Splenic Vein Thrombosis with Oesophageal Varices:

A Late Complication of Umbilical Vein Catheterization

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On the basis of observations made on three infants, a description is given of a late complication of umbilical vein catheterization not hitherto reported. The children showed the symptoms of thrombosis of the splenic vein with secondary splenomegaly and marked gastric and/or esophageal varices, while the portal vein showed no abnormality. The diagnosis was preoperatively established by means of selective angiography of the superior mesenteric artery and the splenic artery. Treatment in these three cases consisted of splenectomy, with good clinical and radiological results.

THE CAUSE OF ESOPHAGEAL VARICES is usually found in excessively high pressure within the venaportal system as a result of intrahepatic or extrahepatic obstruction in this system.

However, esophageal varices can also develop in the presence of an entirely patent portal vein, if the efferent flow through the splenic vein is obstructed while the supply through the splenic artery remains normal. This results in congestion in the spleen, which must drain itself of its blood through collateral veins. The consequence is an enlargement of the spleen and varicose dilatation of the veins between the spleen and the stomach with, in a later stage, dilatation of the veins in the cardia and the esophagus as well. Ultimately this leads to the classical features of (possibly bleeding) esophageal varices (Fig. 1).

Thrombosis of the splenic vein is a rare abnormality. In children, such an obstruction of the splenic vein may possibly be due to the use of an umbilical vein catheter. There are reports on complications of umbilical vein catheterization such as perforation of the vascular wall, thrombosis of the portal vein, liver infarction, pulmonary embolism and colon perforation.[1,6,8–11]

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The possibility of a relationship between isolated thrombosis of the splenic vein with esophageal varices and the use of an umbilical catheter immediately after birth, can be illustrated by the case histories of three patients whom we treated in June and July 1973.

Case Reports

Patient A was a girl born on November 22, 1970 after a pregnancy of 31 weeks; birth weight 1400 g. After birth she was given a 10% glucose solution through an umbilical vein catheter which was removed after 72 hours. The position of the catheter was not radiologically controlled.

After a followup period of 6 months during which development was uneventful, she was discharged from the hospital.

About two years later, on April 17, 1973, she was admitted to the Diaconessen Ziekenhuis, Groningen with anemia (hemoglobin 7.7 g/100 ml; hematocrit 21), hematemesis and splenomegaly. X-rays of the stomach disclosed filling defects in the fundus. After an abundant intestinal hemorrhage, exploratory laparotomy revealed a normal liver and a greatly enlarged spleen; markedly dilated veins were observed against the posterior abdominal wall, in the splenocolic ligament and around the fundus of the stomach. A biopsy specimen from the liver was normal. The possibility of portal vein thrombosis with hemorrhage from gastric or esophageal varices was considered. No further surgical treatment was carried out. The bleeding ceased spontaneously. The postoperative radiograph of the esophagus revealed extensive varices (Fig. 1).

On June 4, 1973 the child was admitted to the Groningen University Hospital for further diagnostic work.

The aortogram showed no abnormality. Selective angiography of the superior mesenteric artery was likewise negative, and during the venous phase the efferent flow through the portal vein was seen to be adequate. The selective angiogram of the splenic artery showed no abnormality of the artery itself but re-

vealed marked splenomegaly. In the venous phase of this selective angiogram a complete stop was observed in the splenic vein, immediately central to the entry of the vena coronatia gastrica. The contrast medium was drained off through this vein and through varicose veins along the gastric fundus, cardia and esophagus.

In view of this finding, splenectomy was performed on June 9, 1973 in an effort to abolish the pressure in the varicose collateral system. In view of the risk of bleeding from the varices, the splenic vein in the upper edge of the pancreas was not dissected free in a central direction, and the site of obstruction was therefore not brought into view. The spleen weighed 80 g. The histological diagnosis was splenomegaly.

The postoperative course was uneventful. The child received prophylactic antibiotic medication.

