A NOTE ON TWO UNUSUAL CASES OF THROMBOPENIC PURPURA.

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The classification of purpuras has long been a vexed question. The difficulty lies in the belated appreciation that purpura is a symptom and not a disease, while at the same time the word purpura is still retained for use in the classification of those diseases in which it is merely part of the symptom complex. The difficulty would appear to be insuperable with our present very limited knowledge of the underlying pathology. Tidy uses the following classification.

- A. Primary non-hereditary hæmorrhagic diathesis.
 - 1. Purpura hæmorrhagica (acute and chronic);
 - 2. Anaphylactic purpura (Henoch's, rheumatic, etc.).
- B. Secondary purpura from such causes as infections, pyogenic cocci, drugs, etc.

This would appear to be a simple and sound classification. The strictly thrombopenic purpuras are those of the purpura hæmorrhagica group which are usually regarded as idiopathic, but thrombopenia may be found to a marked degree in other severely toxic states affecting the hæmatopoietic system. The term idiopathic is always unsatisfactory.

This is approximately the underlying scheme of classification used by Fowler in a recent survey of some 160 cases of thrombopenic purpura. Much of the value of this critical survey of thrombopenic purpuras is lost in that no details are given of the individual platelet count in these thrombopenic purpuras, either idiopathic or secondary.

There is no constant pathology of either bone, marrow or spleen, so that any attempt at accurate classification based on symptomatology rather than on pathology or on ætiology, while doubtless necessary, must be difficult and may cause confusion.

The essentials in every case of true thrombopenic purpura are 1, a diminution in the platelet count to a figure below 100,000 per c.mm. and usually below 60,000; 2, a prolongation of the bleeding time, while the coagulation time remains normal, and 3, a positive capillary resistance test. These are the essentials, but there is usually fever and a secondary anæmia.

The two cases recorded here are of interest in that they present many unusual features, and they cannot easily or with confidence be placed in any of the usually accepted groups without reservations. There is no doubt that the purpura, the diminution in platelets, and bleeding and coagulation times, were in each case typical of idiopathic hæmorrhagic purpura, the true thrombocytopenic purpura.

On the other hand, there is in each case a very definite double infective focus, the tonsil and the alimentary tract, either of which might be sufficient to exert a profound toxic effect on the host. In the case of the boy improvement followed rapidly on a free evacuation of a loaded colon which was heavily infected with *B. proteus*, etc. He certainly relapsed at a later date with a second tonsillar infection, but the platelet count never went below 200,000 at this time and recovery was complete after tonsillectomy.

In both these cases of thrombopenic purpura very definite foci of toxic absorption were present, identified, and apparently eradicated, resulting in a complete restoration of health.

CASE I. W.M., a female, aged 35 years. First seen on 5.10.36 when she complained of excessive menstrual flow, and generalized bruising.

She was febrile, pale and obviously anæmic, with some bleeding from the gums, and numerous large ecchymoses over the trunk and limbs. The abdomen was quite soft, not tender, and neither liver, spleen, nor glands were palpable. The tonsils were small, fibrotic but obviously infected. The lungs were normal. The cardiac signs were of no significance, apart from a soft hæmic murmur.

She gave a history of general malaise with dyspnœa and palpitation for some five weeks previously. The previous period had been very severe, and there had been "bruising" at that time also. She had had some slight bleeding from the gums and nose.

The bleeding time was markedly prolonged.

Clotting time was normal.

The platelet count was 28,000 per c.mm.

The capillary resistance test was positive.

The blood count was as follows:—

Hb. 53%.

R.B.C. 2,600,000 per c.mm.

C.I. 1.02.

W.B.C. 10,200 per c.mm.

She was admitted to Hospital the next day, and as the platelet count had dropped still further to a practically uncountable 4,000, blood transfusions were given, which resulted in a temporary improvement. The tonsils yielded a pure culture of hæmolytic staphylococcus aureus. The pelvis was examined, and found to be normal.

