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INTRACTABLE UTERINE HAEMORRHAGE DUE TO ABNORMAL SUBENDOMETRIAL BLOOD-VESSELS AFTER CHILDBIRTH AND MISCARRIAGE

BY

R. SALM, M.D., L.R.C.P.&S.Ed.

Consultant Pathologist

AND

P. N. SIMONS, F.R.C.S.Ed., M.R.C.O.G.

Consultant Gynaecologist

Camborne-Redruth Hospital, Redruth, Cornwall

[WITH SPECIAL PLATE]

Two cases of severe and intractable menorrhagia in young women are presented which, clinically, were considered to be functional in origin, as no organic cause was clinically demonstrable. Both patients were finally subjected to the drastic procedure of hysterectomy, as every conservative measure taken had failed to stop the recurrent severe uterine haemorrhages. Detailed examination of the operation specimens revealed the presence of large, thick-walled subendometrial blood-vessels, which were interpreted as being normal radial myometrial vessels, probably arteries, which had originally supplied the placental site, had increased in calibre during pregnancy, but had failed to involute during the puerperium. Erosion of these vessels during menstruation we regard as the cause of the exceptionally severe and recurrent menorrhagia—a cause which, to our knowledge, has not been recorded previously.

Case 1

A woman aged 25 was admitted as an emergency on February 15, 1958, because of sudden, severe, painless vaginal haemorrhage on the second day of a period which had begun normally. She had two children, the first being born in 1954. A miscarriage, concerning which no details were available, occurred in 1956, and in August, 1957, a normal delivery at home was followed by an uneventful puerperium. A few weeks afterwards she had a heavy period with the passage of clots. A regular 28-day cycle was re-established, but menorrhagia continued and was at times severe.

On examination she showed the clinical signs of acute blood loss. Morphine, 1/4 gr. (6 mg.), was given intramuscularly and 1,600 ml. of whole blood was transfused. When her general condition had improved sufficiently a quantity of blood-clot was removed from the vagina. No abnormality could be found in the cervix, uterine body, and adnexae, nor was there any evidence of systemic disease or blood dyscrasia. A full blood-count gave the following results: haemoglobin, 7.4 g. per 100 ml.; red cells, 2,800,000 per c.mm.; total white cells, 7,700 per c.mm., with polymorphs predominating; platelets, 433,000 per c.mm. Bleeding and clotting times were normal.

Radiological examination of the chest and skull revealed nothing of significance. The urine contained no abnormal constituents, and a male-toad pregnancy test gave a negative result. A curettage was performed, and the histological report on the endometrium proved this to be non-secretory in type. Androgen therapy was instituted, and, no further loss having occurred, she was discharged on February 25, 1958.

On March 17 another severe vaginal haemorrhage on the second day of menstruation led to her urgent readmission. Treatment was by blood transfusion, which had to be twice repeated for intermittent and at times heavy loss. A norethisterone preparation was given, and she was well when discharged on April 21.

Her period in May, 1958, was moderately heavy. In June she was once more admitted as an emergency, again with a history of having had flooding on the second day of the menses. Hystero-salpingography had been attempted twice previously, but free bleeding on the entry of the dye into the uterine cavity had made results inconclusive. When this investigation was repeated as soon as practicable the radiological report was as follows:

"The cavity of the body of the uterus presents an abnormal contour. The left lateral angle is not present, and this is suggestive of the presence of a filling defect within the cavity, most probably due to a tumour. No contrast medium has entered the left tube."

In view of these findings laparotomy was considered advisable. On opening the abdomen a diffuse fusiform enlargement of the left uterine cornu was noted. Through a longitudinal incision in the anterior surface of the uterus the cavity was opened and searched for a polypus or submucous fibroid, but none was found. It was decided to remove the left uterine cornu together with the corresponding tube, as it was thought that a small tumour at this site might have escaped detection. Histological examination of the excised tissues revealed nothing significant. The post-operative course was smooth and the patient was discharged on the 14th day.

