Summary

The condition of spontaneous circumrenal haematoma is described, with two personal case histories.

Though rare the condition is an important one to bear in mind, as it may enter into the differential diagnosis of the acute abdomen and if treated expectantly is nearly always fatal.

The aetiology and clinical course are discussed. An acute and a chronic form can be distinguished.

Treatment should consist in surgical intervention and, in most cases, nephrectomy.

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APLASTIC ANAEMIA FOLLOWING NEOARSPHENAMINE

BY

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Aplastic anaemia is a well-recognized but rare complication of organic arsenical therapy. Wintrobe (1946) states that fewer than 100 cases have been reported in the literature, also that less than one-half of the cases described showed complete aplasia of the marrow. Recovery after complete aplasia has been recorded, however (Wintrobe, 1946). The case described below, which exhibited anaemia, leucopenia, thrombocytopenia, and a completely aplastic marrow on sternal puncture, seems to fall into this class and to be worthy of record.

Case Report

The patient, a man aged 52, contracted primary syphilis and gonorrhoea abroad in June, 1946. He received 37 injections of neoarsphenamine and 37 of bismuth during the next nine months.

He noticed nothing abnormal until April, 1947, when he had a severe epistaxis lasting 16 hours. The red blood cells numbered less than one million. Injections were stopped, but a month later further severe nose-bleeding occurred, with the appearance of petechiae and "papules" on the arms and legs. His haemoglobin was 30%; colour index, 1; red blood cells, 1,500,000 per c.mm.; white blood cells, 3,000 per c.mm. (L. 78%, M. 1%, P. 21%); platelets, 25,000 per c.mm.; coagulation time, 14 minutes. Wassermann reaction and Kahn test were negative. The spleen was never palpated.

He was treated with iron, a fresh blood transfusion, and 10 ml. of pentnucleotide every 12 hours. He received altogether some 6 pints (3.4 litres) of fresh blood. Four days later a blood count showed: red cells, 1,670,000 per c.mm.; haemoglobin, 30%; colour index, 0.9; bleeding time, 3½ minutes; coagulation time, 6 minutes; leucocytes, 2,000 per c.mm. (L. 73%, N. 2%, E. 3%, P. 22%).

He then returned to England, and during the voyage was given 1 ml. of "hepastab" daily, 45 gr. (3 g.) of iron and ammonium citrate, and 5 mg. of folic acid thrice daily. On his return he stated that he did not feel very ill, but became giddy

and tired on the least exertion. Up to this time he had had 11 blood transfusions in all, the last being on May 19. No further epistaxis had occurred, but he had noticed some oozing of blood from his gums and he found that he bruised easily and "came out in little red spots." Previous illnesses and family history revealed nothing relevant.

He was admitted to hospital under our care on May 31. He was a very co-operative man, very pale, and obviously grossly anaemic. His skin was sallow and sunburnt. There was pronounced foetor oris; his teeth were in a bad condition, with much dental sepsis; the gums were soft and spongy, with pus oozing from them. The tongue was furred but moist. No bismuth line was present. No glands were palpable and no petechiae or bruises were seen. Over his legs and feet the skin was scaly, pigmented, and ulcerated. The blood pressure was 120/60. A soft systolic murmur was present over all areas in the praecordium. The capillary resistance test was strongly positive in three minutes at a pressure of 90 mm. Hg. Nothing abnormal was detected in the chest apart from an occasional rhonchus at the bases. The spleen was not palpable.

The pupils reacted normally to light and to accommodation. Ophthalmoscopy showed normal optic disks. No exudates were seen, though multiple large haemorrhages were present in both retinae. The central nervous system was otherwise normal.

Chart showing haemoglobin findings

The diagnosis of aplastic anaemia due to arsenical therapy was made. The haemoglobin findings are shown in the accompanying graph. Radiographs of lungs and tibiae were negative. The B.M.R. was 4%.

The patient was kept in hospital until Sept. 16, when he was discharged home. During this time his haemoglobin rose from 20% to 104%. A total of 2 pints (1.14 litres) of whole blood and 29 pints (16.5 litres) of packed red cells was given over this period. He did not react adversely to any transfusion, and over the whole period made slow but steady improvement. Penicillin was administered, 40,000 units four-hourly, for the first month, the dosage gradually being reduced over the following three months to 100,000 units twice daily. Liver extract was given by injection (hepalon) 2 ml. daily at the onset, being reduced to twice a week after two months. He also received pyridoxin 100 mg. daily, ferrous sulphate 10 gr. (0.65 g.) thrice daily, two multivitamin tablets thrice daily, and folic acid tablets (15 mg.) daily.

On Oct. 2, 14, and 27 he was readmitted for the transfusion of 2 pints (1.14 litres) of packed cells on each occasion. The condition of the skin of the feet and legs greatly improved, and the capillary resistance test, still positive on Oct. 14, became negative after this time.

Two further pints of packed cells were given on Nov. 20 and Dec. 18. It was apparent at this time that he was making steady progress, and that he was beginning to pick up on his own. He was last seen on May 31, 1948, when he was in excellent general condition. The Wassermann reaction and Kahn test were negative. Our last report, received from abroad in October, 1948, was that he was in perfect general health and had received no further treatment.

Haematological Findings.—On June 2, 1947, these were as follows: haemoglobin, 22% (3.03 g.%); red cells, 1,210,000

per c.mm.; colour index, 0.9; white cells, 900 per c.mm. (lymphocytes 80% and neutrophil polymorphs 20%, approximately). No red cell abnormalities were noted. On June 3 the bleeding time was longer than 10 minutes, the clotting time (venous blood) 11 minutes, the prothrombin time 11 seconds (normal, 8 seconds); blood platelets, 3,400 per c.mm.; reticulocytes were not found. The blood was Group O (IV) Rhesus-positive.

