Butachlor-induced acute toxic hepatitis

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Butachlor is a highly effective herbicidal substance widely used by farmers. We report a 60-year-old man with exfoliative dermatitis, jaundice, increase in liver enzymes, and eosinophilia one day after accidental dermal exposure to butachlor toxin. The diagnostic workup showed no other cause, and liver histology was consistent with substance-induced toxic hepatitis. Within two weeks of conservative therapy, his liver function tests returned to normal. [Indian J Gastroenterol 2007;26:135-136]

Butachlor (2-chloro-2’, 6’-diethyl-N-(butoxymethyl) acetanilide) is a pre-emergent herbicide used mainly to control weeds in transplanted, direct-seeded rice and barley fields. It is a member of the chloroacetanilide class of chemistry and is an ingredient in Macheter® EC (Monsanto, USA). Butachlor toxicology studies in laboratory animals indicate low toxicity following acute oral, dermal, and inhalation exposure; however, chronic exposure may lead to liver and kidney toxicity.1

A 60-year-old man, a farmer in the north of Iran was referred with icterus. He had been in good health until 20 days before admission when, after dermal exposure to butachlor toxin for a few minutes, he noted redness of hands and feet with pruritus that made him visit a primary-care physician. An injection of antihistaminic led to rapid improvement in pruritus, but pink scaly lesions on the skin remained. Two days later, nausea, fever, and icterus developed. He also noticed mild pain in the right upper abdominal quadrant. Fever resolved after one day. Two years earlier, he had received vaccination for hepatitis B virus. He did not smoke, drink alcohol, or use illicit drugs. The family’s drinking water came from a well. He had no muscle weakness or neurologic symptoms.

On examination, vital parameters were normal. He was icteric. The skin showed no rashes or lesions; however, it was pink and scaly at the extremities. No other abnormalities were observed on clinical examination.

Investigations revealed remarkable eosinophilia (eosinophils 20%) and elevation in liver enzymes (ALT 2440 U/L, AST 1200 U/L, alkaline phosphatase 648 U/L), with direct hyperbilirubinemia (total bilirubin 24.7 mg/dL, direct 17.6) and elevated serum amylase (1058 U/L). Urine analysis was positive for albumin, but kidney function tests were normal. HBsAg and anti-HCV were negative, but anti-HBs antibody and anti-hepatitis A virus immunoglobulin G were positive. Erythrocyte sedimentation rate, C-reactive protein, anti-mitochondrial, anti-smooth muscle, and anti-nuclear antibodies were normal or negative. Serologic test for Fasciola hepatica was positive.

There were no specific findings on abdominal sonography. ERCP and magnetic resonance cholangiopancreatography did not show any abnormality. Histology of liver showed a moderately severe acute hepatocellular necrosis and acute cholangitis, compatible with acute toxin-induced hepatitis (Fig).

Topical treatment was applied for skin lesions. Biochemical parameters gradually returned to near-normal levels. Two weeks after admission the patient was in good condition and icterus had decreased.

Acute onset of disease after dermal exposure to butachlor suggested a toxin-induced hypersensitivity reaction. Clinical recovery after two weeks of conservative therapy, exclusion of other probable causes, and liver biopsy results provided additional evidence for the diagnosis. To our knowledge, this is the first case of human toxic hepatitis that can be ascribed to butachlor toxin.

Although serologic test for Fasciola hepatica was positive, it is unlikely that this infection was the cause of his signs and symptoms.

The typical features of liver fascioliasis were absent, except for jaundice and peripheral eosinophilia; however MRI was normal in this patient, thus ruling out fascioliasis. ERCP also did not show evidence of fascioliasis. Histological features were also not suggestive.

Machete® EC herbicide is an organic solvent-based liquid formulation containing approximately 32% or 60% butachlor. Primary dermal irritation
studies showed slight erythema and edema resulting from continuous 24-h exposure to butachlor; thus, butachlor can produce allergic skin reactions following repeated or prolonged exposure. Liver toxicity has been shown in subchronic and chronic toxicity studies, in which butachlor was administered in the diet or orally via gelatin capsules. Toxicity was observed only at 1000-ppm dietary level and above in male rats, and at 3000-ppm and above in female rats. In this case, liver damage occurred following acute dermal exposure to toxin.

Evaluation of data obtained from health and safety databases developed to support the registration of butachlor and Machete® products indicates that these are of generally low toxicity and present minimal opportunity for human exposure. When used in accordance with label directions, butachlor does not adversely affect human health.