The next haemorrhage was witnessed in the out-patient department in July, 1958. She was waiting to come into the consulting-room, her period having begun two days previously. Without warning a violent vaginal haemorrhage occurred. The blood saturated her clothing, forming a pool on the chair where she had been sitting and a trail on the floor to the examination couch. She was still bleeding when admitted to the ward, and despite massive blood transfusions her general condition steadily deteriorated. It was decided to remove the uterus as an emergency procedure, as this seemed to offer the only hope of saving her life. A total hysterectomy, which she withstood surprisingly well, was therefore performed with all dispatch. A transfusion of 5,600 ml. of whole blood was given before, during, and after operation, and this added to the 4,000 ml. previously transfused gave a total of 9,600 ml. The patient made an uneventful recovery. When last seen, 13 months after hysterectomy, she was very well and showed no evidence of a haemorrhagic diathesis.

Operation Specimen.—The specimen received was a uterus of average size, measuring $9.5 \times 4.5 \times 3.5$ cm. and weighing 85 g. The right Fallopian tube was attached and was normal. The cervix measured 3.5 cm. in length and the myometrium 1.8 cm. in diameter. The endometrium was smooth and low. The scar of the previous excision was present at the site of the left cornu. On sagittal slicing a brownish discoloured area of pin-point size was found in the subendometrial myometrium some 2 cm. below the operation scar.

Microscopical Examination (58/1718).—The myometrial scar and the brownish-discoloured area below it were cut in two blocks. The section of the former shows myometrium covered by endometrium of intermenstrual pattern. In the

centre of the muscle layer is heavy scarring, incorporating suture material surrounded by a narrow zone of granulation tissue. Below the serosa are ectatic lymph- and blood-vessels and many siderophages. The section from the second shows myometrium covered by endometrium, which is very low in the centre and practically devoid of glands, while its stroma displays, in contrast with the adjacent endometrium, a well-marked decidual reaction, an appearance which is possibly due to marked localized oedema. Here the myometrium bulges upwards in semilunar fashion and contains a single, coiled, large-calibre blood-vessel which runs more or less perpendicularly from the outer myometrium towards the endometrium (Special Plate, Fig. 1). Its thick wall is devoid of muscle and elastic fibres, while its lumen, in spite of an oedematous, proliferated intima, is large and filled with blood. At the endometrial border it gives off some smaller branches, but the main branch continues into the thin, aglandular endometrium just described. Here its wall becomes oedematous, thin, disrupted, and disorganized, while its upper segment is surrounded by a wide crescent of fibrinoid necrosis (Special Plate, Fig. 2), in itself proof of recent extravasation of blood. Further sections cut from this block demonstrate clearly that the wall of this vessel had recently burst, producing a complete breach in the endometrium, though the resulting gap in the endometrium as well as the gap in the vessel wall had already become sealed off and blocked by a comparatively large fibrin-platelet mass (Special Plate, Figs. 3 and 4). The edges of the gap in the endometrium display some epithelization due to downward proliferation of the surface epithelium.

Case 2

A single woman aged 24 was admitted as an emergency on August 11, 1958, because of sudden severe vaginal haemorrhage on the third day of menstruation. In December, 1957, she had a miscarriage at home, and her periods, although regular, had subsequently been heavy and prolonged with premenstrual dysmenorrhoea. On examination she was found to be shocked and collapsed with a weak, rapid pulse and a blood-pressure of 80/40 mm. Hg. Only slight vaginal loss was occurring at this time, and inspection of the cervix showed this to be normal with a little bleeding from the external os. The uterine body was found to be anteverted and a little larger than normal, and both uterine appendages were palpable and somewhat tender. A provisional diagnosis of placental polypus and subacute salpingitis was made. She was given a transfusion of 1,600 ml. of whole blood and intramuscular injections of morphine, 1/4 gr. (16 mg.), and ergometrine, 0.5 mg. When her general condition had improved sufficiently a curettage was performed. Endometrium of non-secretory type was removed, but there were no placental villi. Her post-operative course was uneventful and she was discharged on August 20.

She was urgently readmitted on September 10, 1958, with a history of colicky lower abdominal pain accompanied by the passage of clots per vaginam, estimated as "about a jugful." Her period, which had begun on September 5, had been continuing normally until the sudden onset of pain and haemorrhage a few hours before her admission. When first seen her general condition was satisfactory and bleeding had ceased. No abnormality was found on abdominal or pelvic examination. A full blood-count showed no evidence of blood dyscrasia or defect of coagulation. The haemoglobin level was 10 g. per 100 ml. It was decided to perform a second curettage, as it was thought that a small placental polypus might have been missed previously. On dilating the cervix, blood was seen to be spurting through the internal os, and at the time this was thought to be due to lacerations high in the cervical canal resulting from too rigorous dilatation. No bleeding vessel being visible, the uterine cavity and cervical canal were packed

with gauze. As before, non-secretory endometrium was found on histological examination.

