Primary bronchogenic squamous cell carcinoma presenting as an acute abdomen: Report of case

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Bronchogenic carcinoma metastasizes so rapidly that it may do so before the primary site is diagnosed. A rare case is reported, believed to be only the ninth in the literature, in which primary bronchogenic carcinoma metastasized to the small bowel and the third reported in which the patient presented with an acute abdomen.

A rare case of small bowel obstruction caused by metastasis of undiagnosed stage one bronchogenic carcinoma is reported. The patient presented with vague abdominal pain, distention, and nausea after being treated for recurring respiratory infections. On exploration, a large intraluminal mass was discovered in the distal ileum. Two other sites of involvement were found at the site of a Meckel's diverticulum. Nodal involvement was extensive. Preoperative chest x-ray showed a right lower lobe infiltrate. Following intestinal resection, bronchoscopy was performed. The tumor was found extruding from the right basilar segments. Biopsied specimens revealed squamous cell carcinoma.

This is the ninth case report of primary bronchogenic carcinoma metastasizing to the small bowel,[1-8] and only the third[1-3] case report in which the presenting symptoms of an acute abdomen were actually stage 2 to stage 1 lung tumor.

Report of case

A 49-year-old white man was seen on consultation with a diagnosis of an acute abdomen. For 6 weeks prior, the patient had received three courses of antibiotics for a respiratory infection. There was temporary resolution, but with repeated recurrences. Chest x-ray revealed consolidation of right basilar infiltrate consistent with a possible pneumonia. Examination also revealed a distended abdomen, generalized tenderness, and a palpable lower right quadrant mass.

Auscultation revealed hyperactive bowel sounds with tinkles/rushes. Positive peritoneal signs were also noted. Further examination elicited decreased breath sounds in the right base.

The complete blood count, electrolytes, blood sugar, and BUN were within normal limits. SMA-12 report was not available at that time.

The patient was taken to surgery for a two-part procedure. The first consisted of an abdominal exploration through a right paramedian excision. The abdominal contents were explored. Approximately 10-12 cm. from the ileocecal junction, a large luminal obstruction mass was found. The bowel was exteriorized and two other sites of involvement were found at the site of Meckel's diverticulum. Mesentery lymphadenopathy extended from tumor site to the root of mesentery. No liver involvement was palpable and no pancreatic masses were found.

The large tumor was removed by segmental small bowel resection with EEA. Each of the smaller tumors were removed by enteroplasty; lymph node biopsy and a Meckel's diverticulectomy were also performed.

Frozen sections revealed squamous cell carcinoma-metastatic.

The second part of the operation was a bronchoscopy which revealed a tumor extending from the right basilar segment. Multiple biopsies were performed.

The final pathology report was as follows: (1) bronchoscopic cell block preparation—squamous cell carcinoma; (2) segments of small bowel tumor resection showing a metastatic ulcerative squamous cell carcinoma; (3) Meckel's diverticulum with heterotrophic tissue—pancreatic/gastric tissue; and (4) mesentery lymph nodes—metastatic squamous cell carcinoma.

Postoperatively the patient did well and was maintained on total parenteral nutrition until recovery. Oncologic consultation was obtained with recommendation for palliative chemotherapy. The patient survived 6 weeks on palliative treatments.

Discussion

Perforation of the small bowel from metastatic stage 1 bronchogenic carcinoma is a very uncommon and late phenomenon,[8] which indicates a very poor prognosis.[1-8] The first case involving a small bowel perforation was reported by DeCastro.[1] He collected 25 cases of bowel obstruction caused by metastasis and found 26 other published reports.

One case out of 51 resulted from primary bronchogenic carcinoma. Seven other cases of small bowel perforation have been reported, the most recent by Ejeckam and associates.[8] In 2 out of these 8 cases the presenting problem was an abdominal crisis. Four of the 8 cases which reported abdominal crisis occurred after therapy and the radiation/chemotherapy had been speculated to be the initiating cause of perforation. Ejeckam[8] proposed that compromise of the submucosa and mucosal vessels by the tumor leads to tumor necrosis and perforation.

Bronchogenic carcinoma apparently spreads via the hematogenous route.[2] It will often metastasize before the primary site is diagnosed.[2] The most common sites of metastasis are the suprarenal glands, bones, brain, and the liver.[2] Other metastatic sites can occur.

Inalsingh and associates[2] postulated the influence of the unusual response from local tissue reaction, radiotherapy, and trauma. In the 4 cases reported[2] there was no evidence of secondary disease in the region of metastasis which was thought to suppress the tumor cells and allow metastasis elsewhere. Of the reported cases in the above paper, therapy (radiotherapy/chemotherapy) had been instituted. It was postulated that failure of the immune response, local factors, and even growth-promoting factors may enhance the site of tumorous response.