Table showing Results of Sternal Punctures

Date	June 3, 1947	Aug. 16, 1947	May 21, 1948
Total nucleated count ..	3,400 per c.mm.	10,200 per c.mm.	25,000 per c.mm.
Myeloblasts ..	—	0.4%	1.2%
Premyelocytes ..	—	0.6%	0.8%
Neut. myelocytes ..	1 seen in 3 films	6.8%	9.2%
Neut. metamyelocytes ..	—	3.4%	8.6%
Neutrophils, segmented ..	Present	15.0%	24.2%
Eosin. myelocytes ..	—	1.4%	0.2%
Eosinophils ..	—	1.0%	0.6%
Basophil myelocytes ..	—	—	—
Basophils ..	—	—	0.2%
Lymphocytes ..	Present	32.0%	29.0%
Monocytes ..	—	3.2%	2.0%
Megakaryocytes ..	—	0.2%	Present—mature forms found
Haemocyto blasts ..	—	1.4%	0.2%
Normoblasts, basophil ..	1 seen in 3 films	6.8%	2.6%
Normoblasts, polychromatic ..	—	21.0%	19.8%
Normoblasts, orthochromic ..	—	6.2%	1.0%
Myeloid: erythroblast ratio ..	—	1:1.6	1.87:1
Miscellaneous cells ..	—	0.4%	0.4%
Naked eye ..	A fatty fluid	A fatty fluid	Marrow particles very evident

The sternal puncture findings are presented in the accompanying Table. They show a progressive return of cellularity from a condition of almost complete aplasia.

Haematological Progress.—The neutropenia persisted until May 18, 1948, when for the first time the neutrophil polymorphs amounted to more than 2,000 per c.mm. Reticulocytes were not found in any numbers until Feb. 2, 1948, when they totalled 5% of 3,800,000 red blood cells per c.mm. (190,000 per c.mm.). The platelet count did not rise to normal figures until after Feb. 24, 1948, when it was 40,000 per c.mm. On May 21, 1948, the platelet count was normal.

Discussion

It is of interest to note that the last cell series to recover completely in the circulating blood was that of the neutrophil polymorphonuclears. Neutropenia persisted from April, 1947, to May, 1948—a period of 13 months. Reticulocytes did not appear until after nine months of treatment, and platelets returned to normal only after a full 12 months. The capillary resistance test remained positive for seven months. It was felt that, of all the therapeutic agents given, penicillin and blood transfusions were the most important. The former totalled some 35 mega units, administered over a four-months period. The condition of the mouth and legs gave cause for anxiety in the early months, but about the end of the sixth month of the disease a gradual improvement became apparent.

Summary

A case of aplastic anaemia following neoarsphenamine administration is described. Depression was present in all marrow elements—a condition of almost complete aplasia. Recovery was complete after 13 months.

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The Ministry of Health states that hospitals may provide clothing for those "up" patients unable to provide it themselves and who are not obtaining it from other sources such as voluntary organizations. They would normally be long-stay ambulant patients. The patients may retain this clothing after discharge from hospital if the hospital is satisfied that the patients need clothing and are unable to obtain it from their own resources and without help.

HISTAMINE HEADACHE*

BY

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Headache is the most frequent complaint encountered in medical practice. It presents no diagnostic problem: the diagnosis is made by the sufferer. But the doctor must know the cause if he is to treat the patient efficiently.

Table I classifies the forms of headache that I have treated. The present paper is concerned mainly with the type of headache due to histamine, as described by Horton in 1941.

TABLE I.—Results of Treatment by Histamine

	Histamine Headache	Migraine	Psychical Headache	"Relaxation" Headache	Alcoholic Headache	Headache Associated with Hypertension	Headache with Organic Basis	Total	Controls without Headache
Complete cure ..	29	2	2	2	1	2	2	40	
Considerable improvement ..	8	1	2	1	1	1	2	16	
Some improvement ..	6	2	2	0	0	0	1	11	
No change ..	2	3	2	0	1	0	5	13	Slight or no reaction
Total ..	45	8	8	3	3	3	10	80	10

Clinical Features and Aetiology

Vallery-Radot in 1925, Brickner-Riley in 1935, and Money in 1939 described cases of atypical migraine which seem to be similar to the histamine headache described by Horton. It was Horton, however, who first described the syndrome, made the aetiology clear, produced the same symptoms experimentally, and outlined the method of treatment.

The characteristic symptoms of histamine headache, the frequency of which is shown in Table II, are as follows: it occurs in paroxysms, is unilateral, and is confined to a circumscribed area; on the same side there is congestion of the nasal mucous membrane; attacks frequently come on at night, and the patient often rises in the morning with headache. It is neither hereditary nor familial, has no connexion with menstruation or ocular disturbances, and is unassociated with vomiting. The condition is resistant to all treatment. In these patients headache produced by the injection of histamine is of the same type as occurs spontaneously.

As to the aetiology, it must be presumed that the accumulation of histamine produces an upset in cell metabolism. This accumulation is the result either of overproduction of histamine or of the inhibition of the destructive factor. It is also possible that individual hypersensitivity plays a part in cases in which the production of histamine is normal. According to Horton, in histamine headache the symptoms are caused by dilatation and constriction of the external and common carotid arteries. On the other hand, Wolff, Schumacher, Clark, Butler, Shuterland, and Pickering have proved, on the evidence of many cases, that the internal carotid is responsible for the quality and the intensity of histamine headache, and that the dilatation of the external carotid is of decisive importance in migraine. They demonstrated with photographs, taken during headache produced by the injection of histamine: (1) the amplitude of intracranial pulsation (which is increased); (2) the

*A paper read at the International Congress of Medicine held at Budapest in September, 1948.