Intermittent bleeding, on the whole not profuse, occurred during the next week and was followed by a sudden severe haemorrhage necessitating the transfusion of 800 ml. of whole blood. Four days later a similar transfusion was required for another heavy loss. She was given a preparation of norethynodrel, and discharged on September 30 with instructions to continue treatment at home.

On November 30, 1958, she was admitted for the third time, again as an emergency and again with heavy loss on the third day of a period. Episodes of severe vaginal bleeding recurred, and she suffered a steady deterioration despite repeated blood transfusions. Total hysterectomy was therefore performed on December 19, from which she made a good recovery. On this occasion 4,000 ml. of whole blood was transfused, and this together with the 3,200 ml. previously administered gave a total of 7,200 ml. The patient, when seen again eight weeks after operation, was in perfect health and there were no signs of a haemorrhagic diathesis.

Operation Specimen.—The specimen was a small uterus measuring $7 \times 4.5 \times 3.5$ cm. and weighing 44 g. The cervix measured 2 cm. in length and the myometrium 1.5 cm. in diameter. The endometrium was of average height. On sagittal slicing a tiny brownish-discoloured area was noted situated in the subendometrial myometrium of the lower third of the uterine body.

Microscopical Examination (58/2721).—The entire brownish discoloured area with surrounding tissues was cut by serial step sections in four blocks; the presence of an arterio-venous aneurysm could thereby definitely be excluded. The myometrium, bulging slightly upwards in the centre, is covered by low endometrium of intermenstrual type. In the centre it is very low and practically devoid of glandular tubules (Special Plate, Fig. 5). A large-calibre vessel is coursing in perpendicular fashion through the myometrium towards this area (Special Plate, Fig. 6). It displays a thick, fibrous, oedematous wall which is devoid of elastic fibres and very poor in muscle elements. A short distance below the endometrial border the intimal surface bears several small mural thrombi (Special Plate, Fig. 7) indicative of recent haemorrhage. The outer myometrium contains a vascular plexus and distended capillaries and lymph vessels.

Discussion

It is often difficult to exclude organic disease in cases of functional, or, as some prefer to call it, dysfunctional, uterine haemorrhage. In the cases under discussion a clinical diagnosis was not possible, though every effort was made to establish a basis for effective therapy. As the bleeding followed childbirth in one case and miscarriage in the other, pelvic congestion and infection, placental polypus, subinvolution, and chorion epithelioma were first considered, but later excluded. A physician's advice was asked regarding an underlying systemic cause, but none was discoverable. Possible psychogenic causes were fully considered. The first patient and her husband were interviewed at home in the presence of their family doctor; the second was seen by a psychiatrist while in hospital. The results were of little value.

The cause of the exsanguinating menorrhagia remained unexplained until pathological examination of the excised uteri revealed in both cases the presence of a comparatively large thick-walled blood-vessel coursing in radial fashion from the vascular plexus in the outer myometrium towards the endometrium, giving off

smaller branches below it in Case 1, and thus corresponding exactly to the known pattern of the uterine arteries, which have been designated arcuate myometrial, radial myometrial, subendometrial basal, and endometrial coiled arteries respectively. In the present cases, however, these vessels had undergone considerable pathological changes: they were almost totally defective in muscle and elastic fibres, while fibrous tissue and intimal layers had noticeably increased. Thus it was no longer possible to determine histologically whether the abnormal vessels were arterial or venous branches. Considering the rapidity with which each patient became exsanguinated, they may well have been small arteries, but in our opinion this is not of fundamental importance. Whether arterial or venous, the vessel observed in each case was grossly abnormal in comparison with its normal counterpart (see Novak and Novak, 1958).