References


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Eosinophilic pancreatitis with pseudocyst

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Eosinophilic pancreatitis is a rare entity in patients having underlying systemic manifestations such as peripheral eosinophilia, elevated serum IgE levels and/or eosinophilic infiltrates in other organs, including the gastrointestinal tract. We report a 38-year-old woman with peripheral eosinophilia in association with acute pancreatitis, pancreatic ascites and pseudocyst. [Indian J Gastroenterol 2007;26:136-137]
and cholecystectomy was done. Pancreatic tissue along with the pseudocyst the gall bladder histology showed thickened wall with collagen fibers, adipocytes, blood vessels, focal and diffuse collections of mononuclear inflammatory cells with eosinophilic predominance (Fig).

She was treated with tapering dose of oral prednisolone (40 mg/day) postoperatively. The patient recovered uneventfully and is on regular follow up since 8 months without any complications or recurrent symptoms.

The association of pancreatitis with eosinophilia is rare. Increased eosinophils in the pancreas has been found in conjunction with pancreatic cancer, lymphocytic sclerosing pancreatitis, subcutaneous fat necrosis, eosinophilic gastroenteritis and systemic mastocytosis. Patients with eosinophilic pancreatitis show two distinct histological patterns: 1) diffuse, periductal, acinar and septal eosinophilic infiltrate with eosinophilic phlebitis and arteritis and, 2) localized intense eosinophilic infiltrate associated with pseudocyst formation.1

Most patients with eosinophilic pancreatitis have systemic manifestations such as peripheral eosinophilia, elevated serum IgE levels and/or eosinophilic infiltrates in other organs, including the gastrointestinal tract.2 A possible explanation for this is that local pancreatic inflammation might induce a factor causing degranulation of mast cells and release of eosinophilic chemotactic factor A, which would then stimulate the development of eosinophilia. Idiopathic hypereosinophilic syndrome, an entity that includes persistent hypereosinophilia (eosinophil count >1500/mm³ for 6 months), multi-organ involvement and lack of other recognized causes for eosinophilia has been described in association with eosinophilic pancreatitis.3

In all cases with pancreatitis with peripheral eosinophilia, the possibility of eosinophilic pancreatitis has to be considered as it is completely treatable.

References

Primary neuroendocrine carcinoma presenting as mesenteric cyst

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Primary neuroendocrine carcinoma occurring in the mesentery is extremely rare. Surgical resection is the best treatment modality, with a chance of cure. We present a 65-year-old man with large mesenteric cyst and absence of bowel involvement. Histology showed a well-differentiated neuroendocrine tumor. [Indian J Gastroenterol 2007;26:137-138]

Neuroendocrine tumors (NET) are uncommon tumors, representing less than 1% of all visceral malignancies and less than 2% of malignant tumors in the gastrointestinal tract.1 In the digestive system, they are found in the appendix, jejunum, stomach, duodenum, pancreas, colon and rectum.

A 65-year-old man presented with history of constant dull aching pain and palpable lump in the left upper and central abdomen since six months. Diagnostic laparoscopy elsewhere had revealed a 15 cm x 15 cm mass in the mesentery; biopsy did not yield a definitive diagnosis. Clinical examination confirmed the laparoscopy findings. Computed tomography revealed a heterogeneous mass probably arising from mesentery with a segment of proximal jejunum in close relation (Fig). Ultrasound-guided biopsy reported a low-grade tumor with neuroendocrine differentiation.

At laparotomy, there was a large mass in the mesentery of the jejunum about 8 cm distal to the duodenojejunal flexure. About 4 cm of jejunum was stretched out on the mass but was not grossly infiltrated by the tumor. There was no evidence of distant metastasis, ascites or lymphadenopathy. The mass was separated from within the leaves of the jejunal mesentery and R0 resection was done. The segment of jejunum close to the tumor appeared dusky, and jejunal resection with end-to-end anastomosis was done. The patient had a smooth postoperative recovery and was discharged 8 days after surgery.

Histology showed a well-differentiated neuroendocrine carcinoma in a mesenteric cyst. On immunohistochemistry, tumor cells were positive for cytokeratin (CK), epithelial membrane antigen (EMA), synaptophysin and chromogranin. The MIB-1 labeling index was less than 2%. The resected segment of...
jejunum was not involved by the tumor.

NET are typically found to contain numerous membrane-bound neurosecretory granules. These granules are composed of a variety of hormones and biogenic amines.\textsuperscript{1,2} Patients with nonsecreting tumors usually present with a large tumor burden but may lack any of the cancer-associated cachexia or morbidity. Our patient was probably a non-secretor. In 40%-50% of NET of the small intestine, mesenteric lymph nodes may be enlarged due to metastatic disease. In our case the small bowel was normal. The rest of the mesentery of the small bowel was also normal without fibrosis or desmoplastic reaction.

