12 g. Coincident with chemotherapy, his temperature returned to normal. On November 27 he developed a sore throat, which persisted for three days. A blood count showed: white cells, 13,000 per c.mm.; platelets, 8,000 per c.mm. That evening he had an epistaxis after blowing his nose. Dry "stypven" and adrenaline packs failed to stop the bleeding; five hours after the onset of the epistaxis a transfusion of 1 pint (570 ml.) of fresh blood was given. On the next day the bleeding ceased, although a platelet count showed no significant alteration. On November 29 a further transfusion of 1 pint (570 ml.) of fresh blood was given. A blood count (before transfusion) showed: Hb, 92% (Haldane), or 12.7 g. per 100 ml., and 4,000 platelets per c.mm. Microscopical examination of the urine revealed a slight excess of red cells. This was the only occasion on which any urinary abnormality was detected.

December 2 (Eight Days After Admission).—Although epistaxis had not recurred, the purpuric lesions and the glandular enlargement persisted, and the sputum was still blood-stained. The spleen was no longer palpable. A blood count showed Hb, 96% (Haldane), or 13.2 g. per 100 ml.; white cells, 9,050 per c.mm. (including 23% atypical mononuclear cells); platelets, 7,000 per c.mm. The heterophil antibody titre was 1:996 (presumptive test), 1:996 (guinea-pig absorption), and 1:28 (beef-cell absorption).

December 12 (18 Days After Admission).—The lymph nodes were now of normal size. Although the petechiae had almost disappeared and bleeding from the respiratory tract had ceased, a blood count showed only 3,000 platelets per c.mm. The bleeding-time had shortened to six minutes. A tourniquet test was strongly positive. On the following day a few fresh petechiae appeared on the lips and buccal mucosa. These were the last haemorrhagic manifestations to develop during the course of the illness, although the thrombocytes showed little numerical change until December 27 (33 days after admission), when the platelets numbered 30,000 per c.mm. By that date the patient was symptom-free and feeling well.

January 2, 1951 (39 Days After Admission).—The patient was discharged from hospital. A blood count showed a persistence of the thrombocytopenia, although the platelets had increased to 66,000 per c.mm. The total and differential white blood cell counts and the bleeding-time were normal; a tourniquet test was negative.

Follow-up.—A blood count on January 16 showed 164,000 platelets per c.mm., and on January 31 (70 days after the onset of purpura) 215,000 platelets per c.mm. When last examined on September 7, 1951, the patient was in excellent health, his blood picture was normal, and he had had no recurrence of the purpura.

Comment

Laboratory-proved cases of infectious mononucleosis complicated by thrombocytopenic purpura, such as the one reported, are rare. I have found only eight similar cases recorded in the literature (Magner and Brooks, 1942; Lloyd, 1944; Read and Helwig, 1945; Goldbloom and Denton, 1948; Angle and Alt, 1948, 1950; Kutzer and Allen, 1950, two cases; Wallerstein and Madison, 1950). In a further 13 cases a similar diagnosis is probable, although the clinical and haematological evidence is less convincing.

From a study of these 21 case records it would appear that both the purpura and the thrombocytopenia developed during the early stages of the attack of glandular fever. In addition to purpura, a high proportion of the cases showed other haemorrhagic manifestations, most commonly haematuria and/or epistaxis. The thrombocytopenia persisted from one to ten weeks, with an average duration of slightly less than five weeks. Haemorrhagic symptoms persisted for a considerably shorter time, with an average duration of slightly less than two weeks. In at least nine cases recovery occurred without any active form of treatment. In five cases blood transfusions were given, and, although such therapy may have temporarily decreased the tendency to bleed and the bleeding-time, it did not ameliorate the thrombocytopenia. None of the cases gave a previous history of a haemorrhagic tendency; all but one recovered and none recurred. In the fatal case reported by Smith and Custer (1946) death was caused by splenic rupture.

I wish to thank Dr. E. F. Scowen for permission to study this case and Dr. H. F. Brewer for his advice.

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PRODUCTION OF A BLOCKING ANTI-D ANTIBODY BY INJECTION OF D^u RED CELLS

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In 1947 J. J. van Loghem reported that on injecting red cells r'r' and r'r into a donor of group O rr with a view to obtaining anti-C antibody, there had appeared in the serum of the recipient not only anti-C but also blocking anti-D. Race (1947) gave the explanation of this. He proved that the two donors had in fact the D^u antigen on their red cells, and that this antigen was responsible for the appearance of the anti-D antibody.

We have since observed the same result in a series of immunizations of normal male volunteers which were planned with the intention of preparing Rh subgrouping antisera. One of these series of recipients comprised four individuals—Rum., O rr; Lat., A rr; Mas., O rr; and Cusc., B rr—each of whom received twice weekly an injection of 1 to 2 ml. of whole blood from a donor of O group who was thought to be r'r. In fact, this person proved to have a weak D^u antigen in his red cells. Three out of these four sensitized persons gave a reaction and produced an anti-C antibody + anti-D antibody which from the beginning was of a blocking type. A specific D^u antibody did not occur.

The donor Cusc. reacted at the 13th injection, Rum. at the 22nd injection, and Mas. at the 36th injection. The table summarizes our results.

