Cholecystoduodenoplasty for high-output duodenal fistula

We read with interest the description of cholecystoduodenoplasty for high-output duodenal fistula by Rohondia et al. The authors have selected this procedure for use in patients with leak from sutured duodenal ulcer perforations and they have timed their operations two weeks after the leakage was identified ("since the patients were in a morbid state").

Patients who survive until this point of time have a localized drain-site fistula with very little generalized peritonitis. In these cases, the subhepatic area is by that time converted into a granulating abscess cavity and there is likely to be considerable difficulty in fistula repair and use of a live patch (either bowel or gall bladder). The two jejunostomy site leaks are also probably related to severe adhesions, which are invariably present by that time, and inadvertent disturbance of the jejunostomy during the adhesion lysis. Would a tube duodenostomy at this point not be a more appropriate procedure? Resuturing or repair at the site of a leak following perforation closure is always fraught with risk of further disruption as the tissues are inflamed and friable. In the authors' own hands, resuturing had 100% mortality whereas the solitary patient with a tube duodenostomy had a favourable outcome.

We are surprised by the authors' observation that the jejunum is unsuitable for use as a patch as it is "already inflamed", but the gall bladder which is immediately adjacent to the area of the leak and which bears the brunt of the effluent is not!

In our opinion, a second repair of a leak from a perforated duodenal ulcer is doomed to fail, as the local tissue factors are not conducive to healing. Perhaps a larger series of patients may expose the fundamental weakness of this technique? In our experience of 9 leaks from duodenal ulcer perforations, all were managed by tube duodenostomy with successful outcome in 7 cases. In the remaining two, generalized sepsis and multi-system failure resulted in death although the fistula itself was controlled.

The data are confusing and the authors have described a "three tubes method" without stating where the tubes were placed.

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Reference

Reply from the author

Multiple procedures have been described for the management of duodenal fistulae secondary to leaks. This would suggest that none of them is foolproof. In Indians, an additional problem contributing to morbidity and mortality is nutritional deficiency; it is difficult to remedy this with total parenteral nutrition. The three-tube method referred to is described in literature, but is difficult to maintain in our patients due to overlapping electrolyte imbalances.

Closure of the duodenal fistula requires a viable organ with good vascularity. We preferred the gall bladder with a thumping cystic artery to a jejunal loop. The operative method has been described in our article. The point about the gall bladder being inflamed is valid, except that any operating surgeon would notice that this inflammation is confined to the outer wall and the mucosa is spared. We mobilize the gall bladder in such a way (fundus first) that it falls on the site of the perforation. A good cholecystoduodenoplasty with tensionless suturing is therefore possible.

We appreciate the success with tube duodenostomy, but our experience with the procedure has not been satisfactory. We would all agree that treatment of such fistulae is difficult, and each surgeon should perform a procedure that he is familiar with and gives good results.

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Reference

Sigmoidoscopic removal of misplaced Intrauterine contraceptive device

Migration of intrauterine contraceptive device (IUCD) from the uterine cavity is rare. Patients present with failure of contraception, inability to feel the threads at the external os, or with symptoms referable to organs where the migration has taken place. Migrations have been reported into the peritoneum, omentum, urinary bladder, cecum, appendix, small bowel, large intestine, pelvis and iliac vein. Migration into the rectosigmoid region has also been reported. Most of the migrated IUCDs require either laparotomy or laparoscopy for their removal. Sigmoidoscopic removal has been possible only once earlier.
Post-partum spontaneous hepatic rupture in eclampsia

A 26-year-old second gravida, para one, with severe pre-eclampsia (blood pressure 180/120 mmHg, 4+ proteinuria) was admitted at 31 weeks' gestation with fetal growth retardation. She was put on Pritchard’s regime and labor was induced with dinoprostone endocervical gel. She had generalized tonic-clonic convolution and delivered a male pre-term baby weighing 700 grams, which did not survive beyond 1 hour.

Six hours following delivery, she complained of severe epigastric pain radiating to the right shoulder, with features of peripheral circulatory collapse. She was stabilized with transfusion of two units of whole blood. Ultrasonography was reported as showing ascites. A surgical consultation at this stage was inconclusive. After 24 hours, the patient had a second convolution with uncontrollable fall in hematocrit. A second ultrasonography revealed a tear in the right lobe of the liver with free blood in the peritoneal cavity. Repeat surgical consultation resulted in peritoneal paracentesis revealing frank blood. All her laboratory values were within normal limits except raised serum bilirubin (3.9 mg/dL). She underwent laparotomy; 2500 mL of blood was evacuated from the peritoneal, gelofoam packing done over the 2-cm tear in the liver, and peritoneal lavage and drainage was done. Her postoperative course was uneventful.

Spontaneous rupture of the liver is a rare and potentially life-threatening complication of pre-eclampsia; the incidence is approximately 1 in 45,000 live births. Treatment, depending on the severity of the rupture, ranges from conservative management to liver transplantation. Right upper quadrant/epigastric pain radiating to the shoulder, the syndrome of hemolysis, elevated liver enzyme levels and low platelet count (HELLP), and signs of hemodynamic instability should prompt urgent ultrasonographic examination to look for hematoma/rupture and free blood in the peritoneal cavity. The classical clinical triad may vary considerably. Incorrect diagnosis and late treatment can lead to a high mortality rate.

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References