These abnormal radial myometrial blood-vessels had in both instances been bleeding recently, for which we have adduced histological proof, and we regard them as enlarged, persistent blood-vessels of the placental bed which had failed to involute in the normal way. That such blood-vessels may persist after delivery owing to failure of the physiological process of obliteration has been proved by Rutherford and Hertig (1945). The fact that the severe menstrual bleeding had followed a normal pregnancy in Case 1 and a miscarriage in Case 2 is strong support for this thesis. In each case the uncontrollable bleeding started on the second or third day of the period. It is just about that time that the shedding of the upper endometrial layers would expose these large-calibre, abnormal basal blood-vessels which had been bleeding previously, and any thrombotic occlusion of their stumps could be expected to be removed by fibrinolysis and vascular congestion, thus giving rise to the sudden uterine haemorrhages. This process would be accentuated by the thickened and rigid diseased vessel wall, which almost entirely lacked elastic and muscle fibres, and thus no longer possessed the ability to contract or to be easily compressed by uterine contraction.

The misleading radiological report in Case 1 was most probably due to blood-clot which had accumulated at the site of the bleeding, and was thus responsible for the filling defect observed.

Macroscopically, the site of the bleeding had been characterized in each instance by brownish discoloration which, in conjunction with the clinical history, was so typical that the nature of the lesion was immediately suspected in our second case. It should be noted, however, that the brown colour observed was due to the action of formalin, and not to breakdown products of haemoglobin. In unfixed specimens the colour might be expected to be red.

We do not regard the mechanism recorded in this paper as the explanation of many post-abortion or post-puerperal menorrhagias. On the contrary, on the basis of our experience we regard this as a very infrequent and exceptional occurrence. Moreover, it may well be that in some cases of this type spontaneous cure will eventually result. But we are of the opinion that in a small number of intractable menorrhagias occurring after childbirth or miscarriage the mechanism advanced by us will be found to be the explanation.

Summary

Two cases of recurrent exsanguinating menorrhagia following childbirth and miscarriage, which finally had to be subjected to hysterectomy, are described.

On pathological examination the cause for the severe uterine haemorrhages was found to be a surviving large-calibre vessel of the placental bed, possibly an arterial branch, which had failed to undergo involution in the normal way during the puerperium.

To the authors' knowledge this syndrome has not been described previously.

We would like to express our gratitude to Dr. R. Pollok for allowing us to include his case as Case 2 in this paper, and to Professor R. A. Willis for reading the manuscript. We are indebted to Miss Phyllis E. Coleman for the photography.

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HAEMOGLOBIN "SINGAPORE-BRISTOL"

A "FAST" HAEMOGLOBIN FOUND IN INFANTS

BY

A. B. RAPER, M.D., M.R.C.P.
Royal Infirmary, Bristol

J. A. M. AGER, M.B., B.S.
St. Thomas's Hospital, London

AND

H. LEHMANN, M.D., Sc.D., M.R.C.P., F.R.I.C.
Department of Pathology, St. Bartholomew's Hospital,
London

At a symposium on abnormal haemoglobins held in Istanbul in 1957, Fessas (1959a) described a "fast"-moving haemoglobin which he had seen in an infant but not in its parents. The component disappeared from the child's blood when it grew older. This observation was interpreted by Fessas as being possibly the first demonstration of an abnormal foetal haemoglobin. Further details were published by Fessas and Papaspyrou (1957). Also in 1957 Vella (personal communication) noted in Singapore that some cord bloods of Chinese infants contained a fast-moving haemoglobin which could not be demonstrated in the blood of either parent. Early in 1958 he sent specimens to Fessas in Athens and to us in London. Fessas considered that this haemoglobin variant was the same as that which had been seen by him and his colleagues in the Greek infant. At that time Ager and Lehmann (1958) investigated a haemoglobin found in an infant at St. Bartholomew's Hospital. This haemoglobin differed from that sent by Vella and from that described by Fessas and Papaspyrou (1957). It was decided to call this pigment haemoglobin "Bart's," and to refer to the other not yet named abnormal foetal haemoglobin as the "haemoglobin of Fessas and Papaspyrou" ("F & P").

The principal differences between the two haemoglobins were that Bart's moved on paper electrophoresis at alkaline pH in front of haemoglobin J, whereas F & P did not, and that Bart's was resistant to alkali denaturation in the "one-minute alkali denaturation test" of

Singer, Chernoff, and Singer (1951) and by other alkali-denaturation procedures, whereas F & P was denatured as haemoglobin A.