Followup radiography of the esophagus on July 25, 1973 (more than six weeks after the operation) revealed unmistakable regression of the esophageal varices, even though they had not yet disappeared completely.

Patient B was a boy born on 10th April 1967 after a pregnancy of 31 weeks; birth weight 1200 g. After birth he was given a 10% glucose solution through an umbilical vein catheter. The period during which this remained *in situ* is unknown. The child developed uneventfully. Routine followup, however, revealed gradual enlargement of the spleen. Blood morphology and chemistry were normal. Scanning disclosed a small liver and a greatly enlarged spleen. Radiographs of the long bones showed juvenile bone cysts; a diagnosis confirmed by biopsy. Radiographs of the stomach and the esophagus showed unmistakable varicose filling defects.

The aortogram showed no abnormality. Selective angiography of the superior mesenteric artery was likewise negative; during the venous phase the efferent flow through the portal vein was seen to be adequate. The selective angiogram of the splenic artery showed that this artery was patent, but the splenic vein failed to fill. The greatly enlarged spleen drained itself through varicose veins along the stomach and the esophagus.

In view of this finding, splenectomy was performed on July 2, 1972 in an effort to abolish the pressure on the varicose veins. The spleen weighed 300 g and showed marked local fibrosis. At the site of the hilus the splenic vein was patent, but at a more central level it was obliterated. Extensive varices were present along the stomach and the left dorsolateral abdominal wall.

The postoperative course was complicated by a protracted subfebrile temperature, which disappeared spontaneously. The boy was considered too old for prophylactic antibiotic medication.

Followup radiography of the esophagus on August 8, 1973 (5 weeks after the operation) showed unmistakable regression of the esophageal varices, and normal plication of the esophageal mucosa began to be visible again.

Patient C was a boy born on August 16, 1972 after a pregnancy of 34 weeks; birth weight 1750 g. In view of hypoglycaemia he was given a 10% glucose solution through an umbilical vein catheter during 24 hours after birth. The position of the catheter was not radiologically controlled. The child's development was uneventful, and after 6 weeks he was discharged from hospital. Gradual enlargement of the spleen was observed in the course of the followup. Blood morphology and chemistry were infectious mononucleosis were negative. Esophageal radiographs showed no varices. Urinalysis and IVP were negative. After hospitalization for further investigation the child's hemoglobin concentration fell to 8.8 mg/100 ml; the leucocyte count fell to 1600/mm³ and the platelet count to 90,000/mm³. The bone marrow punctate was normal. On June 26, 1973 he was admitted to the Groningen University Hospital.

