Regressing vitelline vein and the initial development of the superior mesenteric vein in human embryos

By

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Summary: The superior mesenteric vein was considered to develop in situ in the midgut mesentery secondary to regression of the left vitelline vein. We revisited the morphology using serial sections of 20 embryos at 5–6 weeks (CRL 9–15 mm). The regressing vitelline vein provided a long peritoneal fold in the immediately superior side of the midgut mesentery containing the thick superior mesenteric artery. Notably, in a half of specimens, there were tissue clefts along the superior mesenteric artery in the mesentery and they were communicated with the left vitelline vein at the superior end of the peritoneal fold. The tissue clefts appeared not to carry the endothelial lining. We considered the cleft as the initial superior mesenteric vein. Conversely, the initial vein seemed not to develop from budding or venous plexus.

Introduction

Although it might not be widely known, the vitelline vein does not persist in the superior mesenteric vein, but the latter develops secondarily in situ in the mesentery during or after regression of peripheral courses of the vitelline vein (left vitelline vein). Thus, only a small part of the vitelline vein is incorporated in the portal vein of the adult, namely, that part proximal to a confluence between the superior mesenteric and splenic veins (Keibel & Mall, 1912; Hamilton et al., 1978). In the present study, we reexamined the manner of the de novo development of the superior mesenteric vein. Does the vitelline vein make a budding for the de novo development? In addition, there might be no photographic demonstrations of a fact that, outside of the midgut mesentery containing the superior mesenteric artery, the vitelline vein crosses the coelom or peritoneal cavity independently (Keibel & Mall, 1912; Hamilton et al., 1978).

Materials and Methods

This study was performed in accordance with the provisions of the Declaration of Helsinki 1995 (as revised in 2013). We observed serial horizontal or sagittal sections of the abdomen from 20 embryos at 5–6 weeks (CRL 9–15 mm). All sections were a part of the large collection kept at the Embryology Institute of the Universidad Complutense, Madrid, and were the products of miscarriages and ectopic pregnancies at the Department of Obstetrics of the University. Most sections were stained with hematoxylin and eosin (HE), while a minor part with azan, orange G or silver staining. The study was approved by Complutense university ethics committee (B08/374).

Results

Although the present photos are shown from the small-
er specimen to the larger one, the size was not always connected to the difference in venous morphology. Usually (16 of 20 specimens), a long peritoneal fold containing the regressing left vitelline vein was located in the superior and right side of the midgut mesentery containing the superior mesenteric artery (Figs. 1, 3 and 4). Along the entire part, the fold was closely to but not fused with the midgut mesentery for the artery. The vitelline vein was narrow and extended along the supero-inferior axis in the peritoneal fold. The vein was communicated with the portal vein in the superior end of the fold or in the dorsal side of the duodenum and ventral pancreas.

In 4 specimens (CRL 6 mm, 8 mm, 10 mm, 12 mm; Fig. 2), the vitelline vein was dilated and contained in

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Fig. 1. A connection between the left vitelline and portal vein.
An embryo with 10 mm CRL. Sagittal sections including the midline area. HE staining. Panel A (or F) displays the most right (or left) site in the figure. In panels B and C, a distal part of the left vitelline vein (LVV) is broken (arrowheads) during the histological procedure. Tissue clefts are evident (arrows) along the superior mesenteric artery (SMA) and they are communicated with the left vitelline vein (panels C and D). A definite vein does not accompany the superior mesenteric artery. All panels were prepared at the same magnification (scale bar in panel A, 1 mm). Other abbreviations, see the common abbreviation.
a short fold attaching to the inferoventral aspect of the duodenum and, via a slit-like portion, the vitelline vein was communicated with the portal vein. This morphology appeared to be an earlier stage of the venous regression since, along the almost all aspects, the dilated vitelline vein was distant from the midgut mesentery containing the superior mesenteric artery. The vitelline vein carried a tributary with a blind end (Fig. 2EF), but the latter

Fig. 2. A closing connection between the left vitelline and portal vein. An embryo with 12 mm CRL. Horizontal sections. Silver staining. Panel A (or F) displays the most superior (or inferior) site in the figure. In panel C, the most superior or proximal part of the left vitelline vein provides a slit-like appearance (arrowheads) to connect with the portal vein (PV). In panels E and F, the left vitelline vein (LVV) carries a tributary with a blind end (arrows). A definite vein does not accompany the superior mesenteric artery (SMA). Panels A–D and F were prepared at the same magnification. Scale bars: 1 mm in panel A; 0.1 mm in panel E. Other abbreviations, see the common abbreviation.
Fig. 3. Tissue clefts along the superior mesenteric artery.

