

Preduodenal Portal Vein Causing Duodenal Obstruction with Bleeding Duodenal Ulcer *

A Case Report

LOUIS J. BERNARD, M.D., FRANK A. PERRY, M.D., F.A.C.S.,
MATTHEW WALKER, M.D., F.A.C.S.

From the Department of Surgery, Meharry Medical College, Nashville, Tennessee

CONGENITAL malformations in the portal venous system are uncommon. Preduodenal position of the portal vein is rare. In the American literature Snavely and Breakell¹ record one case in which two separate portal veins passed over the anterior surface of the second and third parts of the duodenum and the head of the pancreas, joined together in an anastomotic loop at the level of the lower border of the duodenum. Because of stenoses in the portal system, their patient developed portal hypertension with subsequent demise from esophageal hemorrhage. In the German literature Stengel² reported two cases of preduodenal position of the portal vein with normal position of the stomach and duodenum. She stated that Schnitzler had encountered a similar case at operation in 1926, and that Lehmann had verbally reported another case to her professor, Pernkopf. She discussed a few previously reported cases of preduodenal position of the portal vein associated with situs inversus or malrotation of the stomach and small intestine. In view of the rarity of the lesion and its implications, a recently encountered preduodenal portal vein associated with a minor degree of malrotation of the gut is here reported.

Case Report

G. J., a 48-year-old Negro female, was admitted to the medical service of Hubbard Hospital on January 29, 1958, because

* Submitted for publication November 24, 1958. Revised April 23, 1959.

of epigastric pain with hematemesis and melena. Her illness had begun 48 hours previously with dizziness, nausea, anorexia and vomiting of clear fluid, dark stools and hematemesis first appearing shortly prior to admission. The patient was a known diabetic of seven years who had ceased taking insulin on her own accord about one month prior to admission.

On admission the temperature was 37.9° C., pulse 120, and respiration 18, with systolic blood pressure 150 and diastolic 110. Shortly after admission the blood pressure dropped to 98/70 and the patient became listless and uncooperative. Stools were dark and positive to hematest. Urinalysis showed ++ albumin, +++ sugar and was strongly positive for acetone. The hemoglobin was 12.99 grams% with 37% hematocrit. The blood nonprotein nitrogen was 37 mg.%, sugar 367 mg.%, and carbon dioxide combining power 16.8 meq./l. Plavolex 500 cc. and whole blood 650 cc. were given in addition to 2,000 cc. crystalloids with insulin with marked improvement in the patient's condition. No further active bleeding was evident until 5:00 p.m. on January 30, at which time the patient vomited 800 cc. of blood. This amount of blood was promptly replaced. On the following day an upper gastro-intestinal x-ray series was done and is shown in Figure 1. Surgical intervention was deemed necessary and on the evening of January 31, 1958, laparotomy was carried out.

