicious anæmia.

The association of pernicious anæmia and a weakly positive antiglobulin test is not considered significant. Recorded cases were not complicated by overt hæmolytic anæmia or even compensated hæmolysis (Forshau & Harwood 1965, Henneman 1956, Wenner 1959). It seems possible that in these cases gammaglobulins, devoid of anti-erythrocytic activity, were nonspecifically bound to the red cell membrane.

Quite different is the patient described by Schwartz & Costea (1966) in whom an autoimmune hæmolytic anæmia was followed, after some time, by pernicious anæmia. This was probably due to an increased consumption of folic acid and vitamin B_{12} during the enhanced erythropoietic response to the destruction of circulating red cells.

It is also well known that pernicious anæmia may be associated with systemic lupus, thymoma, chronic rheumatism and hyperthyroidism (Pirofsky & Vaughan 1968), diseases which may be caused by a disturbance in the immunologic mechanisms of the body.

Different again is the case described here: in our patient pernicious anæmia was followed by hæmatological and clinical signs of uncompensated autoimmune hæmolytic anæmia. It is possible that she developed an asymptomatic viral infection which triggered the autoimmune hæmolytic process, or that the autoimmune hæmolytic crisis which followed pernicious anæmia was only an occasional (causal) association. A third possibility is that the patient is now developing a lymphoproliferative disease, the first sign of which was the severe hæmolytic anæmia with positive Coombs test.

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Dr J S Stewart (*West Middlesex Hospital*) said that the case reported by Schwartz & Costea (1966) would fit the diagnosis of Addisonian pernicious anæmia a good deal less well than Professor Salvidio's case. The megaloblastic anæmia which followed the autoimmune hæmolytic anæmia in that case seemed to be due to folic acid deficiency.

Dr Graham Neale (*Royal Postgraduate Medical School*) said that this interesting case report possibly represented a rare but true association between the two diseases which have an autoimmune basis. Four or five similar cases had been seen at Hammersmith Hospital by Professor Dacie but unlike the patient of Charache *et al.* (1968, *Johns Hopkins Medical Journal* **122**, 184), were not recorded in the literature.

Dr Neale then asked whether treatment of the hæmolytic anæmia with prednisone had led to the reappearance of acid in the stomach.

Professor Salvidio replied that no further examination of the gastric juice had been performed on the patient after prednisone.

(Meeting to be continued)