MASSIVE HAEMATEMESIS IN A CHILD TREATED WITH PREDNISOLONE

BY

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It is well known that gastro-duodenal complications not infrequently arise during treatment with corticosteroid compounds. Several cases of perforation and haematemesis have been reported in adults, and the incidence of such complications in patients treated for rheumatoid arthritis is said to be as high as 7.5% (Sandweiss, 1954; Bollet *et al.*, 1955). The purpose of this communication is to record the occurrence of a series of massive haematemeses in a child while receiving prednisolone for the treatment of rheumatic fever.

Case History

The patient, who was born in June, 1942, had her first attack of rheumatic fever in 1953 and was treated at home for five months. She then attended school irregularly for two years, during which period she was not really well and complained of shortness of breath. In October, 1955, pain and swelling of her ankles developed, followed by involvement of several other joints. She was put to bed in March, 1956. At the beginning of April she was seen at another hospital because of dyspnoea and inability to lie flat. Tachycardia and an enlarged heart with loud apical systolic and diastolic murmurs were present, but there was no involvement of the joints at the time. She had pyorrhoea, her sedimentation rate was 40 mm. in one hour (Westergren), and a throat swab grew β -haemolytic streptococci. A radiograph of the chest showed enlargement of the heart. After oral penicillin for three weeks her throat swab was clear of streptococci.

On May 19 she was admitted to the Rheumatic Fever Hospital School at Sheffield with a view to prolonged convalescent care. On admission she was a well-nourished, well-developed girl of nearly 14 years of age. She had a tachycardia with a sleeping pulse rate of 86–96 a minute. Her heart was clinically and radiologically enlarged, and loud systolic and diastolic mitral murmurs were heard. At this stage there was no evidence of an aortic valvular lesion. She had several badly carious teeth and her E.S.R. was 49 mm. in one hour.

Four carious teeth were removed under general anaesthesia and intramuscular penicillin cover, and thereafter 125 mg. of phenoxymethyl penicillin was given twice daily to prevent streptococcal infection. During the next 19 weeks there was evidence of a smouldering rheumatic activity and she was kept in bed. Her E.S.R. was persistently high, varying from 28 to 52 mm. in one hour. During this period a soft aortic diastolic murmur gradually developed, her apical systolic murmur became louder, and there was a systolic thrill.

In view of these developments treatment was started on October 5 with prednisolone and salicylates. Her E.S.R. was 38 mm. Treatment consisted of prednisolone by mouth, 20 mg. three times daily for three days, followed by 10 mg. four times daily for 11 days, three times daily for 14 days, and twice daily for a further 35 days, together with "disprin," 10 gr. (0.65 g.) three times daily, and potassium chloride, 2 g. once daily. Her condition was very good during this period; the apical systolic murmur became less loud, the thrill was no longer palpable, and the apical diastolic murmur became inaudible. Her E.S.R. returned to

normal on the 14th day of treatment, but it rose again three weeks later without any apparent cause.

Nine weeks after the beginning of treatment the E.S.R. rose to 31 mm. and she complained of mild abdominal pain. For fear of masking an acute inflammatory condition—for example, appendicitis—prednisolone treatment was discontinued. No complicating illnesses were found while she was off treatment, and as the pain disappeared but the E.S.R. remained high prednisolone was started again a week later in a dose of 40 mg. daily. Mild abdominal pain returned, but she had no abdominal tenderness or guarding and no fever. Her urine was normal. This slight discomfort continued and her E.S.R. remained high (22–30 mm.) without an apparent cause.

On January 18, 1957, an unexpected major haematemesis occurred. She vomited about 1 pint (570 ml.) of bright red blood. As the haematemesis might have been due to either the prednisolone or the disprin, both these drugs were discontinued. Nevertheless a further major haematemesis occurred two days later, to be followed by seven major haematemeses in the next eight days. Several melaena stools were passed during this period. Almost continuous blood transfusions were given—a total of 17 pints (9.6 litres)—either as whole blood or in the form of packed cells (after the bleeding had been arrested). As the bleeding continued for so long, an emergency gastrectomy was contemplated, but as a last measure the procedure suggested by Brandon (1956) was adopted. The stomach was washed out with normal saline and 15 ml. of 1 in 1,000 dilution of adrenaline was left in it after the washout. The procedure was repeated one hour later, and 4 ml. of "stypven" was left in her stomach along with 5 ml. of adrenaline. Her general condition greatly improved and she had no further haematemesis. Her last melaena stool was passed two days later.

Since then progress has been uneventful so far as her gastric condition is concerned, but her E.S.R. was still 40 mm. in one hour on February 21. A barium-meal examination on March 18 showed no evidence of gastric or duodenal ulcer, and her haemoglobin on that day was 10.5 g. per 100 ml.

Discussion

Haematemesis in children is a rare complication even during prolonged treatment with cortisone or its derivatives. During the past few years we have given cortisone or its derivatives to 80 children with rheumatic fever in the Children's Hospital and Rheumatic Fever Hospital School, and to many others with such conditions as the nephrotic syndrome, adrenocortical hyperplasia, and leukaemia, but we have not encountered gastro-intestinal complications in any other child. In a series of 340 children treated for a variety of conditions Good *et al.* (1957) observed two children, aged 2 years, who developed gastro-intestinal bleeding on the 10th day of treatment for the nephrotic syndrome with metacortisone. Goldberg (1954) reported the occurrence of peptic ulcer in a boy of 6½ on cortisone treatment for the adreno-genital syndrome, and Busby *et al.* (1952) observed a bleeding gastric ulcer in a boy of 12 after he received treatment with corticotrophin and cortisone for rheumatic fever.

It therefore seems that, although these complications are rare in children, it might be of advantage to combine routine antacid treatment with cortisone or prednisolone therapy as suggested by Boland (1956).

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