After prolonged local treatment of the tonsils, combined with blood transfusions and administration of ascorbic acid, the platelet count remained below 50,000 and the hæmorrhagic state remained the same. With a view to hurrying on recovery obliteration of the tonsillar remains by diathermy was under serious consideration, when seven weeks after admission she developed acute abdominal colic over the lower abdomen accompanied by a rise of temperature, and after

retching some blood-streaked vomit was brought up. There were no localizing signs within the abdomen, and no blood in the stool. At this time the platelets practically disappeared. This sharp attack subsided in two days at which time small petechial hæmorrhages were observed for the first time.

Five days later there was another sudden attack of acute abdominal pain, she was obviously distressed, the abdomen was now generally tender on palpation, and both full and resistant, especially in the right iliac fossa. The blood picture was the same. The signs suggested the possibility of an extensive alimentary hæmorrhage, and after much anxious thought and consultation with my colleague, Mr. Rodney Maingot, it was decided that splenectomy should be performed. After this had been done, a rapid examination of the cæcal area revealed a large acute inflammatory mass, an undoubted appendix abscess. The original wound was closed and the appendix abscess drained by a separate incision in the lower abdomen. A blood transfusion was given on the table, and the patient returned to bed in a very fair condition.

The blood picture 48 hours later was as follows:—

Blood platelets 230,000 per c.mm.

Bleeding time half a minute.

Blood count;

Hb. 74%.

R.B.C. 4,000,000 per c.mm.

C.I. 0.92.

W.B.C. 21,000 per c.mm.

After a critical post-operative period she subsequently made an uneventful recovery, is now perfectly fit and well and has resumed her work. Presumably the appendix has sloughed away, but the tonsillar infection has not been cleared up. With regard to the former it is proposed to do nothing at present. If on further examination of the tonsillar exudate a similar hæmolytic organism is still found, it may be considered advisable to obliterate the tonsillar tissue by diathermy rather than by dissection. But if the general health remains good, masterly inactivity may be the wiser course.

CASE II. E.B., a boy, aged 5 years. Admitted to Hospital on 29.9.36. There was a history of a recent fall, with injury to his right eye three days before admission; the trauma had produced a large hæmatoma over his right eye. The following day he was not well, went off his food, and was rather drowsy. The next day he was found to be covered with an extensive purpura, and was admitted to the ward.

Prior to this he had been a normal healthy boy. There was no history of any blood or allergic disease in the family.

On examination he was well nourished but pale, temperature 103.2° F., and pulse 116. The skin of the trunk and limbs was mottled by very extensive purpuric hæmorrhages which extended into the buccal mucosa. There were no palpable glands other than some enlargement of the tonsillar glands. The tonsils were very large and inflamed, and covered by a considerable exudate. There were three carious teeth, and the tongue was coated. There was nothing abnormal with

regard to the heart, and no enlargement of the spleen or liver. The bowels were constipated; an enema produced a very offensive evacuation growing *B. proteus*. A swab taken from the throat grew Vincent's organism.

Bleeding time was over 10 minutes.

The coagulation time was $8\frac{1}{2}$ minutes.

The platelet count was 15,000 per c.mm.

Capillary resistance test positive.

The blood count was:—

Hb. 56%.

R.B.C. 2,640,000 per c.mm.

C.I. 1.05.

W.B.C. 15,000 per c.mm.

A few punctate basophilic red cells were seen. Slight anisocytosis and polychromasia were present.

The boy was evidently profoundly toxic. Hæmoplastic serum was given at once, and later a blood transfusion. The platelet count, however, remained below 50,000. The tonsils were treated with arsenical paint. The alimentary tract was cleared by enema and subsequently by lavage. Ascorbic acid was also given in large doses. There was no improvement in either his general condition or his platelet count until the colon had been well emptied. Within ten days the platelet count had risen to 400,000, and the petechial hæmorrhages had begun to fade. The red blood cells rose to 5,000,000 per c.mm.

Subsequently there was a slight relapse with a recrudescence of purpura, accompanied by a recurrent tonsillitis. This second attack subsided under similar treatment, and some four weeks later the tonsils were removed, after which he made an uneventful recovery. The blood and platelet count, bleeding time, and capillary resistance test were all normal on discharge.