Wedge biopsy from the testicular mass confirmed the primary tumor to be mixed germ cell tumor.

A final diagnosis of mixed germ cell tumor of the testis with the choriocarcinomatous element alone metastasizing to the stomach was made. He was initiated on chemotherapy regimen with actinomycin-D, etoposide and methotrexate along with folic acid. During the first week of chemotherapy the patient developed generalized tonic-clonic seizures. CT scan of the brain revealed a metastasis in the parietal lobe. CSF beta hCG was found to be 280 IU/mL. Intrathecal methotrexate was added to the chemotherapy regimen. Serial CSF beta hCG levels after starting this therapy were 144.5 IU/mL, 115.2 IU/mL, <5 IU/mL, 58 IU/mL and 62 IU/mL. The patient died 6 weeks after starting chemotherapy.

Approximately 50% of patients with testicular germ cell tumors present with metastasis. Apart from nodal metastasis, distant spread can occur to the liver, lung and brain. Hematemesis can be due to metastatic implants in the gastric mucosa or retroduodenal nodal metastasis eroding into the duodenum. Beta subunit of hCG will be raised in high titers due to large tumor burden. Testicular wedge biopsy should be done even in cases without palpable testicular lump or sonological evidence before labeling it as primary gastric choriocarcinoma.

The prognosis of cases with gastric metastasis is poor because bowel metastasis is preceded by extensive nodal metastasis and is usually associated with hematogenous spread to other organs. Management is with chemotherapy, with or without radiotherapy. There are rare instances of massive bleed being managed with laparoscopic gastric resection, although long-term prognosis is poor.

References

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Multiple tubular and cystic intestinal duplications

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Duodenal and colonic duplication presenting as mass in a neonate is rare. We report a 52-cm-long tubular, total colonic and 5-cm-long cystic duodenal duplication in a two-day-old neonate. Both the duplications could be excised without resection of the normal bowel. [Indian J Gastroenterol 2004;23:224-225]

Key words: Bowel duplication, colon, duodenum

Alimentary duplications are uncommon and usually present during the first decade of life. They are mostly single, tubular/cystic, located on the mesenteric side, and less than 10 cm in size. They may present as a mass or as its complications.

A 2-day-old neonate presented with severe abdominal distension and tachycardia. He was a full-term normally delivered child. He had no vomiting and had passed meconium. The child was tachypneic and had icterus. The abdomen was soft but massively distended with visible loops seen. Per rectal examination was normal.

Routine investigations and arterial blood gases were within normal limits. X-ray of the abdomen showed full flanks with few specks of air distributed evenly. Ultrasonography showed a dilated bowel loop and a 5 cm x 4 cm cystic swelling in the subhepatic region separate from the dilated loop. Rest of the viscera was normal.

Laparotomy revealed a long mesenteric-sided total colonic tubular duplication occupying almost the whole abdomen. It was 52 cm in length, ending blindly at both the ends. It could be easily dissected from the mesentery without bowel resection. Another cystic lesion was attached to the second part of the duodenum and had no communication with the intestinal lumen. This could also be easily dissected and removed (Fig). Histology showed both duplications to be lined by normal gastrointestinal mucosa.
The patient had an uneventful recovery.

Alimentary tract duplications are rare congenital anomalies that can develop anywhere along the alimentary tract. They are usually single, tubular or cystic, and located on the mesenteric side.¹ They are most commonly located in the ileum¹ and least commonly in the duodenum.² Duodenal duplications are usually cystic, have no communication with the intestinal lumen, lie posterior to the duodenum, and are attached to the second part.³ They may share part of the muscular wall or blood supply of the bowel. In our case, tubular colonic and cystic duodenal duplications were present simultaneously in a neonate, which has not been reported earlier.

Most of the patients present in the first decade with obstruction, perforation, bleeding, abdominal or mediastinal mass, pancreatitis, pyloric stenosis, intussusception, or distension.³

Congenital anomalies of the vertebrae, intestinal atresia or stenosis, omphalocele or malrotation may be associated with alimentary duplication.⁴

Tubular duplication represents only 5%-10% of duplications and are rarely more than 20 cm in length.⁴ In our case the colonic duplication was complete, 52 cm in length and tubular. There are very few cases of such long tubular duplications, and fewer that could be excised without bowel resection.⁴

References


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