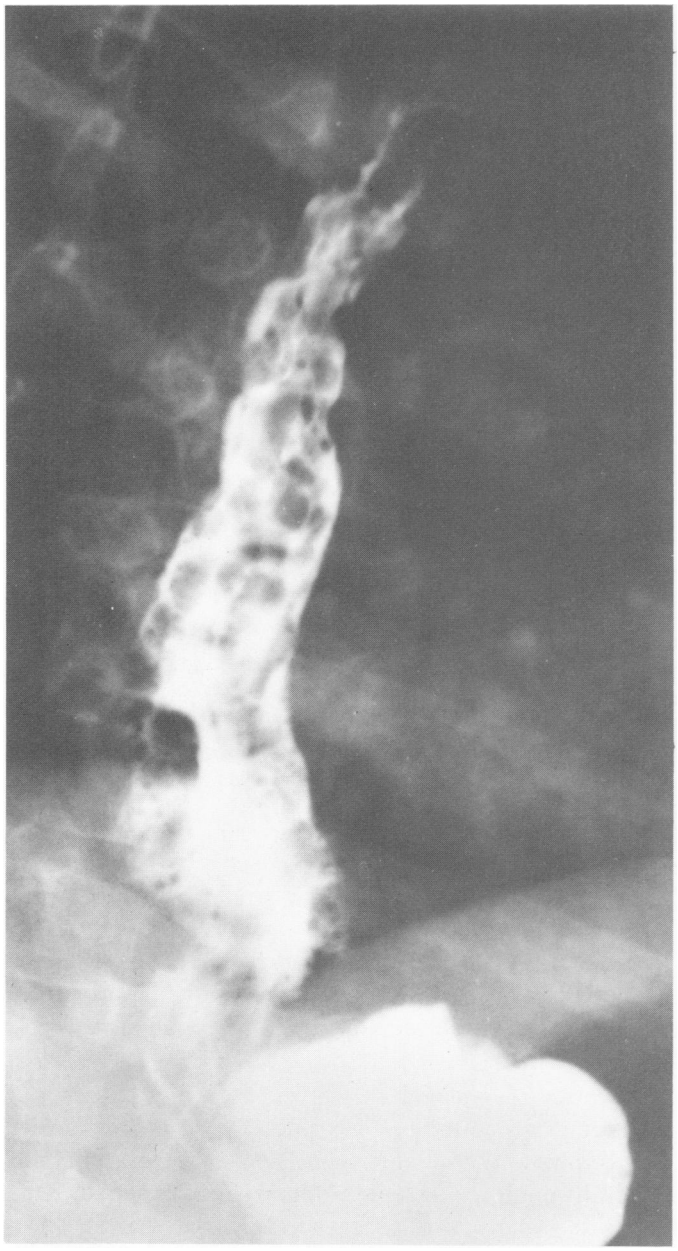

FIG. 1. Unmistakable filling defects in the esophageal radiograph, caused by esophageal varices in a 30-month-old girl with splenic vein thrombosis resulting from umbilical vein catheterization.

The aortogram showed no abnormality. Selective angiography of the superior mesenteric artery likewise failed to show any abnormality; during the venous phase an adequate efferent flow through the portal vein was seen (Figs. 2 and 3). Selective angiography of the splenic artery showed that this was also normal, but during the venous phase a stop was visible in the splenic vein immediately central to the entry of the vena coronaria gastrica (Figs. 4–6). The congested spleen drained itself through varicose veins along the fundus of the stomach.

On the basis of this finding, splenectomy was performed on July 16, 1973, in an effort to abolish the high pressure in the collateral venous system. Extensive varices were found along the

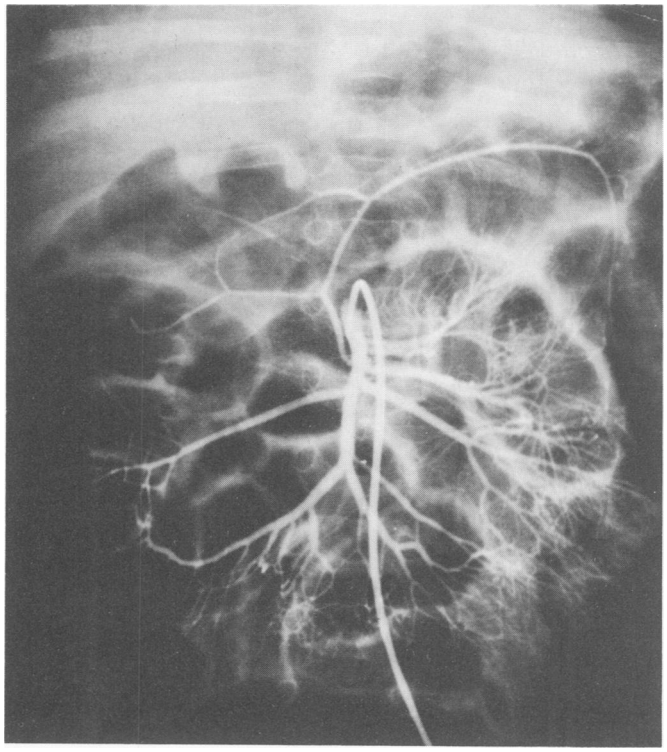

FIG. 2. Normal arterial vascular pattern in selective angiogram of the superior mesenteric artery in an 11-month-old boy with splenic vein thrombosis resulting from umbilical vein catheterization.