At operation, the duodenal bulb was

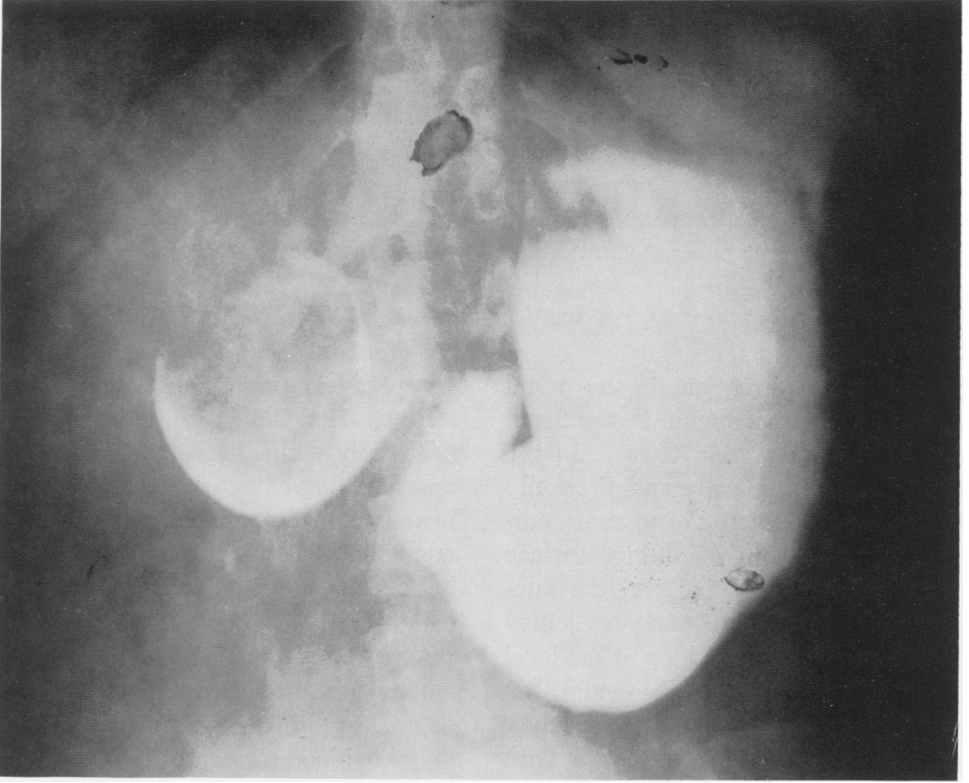


FIG. 1A. Preoperative upper gastro-intestinal x-ray showing apparent marked distention at pylorus.

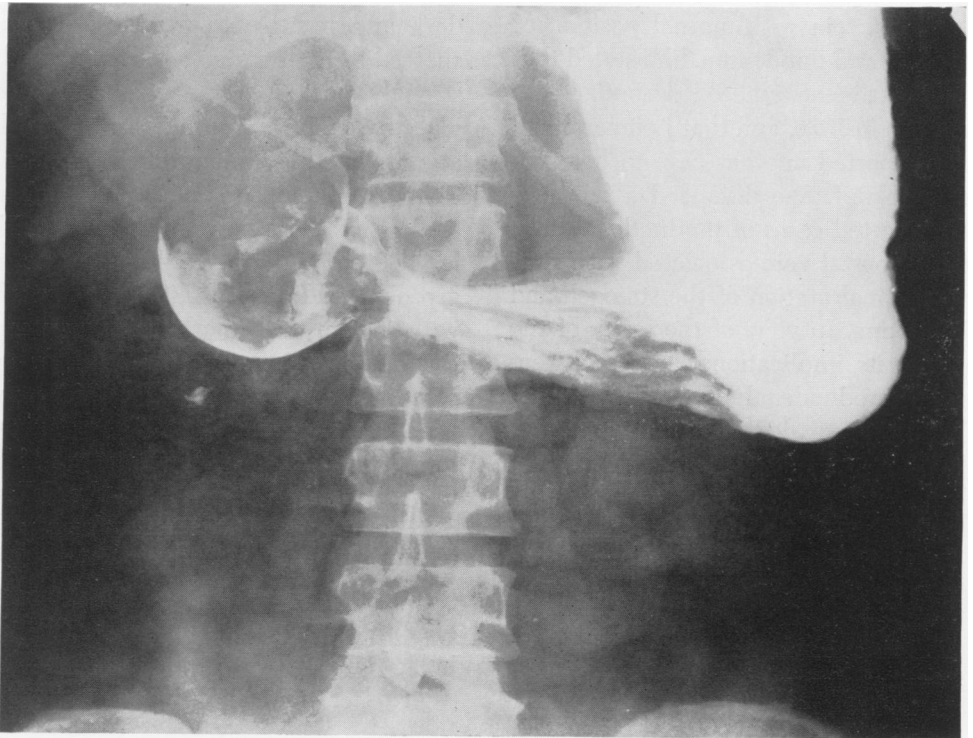


FIG. 1B. Obstruction at or immediately beyond the pylorus five hours following ingestion of barium.

