Discussion: The case presented is one in which there was a recurrence of leiomyomatosis following total hysterectomy and left salpingo-oophorectomy. The two most likely reasons for recurrence were incomplete surgical removal of the tumour mass initially or low grade malignancy. There is no conclusive evidence that either of these factors was present, and other possibilities, such as implantation from initial operation, multiple sites of origin of the leiomyomata, or metastasis of histologically benign leiomyomata were considered.

Conclusion

The etiology of this recurrence is uncertain, but the existence of low grade malignancy in these tumours is very difficult to disprove, and the fact that they responded so readily to irradiation and actinomycin-D might suggest the presence of a leiomyosarcoma. Another reason for this effect, however, could be that irradiation had brought about cessation of function in the remaining ovary.

It would seem that in cases of this kind, where surgical removal is technically difficult or impossible, irradiation and actinomycin-D could be a useful form of therapy.

Glanzmann's Thrombasthenia and Pregnancy

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Mrs J T, aged 23

History: First seen at the Antenatal Clinic, Radcliffe Infirmary when 8 weeks pregnant. She gave a history of epistaxes, dental bleeding and excessive bruising, and had previously been told that she was suffering from von Willebrand's disease.

Family history suggestive of a bleeding diathesis. She had two brothers: one died aged 3 months; the other died at the age of 3 years following splenectomy.

She was referred to the Department of Hæmatology where investigations showed that she was suffering from Glanzmann's thrombasthenia.

Investigations: Hb 12·8 g/100 ml. Platelets 300,000/mm^3; WBC 9,200. Bleeding time 15 min; factor VIII normal (85%).

Platelet function tests: Adhesiveness very low, 1·9%; ADP clumping, nil; platelet reaction to thrombin, no clumps formed before fibrin; Hardisty platelet factor III showed platelet defect; clot retraction 20%; prothrombin consumption index 22%; kaolin-cephalin clotting time normal.

Pregnancy: Uneventful until the 38th week when a transient bout of hæmaturia occurred. At the 39th week a further bout of hæmaturia cleared spontaneously. Throughout pregnancy the bleeding time remained prolonged despite a normal platelet count.

Proposed management: It was decided that there should be minimal interference in the management of her labour and that platelet-rich plasma should be transfused during labour and in the puerperium.

Labour: Labour developed spontaneously 5 days past term and the patient had a normal vaginal delivery of a live male infant weighing 8 lb 7 oz (3·8 kg). The first stage lasted 16½ hours and 4 units (800 ml) of platelet-rich plasma was infused. The second stage lasted one hour and a further 4 units (800 ml) of platelet-rich plasma was infused. Episiotomy was performed and after repair the site was manually compressed for 20 min to prevent the formation of a hæmatoma. There was bruising of the vulva and perineum; the bruising increased markedly over the next 24 hours. The third stage was uneventful. Estimated blood loss during delivery was 10 oz (295·7 ml).

An oxytocin (Syntocinon) infusion of 10 units in 500 ml dextrose saline was set up during the third stage. Although the vaginal loss was initially slight, she continued to bleed over the next 24 hours and the total loss at the end of this time was estimated at 81 oz (2,395 ml). During this time she received 4 units (800 ml) of platelet-rich plasma and 4 units (1,200 ml) of fresh blood. The oxytocin infusion was maintained and one injection of 10 units made directly into the uterine muscle. Over the second 24 hours the vaginal loss was slight. A hæmoglobin estimation on the second day was 8 g/100 ml (56%). A further unit (300 ml) of fresh blood and 2 more units (400 ml) of platelets were transfused.

The patient's general condition remained excellent throughout and she received a total transfusion of 14 units (2,800 ml) of platelets and 5 units (1,500 ml) of fresh red cells. Fourteen days after delivery the uterus had involuted normally, the episiotomy had healed perfectly and there was no evidence of bruising.

Baby's progress: Forty minutes after birth marked bruising occurred over the baby's face,

later spreading to the trunk and limbs. The hæmoglobin was 14·6 g/100 ml but no platelets were seen on a blood film. A 30 ml transfusion of platelet-rich plasma was given via the umbilical vein. Ten days after birth the platelet count had risen to 30,000/mm³. No further bruising occurred and the baby continued to thrive. The bruising and absence of platelets in the baby was unexpected, but careful follow up over the next few months should establish whether the thrombocytopenia is temporary or of more serious import.

Discussion

Glanzmann's disease is a rare platelet disorder. The characteristic feature is failure of the platelets to aggregate and adhere, leading to a prolonged bleeding time, abnormal clot retraction and failure of the platelets to clump on addition of adenosine diphosphate.

Two previous cases of Glanzmann's disease have been reported in association with pregnancy. In one case (Roversi *et al.* 1963) the pregnancy, labour and puerperium were all uneventful. There was no excessive bleeding. In the second case (Caen 1966) heavy bleeding occurred from a cervical laceration, necessitating a transfusion of 6,000 ml of blood. Hardisty *et al.* (1964) found that a transfusion of platelet-rich plasma was effective in controlling the bleeding time for 2 hours only. Pittman & Graham (1964) describe their findings in a family of 11 children, 3 girls being affected. All 3 sisters had a history of epistaxes, bruising and menorrhagia, and all 3 had a prolonged bleeding time, poor clot retraction and a positive tourniquet test. Menorrhagia due to Glanzmann's disease can be controlled by progestogen therapy, and Ledermair & Vinnazer (1967) noted some improvement in the hæmorrhagic tendency of their patient following this treatment.

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Immunology in Relation to the Placenta [*Abridged*]

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Trophoblast Extensions from the Placenta

Most studies on the transplantation immunity of the placenta have quite properly concentrated on the resident trophoblast since this tissue appears to play a major role in protection of the fœtus against potential maternal immunological reactions (Currie 1968). Nevertheless, in many mammalian species, and especially in the human, trophoblast is not confined to the placenta and quite substantial amounts are located in various maternal sites during normal pregnancy. These trophoblastic extensions may occur from the early stages of placental ontogeny and throughout the remainder of pregnancy.

Trophoblast giant cells: These are found in large numbers in the decidua basalis and myometrium of the human pregnant uterus. The significance of their existence in these sites is unknown, although Boyd & Hamilton (1960) consider that they could have marked hormonal activity. There is normally no maternal cellular reaction against these foreign cells even though they may persist into the post-partum period. Park (1965) has actually considered the possibility that the *formation* of giant cells may represent a special kind of homograft reaction.

Intra-arterial invasion: During the early phases of human placental development there is a migration of cells from the cytotrophoblastic shell into the terminal portions of the endometrial spiral arteries (Hamilton & Boyd 1966). The only function so far proposed for these cells is the control of blood flow into the intervillous space of the placenta, by plugging the ends of the arteries and reducing their effective diameter. A similar phenomenon has been reported in a number of other species although the fœtal origin of the invading cells has yet to be established. In the pregnant hamster, however, there is an undoubted migration of trophoblast cells into the