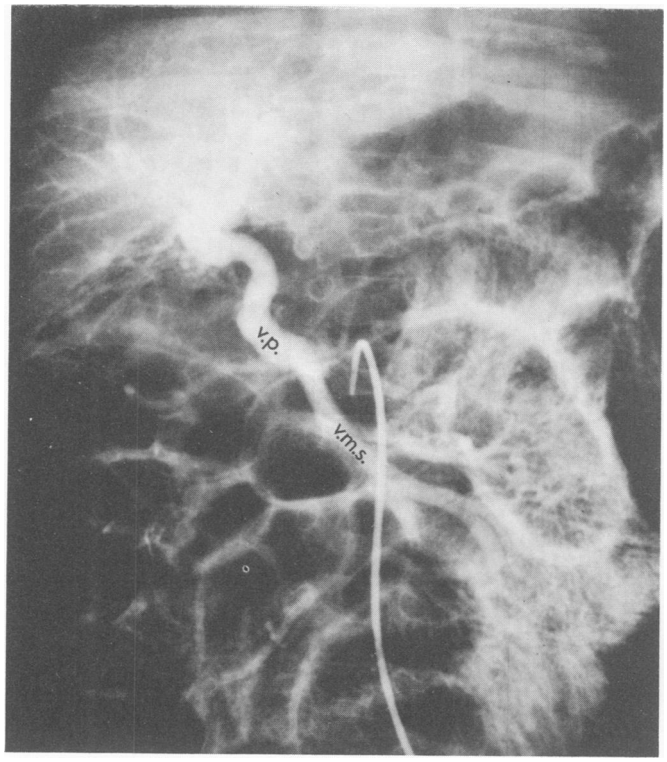

FIG. 3. The venous phase of the selective mesenteric angiogram of Fig. 2 shows adequate efferent flow through the portal vein. (v.p. = vena portae, v.m.s. = vena mesenterica superior.)

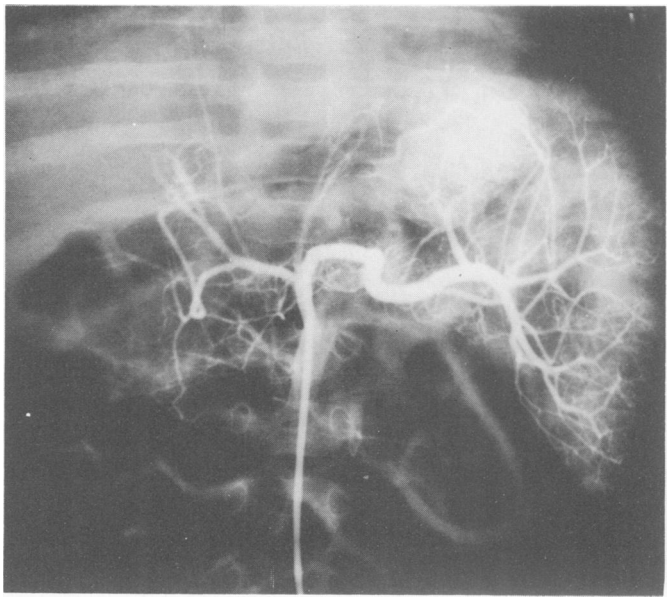

FIG. 4. Selective angiogram of the splenic artery of the same patient (Figs. 2 and 3). Normal splenic artery. Enlarged spleen.

stomach and the posterior abdominal wall. In view of the hemorrhagic risk, the splenic vein was not dissected free from the pancreatic edge, and the site of the venous stop was therefore not brought into view. The spleen weighed 105 g and at histological examination showed the features of chronic congestion with unmistakable diffuse fibrosis. The postoperative course was uneventful. The patient was given a prophylactic maintenance dose of antibiotics until he reaches age 5.