Summary of Results

Date	Serial No. of Injection	Quantity of Blood Injected	Type of Anti-Rh Antibody			
			Early Immune		Hyperimmune	
			Anti-C	Anti-D	Anti-C	Anti-D
Donor Cusc. (B rr group)						
21/7/50 ..	13	2 ml.	0	0	1	¼
10/10/50 ..	16	2 ,,	0	0	¾	⅛
15/11/50 ..	21	2 ,,	0	0	¼	1/16
15/1/51	26	1 ,,	0	0	¼	1/16
Donor Rum. (O rr group)						
21/12/50 ..	22	2 ,,	0	0	1	½
15/1/51 ..	25	2 ,,	0	0	1	¼
Donor Mas. (O rr group)					1	1
21/12/50 ..	36	2 ,,	0	0	1	½
15/1/51 ..	38	2 ,,	0	0		

These results prove that the injection of D^u red cells can stimulate the production of anti-D antibody (as van Loghem has already observed), that this antibody can be of the blocking variety, and that it has been impossible to detect a specific anti-D^u antibody as was considered possible by Knud Eldon (1950).

We wish to thank Dr. J. J. van Loghem for having checked our results.

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Medical Memorandum

Simple Gastric Ulcer with Achlorhydria

Since Schwarz (1910) enunciated the dictum "no acid—no ulcer," there have been a number of reports—for example, by Abramson (1931), Morlock and Ratke (1949), and Heffernon and others (1949)—of undoubted simple gastric ulcers associated with even histamine-fast achlorhydria on at least one occasion. Nevertheless, Palmer, who has written much on this subject in the last 25 years, has recently concluded (Palmer and others, 1949): "Chronic gastric ulcer does occur in patients with transitory achlorhydria and in patients with low secretory ability, but not in complete and permanent achlorhydria. . . . Further evidence of the validity of the concept of 'peptic' ulcer is provided by the invariable healing observed following the appearance of achlorhydria (spontaneous or induced) lasting 90 days or longer, by the very high incidence of healing in phases of achlorhydria of less than 90 days' duration, and by the failure of the ulcer to recur during the period of achlorhydria." The evidence on which this statement is based seems most convincing.

The following case report, although it does not disprove Palmer's contention, provides another example of the association of simple gastric ulcer with more than transient achlorhydria.

Case Report

A 60-year-old housewife was admitted to hospital in May, 1948, after a small haematemesis. She gave a previous history of a "bad stomach" for some 20 years, her main symptoms apparently being "dreadful pain" in the epigastric region not definitely related to food, wind, distension, and nausea. She also stated that even during periods of comparative freedom from her digestive symptoms she could not eat new potatoes, meat unless minced, eggs, oranges, solid puddings, and many other articles because they were too "heavy" and made her vomit. During all this period her home life had been very unhappy because her husband regularly beat her.

Her condition on admission was fair, there were no definite physical signs, and her haemoglobin level was 8.7 g. per 100 ml. Two-hourly feeds alternating with two-hourly aluminium hydroxide emulsion were given and she steadily improved. On the eleventh day the benzidine reaction on her stools was negative; on the twelfth day a barium meal radiograph revealed no significant abnormalities; on the eighteenth day a gruel gastric analysis without histamine showed a complete absence of free hydrochloric acid. Her haemoglobin rose to 13 g. per 100 ml. on discharge. I concluded that she had probably had some form of acute ulceration, her previous history not being very suggestive of chronic peptic ulcer.

She attended the out-patient department on August 29, 1950, and stated that she had remained free from appreciable indigestion after her discharge until five weeks previously, even eating freely of those "heavy" articles which had caused her to vomit in the past. She had then become liable to epigastric pain, again unrelated to meals, associated with a poor appetite, some nausea, and general malaise, though without vomiting. Examination revealed slight but definite tenderness over the upper part of the right rectus abdominis muscle. A barium meal radiograph showed a penetrating ulcer crater on the posterior wall of the stomach above the incisura angularis. Two gastric analyses with histamine were performed at an interval of seven days by the following technique. Nothing was given by mouth after 10 p.m. At 8 a.m. the resting stomach contents were withdrawn and 0.5 mg. of histamine was given (on one occasion subcutaneously, on the other intramuscularly). At 8.30, 9, and 9.30 a.m. specimens were withdrawn. Free hydrochloric acid was always absent on each occasion.

On September 23 Mr. S. C. Raw performed a Hofmeister gastrectomy. In the excised stomach were two almost healed ulcers, ¾ in. (1.9 cm.) in diameter and ¼ in. (0.6 cm.) in diameter, on the posterior wall near the lesser curve. The pathological report by Dr. C. Rickword Lane stated: "The stomach shows two small ulcers on an old healed ulcer-bearing area. Neither shows any evidence of malignancy, nor does the healed area. Both are surrounded by mucous membrane showing acute gastritis." The patient made an uneventful recovery, was discharged on October 15, and has since remained well.

Comment

My reason for reporting this case is not that it is unusual, but because it caused me to change a premise which I had previously accepted—namely, that the association of histamine-fast achlorhydria with a radiologically demonstrated gastric ulcer means carcinoma of the stomach. Admittedly, a patient with this combination of findings must always be considered as a cancer suspect, but if there is other evidence suggesting that the lesion is benign—as there was in the case described—it may be the right policy to refrain from operating for a time, especially as rapid healing of the ulcer can be anticipated if in fact it is benign.

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