A difficulty in nomenclature arose in 1958 when Fessas (personal communication) found more cord bloods and bloods of infants with a fast-moving haemoglobin which had all the properties of haemoglobin Bart's, and was led to the conclusion in retrospect that the first example of an abnormal foetal haemoglobin seen by him in 1957 had also been of this type rather than of that which had meanwhile been referred to as F & P. Nevertheless, there existed undoubtedly two types of fast-moving haemoglobins in cord blood of infants whose parents showed no abnormal haemo-

FIG. 1.—Comparison of a haemoglobin A+J control (middle) with the haemoglobins of Case 1 (right) and Case 2 (left). The fast-moving component in Cases 1 and 2 moves slightly less fast than J. Electrophoresis on cellulose acetate with barbiturate buffer pH 8.6. Strip stained with light green.

globin, one moving on paper electrophoresis at pH 8.6 faster and one moving slower than haemoglobin J (Vella, 1959).

We here record observations on a haemoglobin variant seen in two families in the offspring but not in their parents, which moved on paper electrophoresis at pH 8.6 in front of and separating from haemoglobin A but behind the position occupied under this condition by haemoglobin J (Fig. 1).

Case 1.—B.B. presented with anaemia at the age of 2½ years (under the care of Professor C. B. Perry). The spleen could not be felt. The haemoglobin level was 7.4 g./100 ml., and the smear showed considerable hypochromia, anisocytosis, and target cells. There was a satisfactory response to treatment with iron by mouth. The haemoglobin was examined on three occasions, and each time showed a fast-moving component on electrophoresis on paper and cellulose acetate at pH 8.6. Alkali-resistant haemoglobin (Singer *et al.*, 1951) 5%. **Family:** Father of French, Portuguese, and East Indian extraction; no haematological abnormality; haemoglobin on electrophoresis shows A only; alkali-resistant haemoglobin 1.7%. Mother English; no haematological abnormality; haemoglobin on electrophoresis shows A only; alkali-resistant haemoglobin 1.7%.

Case 2.—E.T. presented with anaemia at the age of 2½ years (under the care of Dr. J. Apley). The spleen was enlarged three fingerbreadths. Haemoglobin level was 7.4 g./100 ml.; P.C.V., 31.5%; R.B.C., 4,400,000/c.mm.; M.C.V., 72 cubic microns; M.C.H.C., 26%. Serum iron 23 µg./100 ml.; serum bilirubin, 0.35 mg./100 ml. The tibial marrow was normoblastic, with evidence of iron deficiency.

R. SALM AND P. N. SIMONS: UTERINE HAEMORRHAGE AFTER CHILDBIRTH

FIG. 1.—Case 1. Low-power view showing myometrium bulging upwards, incorporating large, thick-walled, coiled radial vessel. Aglandular endometrial lining on top. (Haematoxylin and eosin. $\times 8$.)

FIG. 3.—Case 1. Inset of Fig. 1 at higher magnification. Endometrial tear with ruptured endometrial vessel plugged by fibrinous clot. Aglandular endometrium to either side displaying decidual reaction. Beginning epithelization of upper margins of defect. (Haematoxylin and eosin. $\times 32$.)

FIG. 6

FIG. 7

FIG. 2.—Case 1. Ruptured vessel at base of endometrium, surrounded by eosinophil crescent of fibrinoid necrosis. (Haematoxylin and eosin. $\times 60$.)

FIG. 4.—Case 1. Detail of ruptured vessel. Fibrin clot at top. (Haematoxylin and eosin. $\times 60$.)

FIG. 5.—Case 2. Low aglandular endometrium in centre with large, thick-walled, distended blood-vessel underneath. (Haematoxylin and eosin. $\times 24$.)

FIG. 6.—Case 2. Low-power view of large-calibre radial myometrial vessel. Myometrium bulging slightly upwards, covered by low endometrium. (Haematoxylin and eosin. $\times 6$.)

FIG. 7.—Case 2. Mural thrombus (arrowed in Fig. 6), partly covered by endothelium, superimposed on thick, fibrous vessel wall. (Haematoxylin and eosin. $\times 50$.)