An embryo with 15 mm CRL. Horizontal sections. HE staining. Panel A (or H) displays the most superior (or inferior) site in the figure. Tissue clefts are evident (arrows) along the superior mesenteric artery (SMA) and they are communicated with the left vitelline vein (panels D–G). A definite vein does not accompany the superior mesenteric artery. A definite vein does not accompany the superior mesenteric artery (SMA). All panels were prepared at the same magnification (scale bar in panel A, 1 mm). Other abbreviations, see the common abbreviation.
appeared not to correspond to any mesenteric veins because it did not reach the midgut mesentery.

Notably, in a half of the specimen, tissue clefts were seen along the superior mesenteric artery in the mesentery and they were communicated with the vitelline vein at the superior end of the peritoneal fold (Figs. 1 and 3). The tissue clefts appeared not to carry the endothelial lining. However, in 4 specimens with a dilated vitelline vein clefts were absent in the midgut mesentery. We did not find morphologies suggesting budding from the vitelline or portal vein. Likewise, there were no splenic and inferior mesenteric veins draining into the portal vein. In

![Fig. 4](image_url) A connection between the left vitelline and portal vein remains in an embryo with 15 mm CRL.

Sagittal sections. HE staining. Panel A (or F) displays the most right (or left) site in the figure. No tissue clefts are seen along the superior mesenteric artery (SMA). All panels were prepared at the same magnification (scale bar in panel A, 1 mm). Other abbreviations, see the common abbreviation.
addition, the midgut mesentery was fused with parts of a mesentery of the descending colon (Fig. 3DE): the latter contained a thin artery but no definite vein.

**Discussion**

In the development of the portal vein, the initial course rounding the duodenum is well described in textbook (e.g., Skandalakis et al., 1972). The present specimens seemed not to carry such an anastomoses of vitelline veins around the duodenum but the final retroduodenal course after regression of the right vitelline vein. However, we were not able to deny a possibility that the dilated vitelline vein in the 4 specimens might not correspond to the earlier stage of regression but an anomaly connecting to the preduodenal portal vein. At the start of this study, we knew a description that the superior mesenteric vein develops *in situ* secondary to regression of the left vitelline vein (Keibel & Mall, 1912; Hamilton et al., 1978). However, an independent peritoneal fold containing the left vitelline vein surprised us because the vein was separated from the superior mesenteric artery.

In *de novo* development, a venous plexus with endothelium is likely to appear first in the midgut mesentery and, subsequently, the plexus provides a superior mesenteric vein. Otherwise, if the superior mesenteric vein originates from a budding of the left vitelline vein, the latter endothelial cells should be continuous with the former. However, we did not find either the budding or venous plexus. Instead, tissue clefts were present along the superior mesenteric artery in the mesentery. Notably, they were communicated with the left vitelline vein at the superior end of the peritoneal fold. We considered the superior mesenteric vein developed from the tissue clefts without endothelial lining. Since the tissue cleft did not correspond to a fusion plane between the peritoneal fold of the vitelline vein and the midgut mesentery, a mechanical stress between the growing and regressing tissues seems not to contribute to the early development of the superior mesenteric vein. The present specimens might be too early to observe the splenic and inferior mesenteric veins.

**Conflicts of interest**

The authors have no financial conflicts of interests.

**References**

1) Hamilton WJ, Mossman HW: Human Embryology. 4th ED. London: Williams & Wilkins, 1978, pp.272–277.
2) Keibel F, Mall FP. Manual of Human Embryology. Vol 2, Lippincott, Philadelphia, 1912, pp.671–675.
3) Skandalakis JE, Gray SW, Ricketts RR: Small intestine. In Embryology for Surgeons, Eds by Skandalakis JE and Gray SW, 2nd edition, Williams & Wilkins, Baltimore, 1972, pp.190–199.

**Figure legends**

Common abbreviation for figures:

AC, ascending colon;
AD, adrenal;
AO, aorta;
BR, bronchus;
CBD, common bile duct;
D, duodenum;
DC, descending colon;
DP, dorsal pancreas;
GB, gall bladder;
H, heart;
IVC, inferior vena cava;
K, kidney or metanephros;
L, liver;
LS, lesser sac or omental bursa;
LUV, left umbilical vein;
LVV, vitelline vein;
MD, mesonephric duct;
MN, mesonephros;
PC, peritoneal cavity;
PV, portal vein;
SMA, superior mesenteric artery;
SPV, splenic vein;
ST, stomach;
UA, umbilical artery;
UGS, urogenital sinus;
VP, ventral pancreas.