These three children were prematures (31 weeks/1400 g; 31 weeks/1200 g; 34 weeks/1750 g), who as neonates received a 10%

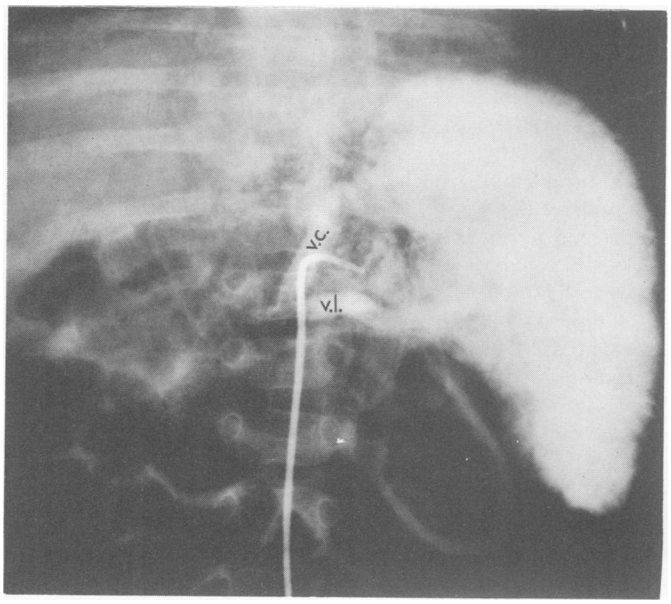

FIG. 5. The venous phase of the selective splenic artery angiogram of Fig. 4 shows an unmistakable stop in the splenic vein; efferent flow through collaterals along the fundus of the stomach. (v.l. = vena lienalis. v.c. = vena coronaria gastrica.)

glucose solution through an umbilical vein catheter. All three gradually developed splenomegaly with varices of the stomach and/or esophagus which were slight in the youngest child (patient C, aged 11 months), but already very severe in the two older children (patients A and B, aged 30 months and 6 years, respectively).

By means of selective angiography of the mesenteric and splenic artery, a patent portal vein and an obstructed splenic vein were demonstrated in these children, in whom the blood from the congested spleen was drained through varicose gastric and/or esophageal veins. Treatment consisted of splenctomy in these three cases, and with good results.

It is justifiable to postulate a relationship between the umbilical vein catheterization and the development of splenic vein thrombosis with esophageal varices in these three cases. So far as we could establish from the literature, this complication of umbilical vein catheterization has not been previously described.

Discussion

The umbilical vein is localized in the free edge of the falciform ligament, and opens up into the left half of the portal sinus (Fig. 7). Opposite its point of entry the ductus venosus arises from the portal sinus. The ductus venosus empties into the left hepatic vein immediately before the latter opens up into the inferior vena cava. Experiments by Soeters[7] on neonatal cadavers in which he catheterized the umbilical vein under radiological control showed that it was sometimes difficult to cannulate the ductus venosus. In these cases the catheter deviated to the left or the right branch of the portal vein.

Anyone who has ever catheterized an umbilical vein under fluoroscopic control, knows how readily the catheter deviates to the left and enters the splenic vein. In the absence of fluoroscopic control there is a ready risk that a solution is infused into the splenic vein through such an ill-placed catheter.

This creates a situation which is virtually ideal for the development of splenic vein thrombosis. The catheter invariably causes slight-to-severe traumatic intimal lesions," And the fluid infused (e.g. a 10% glucose solution) may produce an additional chemical intimal lesion.[9]

A factor which may play an important additional role in the etiology of the intimal lesion is an infection introduced via the catheter. In umbilical artery catheterization, this factor seems of less marked importance.[3] As a result of the intimal lesions, swelling and degranulation of blood platelets give rise to the release of adenosine diphosphate (ADP). The ADP in turn promotes platelet aggregation. In normal situations this process abolished by a rapid flow of blood, but this cleansing mechanism is rendered less effective by the presence of a catheter which is relatively thick as compared with the lumen of the splenic vein (particularly in prematures). Spasm of the vein in response to the intimal irritation further reduces the blood flow rate. And we know that stasis is an important factor in the aetiology of thrombosis.