We found only one case of primary mesenteric carcinoid, which is a type of low-grade NET.\textsuperscript{3} Our case is probably the first documented case of primary neuroendocrine carcinoma (type of high-grade NET) presenting in a mesenteric cyst.

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**Vesicoperitoneal fistula – an unusual cause of tense ascites**

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We report a 55-year-old man who developed tense ascites due to vesicoperitoneal fistula. He had undergone surgery 32 years ago for excision of an infected urachal cyst, the tract and the umbilicus. [\textit{Indian J Gastroenterol} 2007;26:138-139]

Rupture of urinary bladder is usually a consequence of trauma; spontaneous rupture is very rare.

A 55-year-old man presented with rapidly accumulating ascites. He had no history suggestive of liver, renal, or peritoneal disease. Clinical examination was normal except for right paramedian abdominal scar and tense ascites. Investigations: normal blood count and liver and thyroid function tests. X-ray chest and electrocardiogram were normal. Urea 120 mg/dL, creatinine 2.2 mg/dL, 24-hour urinary protein excretion 90 mg. Ascitic fluid examination: white cell count 100 cells/µL, total proteins 1.61g/dL, albumin 1.3 g/dL, amylase 92 U/L and no malignant cells. Upper gastrointestinal endoscopy was normal and ultrasonography revealed only ascites.

The patient complained of scanty urination. He was catheterized and about 3 liters of fluid drained. He was put on frusemide; over a period of 2 days the ascites improved and patient passed 3.5 liters of urine over 24 hours. As the patient’s condition improved, the catheter was again removed. Six hours after removal of the catheter, the patient developed suprapubic pain, tachycardia, ascites and passed scanty urine associated with dysuria. He was given intravenous fluids and was recatheterized. The ascites improved markedly with indwelling catheter and he passed 4 liters of urine over 24 hours.

Due to the sequence of events, a vesicoperitoneal fistula was suspected and cystography was done. This revealed elongated bladder with finger-like projections arising from the dome, with contrast leaking into the peritoneum from one of the projections (Fig). The patient’s medical records were reviewed and it was found that he had undergone exploratory laparotomy 32 years ago for infected urachal cyst. Umbilicus, the tract and the cyst were removed \textit{en mass}, in addition to surgery for hernia. Pelvic ultrasonography revealed benign hyperplasia of the prostrate. The patient was discharged with indwelling catheter and was put on terazosin (alpha adrenergic blocker). The catheter was removed after 2 weeks. The patient is doing well during follow up for 4 months.

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**Fig: CT scan shows a mass probably arising from mesentery with segment of proximal jejunum in close relation to mass**
Embryologically, the bladder develops from the cranial portion of the urogenital sinus. The bladder is initially continuous with the allantois. Over time, the allantois degenerates to form a cord-like structure, the urachus. The urachus goes from umbilicus to the apex of the bladder and forms the median umbilical ligament.

The common anomalies of urachal development include patent urachus, urachal sinus, urachal diverticulum and a cyst. In children, urachal cyst is the common presentation while in adults infected urachal sinuses are seen more frequently. Our patient developed bladder neck obstruction, leading to increased intravesicular pressure. The bladder wall, weakened by previous surgery, might have given way, resulting in vesicoperitoneal fistula and eventually to tense urinary ascites that responded to the placement of indwelling catheter. No such case has been reported in literature so far.

Wandabwa et al reported a patient with spontaneous rupture of bladder during puerperium, while Kawakami et al reported rupture of urachal diverticulum in radiation cystitis and neurogenic bladder after radical hysterectomy. Urinary bladder dome necrosis leading to urinary extravasation and peritonitis due to an infected urachal cyst has been reported.

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Intraductal papillary mucinous neoplasm in tropical chronic pancreatitis

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We report a 56-year-old man with chronic calcifying pancreatitis of the tropics (tropical calcific pancreatitis) who had been asymptomatic, and on follow up developed a pancreatic mass that was identified as intraductal papillary mucinous neoplasm. He has been asymptomatic after distal pancreatectomy. [Indian J Gastroenterol 2007;26:139-140]

The entity of intraductal papillary mucinous neoplasm (IPMN) was first highlighted by Itai in 1986. Over the last decade, there has been an increase in diagnosis of this condition, and its clinicopathological features have been described. There have been difficulties in differentiating chronic pancreatitis from IPMN.

