Prevalence of cholelithiasis in children – a hospital-based observation

Cholelithiasis is relatively uncommon in children. In the US, the reported prevalence is 0.15% to 0.22%, whereas in adults it is 4%-11%.1

Of 13,675 children who had ultrasound examination of the abdomen in our hospital during the period January 1999 to December 2003, 43 (0.31%) were detected to have gallstones. The indications for ultrasonography in these children were fever with hepatosplenomegaly in 1 (2%), recurrent abdominal pain in 5 (12%), ureteric colic in 1 (2%), and miscellaneous in 21 (49%). None of them had cholecodolithiasis, hemolytic anemia or jaundice.

The male:female ratio was 2.3:1. The median age for boys was 5 years (range 3 months to 14 years) and for girls it was 9 years (range 7 months to 15 years); 28% were less than one year old, 23% between one and 5 years, 37% between 5 and 10 years, and 12% between 10 and 15 years. Of the 13,632 children who did not have gallstones on ultrasonography, the male:female ratio was 1.1:1; 27.5% were less than one year old, 25.2% between 1 and 5 years, 27.8% between 5 and 10 years, and 19.5% between 10 and 15 years. All the stones were less than 5 mm in size and they were solitary in 56%. Forty-one (95.3%) children were asymptomatic and two were symptomatic and underwent cholecystectomy. The symptomatic children were 9 and 7 years old; the former presented with recurrent abdominal pain and the latter with fever and vomiting.

Friesen et al2 in a review of 693 children with gallstones reported that infants less than 6 months of age represented 10% of cases, 69% were in the age group 11-21 years, and 21% between 6 months and 10 years. A hospital-based review of cholelithiasis in children less than 12 years of age over a period of 10 years, including a Medline search during the same period, reported that 87% of children with gallstones were asymptomatic.3

We have for the first time outlined the frequency of incidental gallstones detected on routine ultrasonography in children in India. In the absence of hemolytic anemia, the etiology needs to be evaluated. The consequences on long-term follow up in non-operated cases needs to be ascertained.

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References

Treatment of appendiceal mass

The article1 by Dr Kumar and Dr Jain is incomplete in its message. Appendiceal mass is a late presentation of appendicitis. Its treatment could be surgical or expectant conservative treatment. Conservative treatment is less dangerous especially if the surgeon is less experienced and does not have access to adequate equipment. Conservative treatment classically follows the Ochsner-Sherren regimen, about which the authors make no mention. Surgical treatment may be early or planned.

I would recommend planned surgery over a conservative approach in the following or similar situations:

· If the patient is from a rural area, unable to get appropriate treatment near home
· Students preparing for professional examinations, who are unlikely to obtain leave in the near future
· Individuals who travel a lot
· Young women, in whom the right fallopian tube may be affected by the inflammation
· Where there is suspicion of malignancy.

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We have a different viewpoint from Dr Upasani’s about whether conservative treatment is less dangerous if the surgeon is less experienced and does not have access to adequate equipment. Generally, appendectomy is regarded as a Resident’s surgery, supervised by another Resident doctor. This is especially true in a public hospital set-up, where we work. But in the more than 20 years that one of us (SK) has worked in such a hospital, there has not been a single instance when we regretted an appendectomy done by a Resident doctor. Of course, the appendectomy in all these situations was for acute appendicitis. But whenever we followed the rule of doing appendectomy for appendicular mass (in our set-up or in a private set-up), complications invariably followed. That prompted us to undertake this study. So, appendectomy is a basic surgery and risks are negligible unless it is done in the presence of appendicular mass. And that is one of the messages we wanted to convey.

The conservative treatment we followed has been well described in our methods. It is essentially the same as the Ochsner-Sherren regimen.

Despite the results of our randomized trial, one may need to modify our recommendations to suit special situations.

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Henoch–Schönlein purpura probably due to montelukast presenting as subacute intestinal obstruction

Henoch-Schönlein purpura (HSP) is a small-vessel vasculitis that involves the skin, joints, GI tract, and kidneys.1 GI symptoms occur in up to 85% of the patients.2 Montelukast is a leukotrine inhibitor used in the management of bronchial asthma.3 We report a man with HSP who was on montelukast for one month before presenting initially as subacute intestinal obstruction.

A 20-year-old man presented with colicky abdominal pain since one day. The pain was periumbilical in location, associated with recurrent bilious vomiting and obstipation. There was no history of fever. He had history suggestive of allergic rhinitis for which he was taking montelukast for one month. On examination, the patient was pale and in agony; pulse was 110/minute, blood pressure 140/100 mmHg. Per abdominal examination revealed diffuse tenderness, mild guarding but no rigidity. There was no organomegaly and bowel sounds were exaggerated. A diagnosis of subacute intestinal obstruction was made.

Investigations: hemoglobin 10 g/dL, ESR 80 mm in first hour, leukocyte count 26,000/mm³ with 10 percent eosinophils. Liver and kidney function tests, serum electrolytes, calcium and magnesium, and urine examination did not show any abnormality. X-ray abdomen revealed multiple air-fluid levels. Chest X-ray was normal. Montelukast was stopped.

The next day, the patient started passing flatus but continued to have colicky abdominal pain of less severity. Repeat leukocyte count was 24,000/mm³ with eosinophil count of 9000/mm³. On the third day he developed arthralgias and palpable purpura that was distributed over the buttocks and the posterior aspect of arms and legs. Repeat urine examination showed 7-10 RBC/hpf. Stool for occult blood was positive. Serum IgE level was 0.9 mg/L (normal <0.0005). Antineutrophilic cytoplasmic antibodies (ANCA) were positive. Endoscopy revealed diffuse mucosal redness and hemorrhagic erosions in the stomach and multiple ulcers in the first and second parts of the duodenum. Biopsies from the ulcers showed prominent infiltration by eosinophils and red cell extravasation in the mucosa and submucosa. Barium meal follow-through showed thickened jejunal folds, small barium flecks and shallow ulcers in the jejunum. Skin biopsy showed infiltration of small vessels by polymorphonuclear leukocytes, suggestive of leukocytoclastic vasculitis. A diagnosis of HSP was confirmed and the patient was started on tablet prednisolone 60 mg a day. The patient improved clinically and investigation reports normalized.

Montelukast has been reported to cause systemic eosinophilia and small-vessel vasculitis, namely, Chürg-Strauss syndrome.2 An objective causality assessment4 revealed that montelukast leading to HSP as adverse drug reaction in this case was probable. We could not find any reported case of montelukast-associated HSP.

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References


Lymphoplasmacytic sclerosing pancreatitis mimicking pancreatic cancer

Ramachandran et al1 recently reported two cases of autoimmune pancreatitis treated with steroids. This condition often mimics pancreatic cancer in its clinical presentation though the CT findings of a diffusely enlarged pancreas without a discreet mass may suggest the diagnosis of lymphoplasmacytic sclerosing pancreatitis.