suming the program is continued every year, 300,000 (i.e., 60 x 5000) people are saved in that year alone.

This is the conundrum. A population of 1000 million should have an incidence of 184,000 HCC per year. The calculations above suggest that while reckoning cost benefits, any figure from 5000 to 300,000 HCC prevented per year can be used depending on the duration of the program. Yet the real incidence of HCC due to HBV is only 5000 per year. Obviously the calculations and the premises being used are in error.

We expect the government to open its purse strings on the recommendations of medical experts. It behooves us to try and get our sums right.

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References

Reply from the authors

The consensus statement is based on the opinion of two experts, Dr Vinay Dhir and Prof Narendra Nath, whose data were discussed at the conference with other experts and delegates. Dr Pullyel et al are justified in stating that 5000 cases prevented every year will keep adding up, and while reckoning cost benefits any figure from 5000 to 300,000 cases of HCC prevented per year can be used depending on the duration of the vaccination program. There could be many other interactive factors that could help enhance the number of HCC cases prevented per year. It is thus not a matter of simple addition of the cases prevented per year.

It is not appropriate to state that by vaccinating infants, only 1.08 million cases with chronic HBV infection will be prevented. Many more cases will be prevented that could otherwise have become infected by transmission from these HBV-infected children.

Certainly more work needs to be done to estimate the cost of prevention of HBV-related HCC in our country. This may require developing an appropriate model.

Shiv K Sarin

Cholecystoduodenoplasty for high-output duodenal fistula

The article on cholecystoduodenoplasty for high-output duodenal fistula by Rohondia et al was interesting. Conservative treatment and/or delayed surgery achieves fistula closure in a majority of patients provided there is no distal obstruction and no specific pathology (malignancy or tuberculosis) at the ulcer site.

Primary closure or early repair or reconstructive procedures carry high incidence of re-bleak and mortality as there is hyperemia and inflammatory edema around the fistula and the patients are in septicemia and negative nitrogen balance. In the authors’ series, the overall mortality from duodenal fistula treated by various techniques is approximately 80% (22/27). With cholecystoduodenoplasty alone, four and not three, as stated by the authors of six patients died, three of them from leak from earlier jejunostomy.

In patients with duodenal fistula, the gall bladder wall, being in close proximity, is always inflamed and adherent to the duodenum, liver and even the colon. Mobilization of the gall bladder may thus lead to serosal tear and vascular compromise. Instead, a jejunal loop may be a better choice for closure of perforation; it is more vascular, mobile and thicker than the gall bladder wall. The authors state that cholangitis after cholecysto-duodenoplasty is unlikely to occur if the cystic duct gets blocked due to inflammation. This is a presumption, not substantiated by this or any earlier study.

We have experience of managing 78 patients with gastrointestinal fistula in the last 4 years (1997-2000); of these, 17 were following simple closure of duodenal ulcer perforation. Ten patients with duodenal fistula (output 500-1000 mL/day) were managed with conservative treatment. This consisted of maintaining nutrition through endoscopically placed nasojejunal tube or feeding jejunostomy, re-feeding fistula output through jejunostomy after filtering it, broad-spectrum antibiotics, maintenance of fluid and electrolytes and transfusion of blood and plasma.

Fistula healed in six patients in 18-47 days and in two after delayed surgery (Billroth II gastrectomy-1, vagotomy and pyloroplasty-1). Eight of 10 patients in this group survived. The remaining 7 patients required emergency surgery (duodenostomy and jejunostomy-4, Billroth II gastrectomy-2, duodenostomy, jejunostomy and gastrectomy-1). Four of seven patients in this group survived. Overall, 12 of our 17 patients (70.5%) with duodenal fistula following closure of duodenal ulcer survived.

In our opinion, leak following closure of duodenal ulcer perforation should preferably be treated conservatively for 4-6 weeks. This allows the fistula to close in 60% of cases and surgery, if required later, carries better results. However, if surgery is required early due to spreading peritonitis, duodenostomy, jejunostomy and/
or gastrostomy give acceptable results. It is too early to accept cholecystostomy for closure of duodenal fistula. A prospective randomized trial of sufficient number of patients undergoing this surgery may give the answer.

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References

Reply from the authors
We thank Drs Verma and Bose for their valuable comments on our article. It is standard practice that conservative treatment be given a fair chance in the management of these patients, but it is difficult to maintain nutrition and prevent infection over a long period.

We apologize for the obvious error in the number of deaths we stated. The total number is indeed four; we had wished to emphasize that three of these occurred despite successful treatment of the leak.

Finally, we found it interesting that Drs Verma and Bose have treated 78 such cases over a period of 4 years, a workload much beyond ours in a tertiary referral center and large public hospital. It will be interesting to evaluate the cause of such re-leaks in their center. Their experience with the standard technique is different from ours. We would like to congratulate them on their excellent results.

Omprakash Rohondia

Ascaris lumbricoides leading to esophageal bleeding

Ascaris lumbricoides, an inhabitant of the small bowel, has occasionally been noted to explore adjoining orifices, ducts and cavities, and migrate into unnatural sites.1 We report a patient with chronic liver disease in whom variceal bleeding was probably induced by a live roundworm.

A 30-year-old man presented with massive hematemesis and melena of one day's duration. He neither had abdominal pain nor ingested non-steroidal anti-inflammatory drugs (NSAIDs). There was no history of jaundice, encephalopathy, pedal swelling or prolonged fever. He was a chronic alcoholic. He had tachycardia and was hypotensive. There was pallor but no icterus. He had peripheral stigmata of chronic liver disease in the form of spider nevi and parotidomegaly. Abdominal examination revealed firm hepatomegaly, splenomegaly and free fluid.

 Investigations: hemoglobin 7.0 g/dL, liver function tests showed normal serum bilirubin, elevated (2-3 times) liver transaminases, normal alkaline phosphatase and low serum albumin. Serum-protein albumin gradient was 1.4 g/dL and there was no evidence of spontaneous bacterial peritonitis. Upper gastrointestinal endoscopy revealed grade III and grade IV long-column esophageal varices. There was a fresh ulcer over one of the varices but there was no active bleeding. In addition there was a bulblous varix in the gastric fundus. A single live worm was seen in the body of the stomach; it was moving actively. The worm was removed endoscopically with the help of a snare, and was identified as Ascaris lumbricoides. When it was dissected on a blotting paper, blood-tinged ascariasis was seen and the blotting paper stained pinkish red. Endoscopic variceal sclerotherapy was done using 1.5% ethoxysclerol. Albendazole 400 mg was given to eradicate any other worm.

Circumstantial evidences in this case suggest that the ulcer over the varix was created probably by the roundworm, which in turn precipitated bleeding. We could not find another report of esophageal variceal bleeding induced by parasitic infestation. In veterinary medicine esophageal ulcerations have been reported to be caused by Ascaris suum.2 Ascaris lumbricoides has been reported to cause intestinal ulcerations, perforation and obstruction. It causes upper gastrointestinal bleeding by various mechanisms, including gastric erosions, hemobilia, and precipitation of bleeding from a pre-existing duodenal ulcer. Gastro-esophageal ascariasis is an unusual occurrence.3,4

It is well known that ascaris does not ingest blood. However, we found that the ascariasis of the index worm was blood-tinged. Ascaris lumbricoides infestation should be considered as a cause of precipitant factor for gastrointestinal bleeding in a tropical country.

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References