A 56-year-old man had been diagnosed to have chronic calcifying pancreatitis 20 years ago. Seven years ago, he developed diabetes mellitus and remained on oral hypoglycemic agents until 2 months ago when he required insulin therapy. He had experienced only mild pain in the past which responded to medication. Ultrasonography on several occasions over the past 10 years had not revealed any mass lesion in the pancreas. Two months ago, he developed severe unrelenting pain with anorexia, and weight loss of 10% of his body weight. Clinical examination did not reveal abdominal mass or ascites; there was tenderness in the epigastrium. There were no peripheral lymph node enlargements.

Biochemistry and hematology were normal and CA 19-9 level was 32 U/mL (normal up to 37). CT scan revealed bulky tail of pancreas with a heterogeneously enhancing soft-tissue lesion (Fig). There was atrophy of the body with dense calcification in the head, body and tail. The duct was dilated to 7 mm. The lesion appeared to encase the celiac axis. CT-guided fine-needle aspiration cytology did not yield malignant cells.

Diagnostic laparoscopy did not reveal any evi-
idence of peritoneal or hepatic disease and hence a laparotomy was performed. The celiac and superior mesenteric arteries were free and the pancreatic mass was resectable. The splenic artery was involved in the mass. The pancreatic duct was 8 mm in diameter, and the mass involved the proximal body and tail. The head was atrophic. Distal pancreatectomy and splenectomy with lymph node clearance was performed. Cut section of the gland revealed cystic spaces with calcific material interspersed with whitish yellow tissue. Microscopy revealed scattered cystically dilated glandular structures lined by tall columnar mucinous epithelium with mild nuclear stratification and focal papillary formation. The glands contained pale basophilic mucinous material and were intervened by dense fibrous tissue with several scattered nests of islet cells and focal collections of inflammatory cells. Several dilated ducts containing calculi were also seen. Endocrine acini were markedly atrophic. Lymph nodes were uninvolved and the spleen did not show any significant pathology.

Postoperative recovery was uneventful and the patient is asymptomatic 12 months after surgery.

To our knowledge, this is the first report of the coexistence of chronic calcifying pancreatitis of the tropics and IPMN. Although it is now well known that IPMN can be indolent and be treated as chronic pancreatitis, it appears that this patient had chronic calcifying pancreatitis without a mass lesion on earlier imaging. Talamini et al. suggested that IPMN may be the cause of chronic pancreatitis and not vice versa.

The superimposition of IPMN on chronic pancreatitis complicates the diagnostic process. The exact extent of the disease is difficult to identify, and therapy may be difficult. Close postoperative surveillance may be necessary.

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Spontaneous regression of patent omphalo-mesenteric duct

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We report a one-month-old male child with a patent omphalo-mesenteric duct that regressed spontaneously in the neonatal period and resulted in a Meckel’s diverticulum. [Indian J Gastroenterol 2007;26:140-141]

Omphalo-mesenteric duct malformations range from a completely patent omphalo-mesenteric duct at the umbilicus to a variety of lesser remnants including cysts, fibrous cords connecting the umbilicus to the distal ileum, granulation tissue at the umbilicus, umbilical hernias, and Meckel’s diverticulum. Patent omphalo-mesenteric duct accounts for about 2% of these malformations.

Symptoms may involve fecal fistulas at the umbilicus, intussusceptions/prolapse of ileum at the umbi-
licus, intestinal obstruction from a variety of causes, melena and anemia, abdominal pain and inflammation. Although symptoms occur most frequently during childhood (first 2 years of life), they may occur through adult years as well.

A 12-day-old boy was admitted with history of fecal discharge from the umbilicus (Fig). With a diagnosis of patent omphalo-mesenteric duct, we planned for laparotomy and excision of the tract with end-to-end anastomosis and umblicoplasty. Due to unavoidable circumstances the child was taken home. He was brought again to us 4 weeks later but this time there was no fecal discharge and the umbilicus was healthy except for raw surface. At exploratory laparotomy there was no sign of patent omphalo-mesenteric duct except the presence of a Meckel’s diverticulum, which was excised and bowel continuity maintained.

Regression of the omphalo-mesenteric duct occurs as a normal embryonic event in the intrauterine life between the fifth and ninth weeks of gestation.

Prompt surgical repair of patent omphalo-mesenteric duct minimizes the risk of intestinal obstruction and prolapse of the ileum through the fistula. In this child, regression occurred spontaneously in the neonatal period and resulted in a Meckel’s diverticulum with no connection with the umbilicus. Only two similar cases of spontaneous postnatal regression were found in the literature. This is the first case reported from India.

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