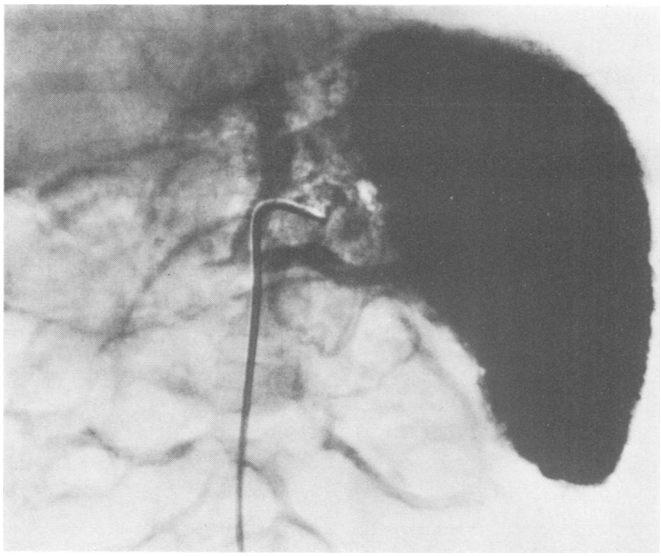

FIG. 6. Subtraction of the angiogram of Fig. 5. Enlarged spleen and stop in splenic vein are clearly visible. The catheter is still in the splenic artery.

When the aetiological factors persist (intimal lesion and stasis), the original platelet coagulum is transformed to a real thrombus by fibrinogenesis. Hoshal *et al.*[4] observed fibrinogenesis within 24 hours when using subclavian catheters, whether they be polyethylene, Teflon nylon or silicone rubber catheters. It was a conspicuous finding that polyethylene catheters pre-treated with graphite-benzalkonium chloride-heparin sodium (GBH) unmistakably gave less fibrinogenesis in this study. Whether this also applies to the much narrower splenic vein in neonates, however, remains a moot point.

Conclusion

Mechanical, chemical and infectious lesions of the intima, combined with a change in the blood flow rate, would seem to be the principal causes of the development of thrombosis following the use of an intravenous catheter.

Although there can be no doubt about the diagnostic, and particularly the therapeutic value of umbilical vein

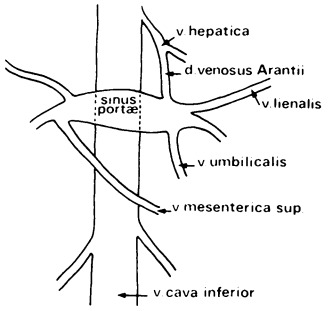

FIG. 7. Diagram of the neonatal portal system.

catheterization, the technique is not without its dangers. There are several possible complications. One such complication, hitherto unknown, is localized thrombosis of the splenic vein followed by splenomegaly and severe gastric and/or esophageal varices, as described in the above case reports. Since selective angiography of the mesenteric artery and splenic artery has become possible also in very young infants (Figs. 2–6), an exact preoperative diagnosis can be made. Treatment is technically simple: splenectomy. Perhaps it is advisable to confine surgery in infants to ligation of the splenic artery without splenectomy, with a view to the development of immunity.[2,5]

However, prevention is bettter than cure. The use of intravascular catheters, particularly in neonates, should be confined to cases which afford no alternative. If catheters have to be used, their calibre should be as small as possible; they should be very flexible in order to avoid fibrinogenesis (GBH-treated polyethylene catheters possible open new perspectives in this context). Their introduction requires strict asepsis, and the position of the catheter should always be fluoroscopically controlled. Infusion fluids which irritate the vascular wall must be avoided as much as possible. All intravascular catheters should be removed as soon as possible. It should always be borne in mind that, by introduction of an intravascular catheter, we place a time bomb inside the child.

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