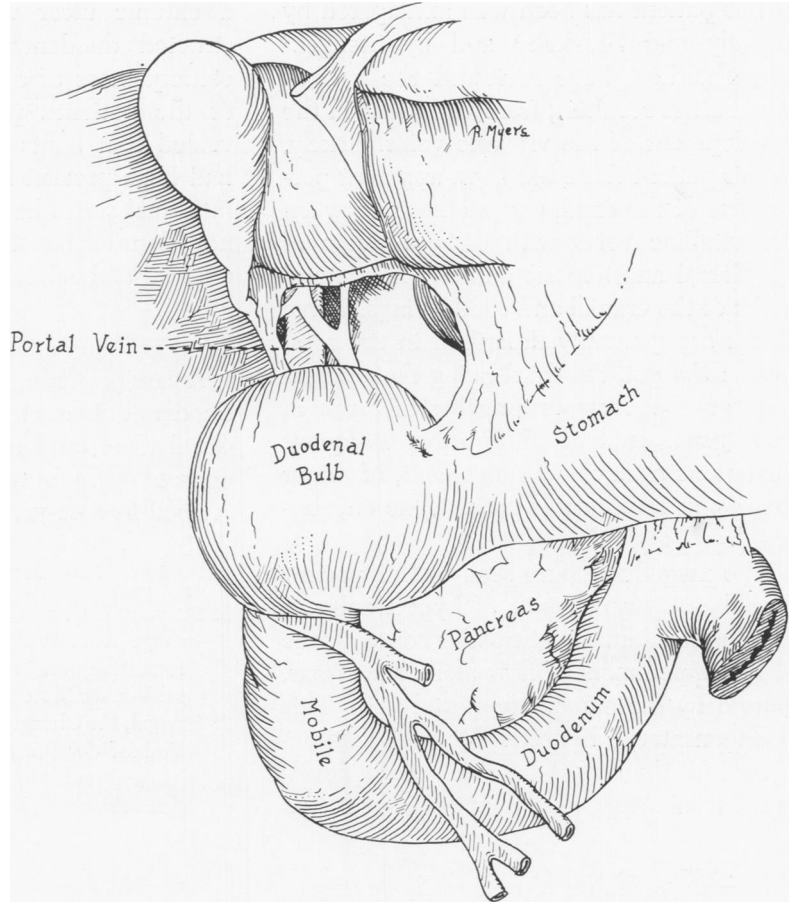


FIG. 2. Diagram of findings at operation.

markedly enlarged. A large vein arising from the base of the mesentery traversed and compressed the anterior aspect of the first part of the duodenum at its junction with the second part, and continued cephalad in the gastrohepatic ligament to the porta hepatis. The second and third portions of the duodenum as well as the cecum and the right colon were unusually mobile, the latter being almost entirely free save at the hepatic flexure. A scar was noted on the anterior duodenal wall about two cm. from the pylorus and close to the lesser curvature. On later opening the operative specimen this was seen to be an eight mm. diameter ulcer, in the crater of which was a small blood clot. In the proximal jejunum there was a broad base pseudodiverticulum measuring about four cm. in length, located

about six cm. from the ligament of Treitz. Seventy-five per cent of the stomach was resected, and antecolic gastrojejunostomy, invagination by plication of the jejunal pseudodiverticulum, and appendectomy were done.

The patient tolerated operation well and her post-operative course was smooth. She was discharged asymptomatic on the 15th day, on a regular diet and with her diabetes well controlled, requiring only occasional doses of insulin. She is being followed in the out patient department and remains asymptomatic with regard to the gastrointestinal system.

Comment

An embryological explanation of the anomaly of the portal system encountered

in this patient has been well anticipated by Snavely and Breakell¹ and by Stengel.² These authors have reviewed the various possibilities resulting from variations in the development of the vitelline veins. Briefly, in this patient there has been apparent persistence of the caudal anastomosis between the vitelline veins with disappearance of the dorsal anastomosis and the left lateral limbs of the cranial and caudal rings. Three stages are generally described in the rotation of the gut, the third being that of fixation of the retroperitoneal structures in their usual anatomical position. Frequent deviations from this arrangement, of minor degree, as seen in this patient, are encountered.

The megaduodenum seen in this patient was the apparent result of hypertrophy of the duodenal musculature as a consequence of the impediment of flow along the viscus, caused by the anomalous portal vein. This "compensatory" hypertrophy probably accounts for the long delay prior to the development of symptoms. It is of interest that

a chronic ulcer was present in the obstructed duodenum and may reflect the consequences of duodenal stasis. A review of the patient's past medical history revealed that bouts of epigastric discomfort had been present for some time. A gastrointestinal series had once been advised but not done some five years prior to her episode of hematemesis.

Summary

A case of preduodenal portal vein causing duodenal obstruction, with a bleeding duodenal ulcer, and accompanied by a minor degree of malrotation of the gut is described in a 48-year-old female.

References

1. Snavely, J. G. and E. S. Breakell: Fatal Hemorrhage from Esophageal Varices Due to Malformation and Congenital Stenoses in the Portal Venous System. *Am. J. Med.*, 16:459, 1954.
2. Stengel, F.: Über zwei Fälle von präduodenalem Verlauf der Pfortader bei normaler Lage von Magen und Duodenum. *Ztschr. f. Anat. u. Entwicklungsgesch.*, 102:661, 1934.