Traumatized tissue is known to have a higher probability of metastasis and previous factors discussed are felt to play an insurmountable role as the cause.

In 1960, Midell and Lochman[3] and Morgan[4] reported similar cases of small bowel perforation following resection of primary lung tumors. Morgan[4] suggested a relationship between perforation and chemotherapy which his patient underwent.

DeCastro[1] in his series of small bowel metastases, the skin, cervix, and kidney were frequent sites of primary tumor. Lung carcinoma was not listed.

Midell and Lochman[3] speculated that spread occurred either by the lymphatics network connecting the pulmonary structures and the intestines or by hematogenous route through the vertebral venous plexus. They also alluded to the friability of tissue and tissue necrosis caused by radiotherapy, either subsurface or deep penetration.[3,7]

Conclusion

Of the cases reported of primary bronchogenic carcinoma metastasizing in the small bowel, 6 had previous diagnosis with post-therapy. The remaining 2 were undiagnosed with no treatment. All 8 cases reported perforation of the small bowel as presenting causes of an acute abdomen. This is the ninth case reported, third undiagnosed, and the first reported as small bowel obstruction.

Various facts and theories have been presented and many conclusions can be drawn from the reported literature. The consensus is that failure of the immune response, trauma, and post-therapy treatments play an initial role in promoting tumor growth and complication.

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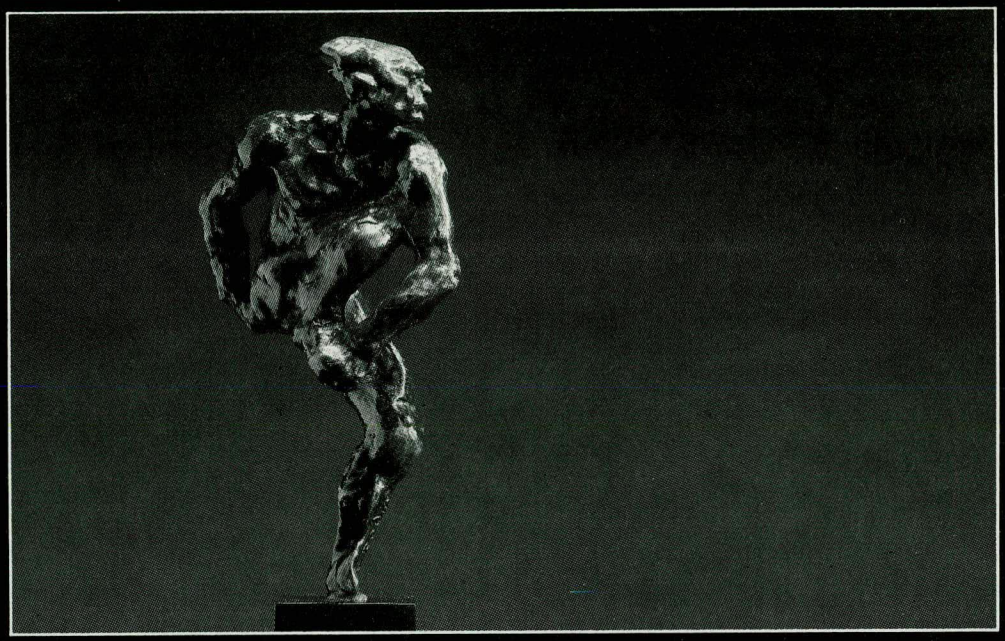

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Usage in Pregnancy: In view of embryotoxicity findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefit outweighs the potential risk to mother and fetus.
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Precautions: When discontinuing Combipres, reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of Combipres therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect of the clonidine hydrochloride component. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other antihypertensive agent,Combipres should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure. As an integral part of their overall long-term care, patients treated with Combipres should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmological findings have been recorded with Catapres, in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.
Patients predisposed toward or affected by diabetes should be tested periodically while receiving Combipres, because of the hyperglycemic effect of chlorthalidone.
Because of the possibility of progression of renal failure, periodic determination of the BUN is indicated. If, in the physician's opinion, a rising BUN is significant, the drug should be stopped.
The chlorthalidone component of Combipres may lead to sodium and/or potassium depletion. Muscular weakness, muscle cramps, anorexia, nausea, vomiting, constipation, lethargy or mental confusion may occur. Severe dietary salt restriction is not recommended in patients receiving Combipres.
Periodic determinations of the serum potassium level will aid the physician in the detection of hypokalemia. Extra care should be given to detection of hypokalemia in patients receiving adrenal corticosteroids, ACTH or digitalis. Hypochloremic alkalosis often precedes other evidence of severe potassium deficiency. Frequently, therefore, more sensitive indicators than the potassium serum level are the serum bicarbonate and chloride concentrations. Also indicative of potassium depletion can be electrocardiographic alterations such as changes in conduction time, reduction in amplitude of the T wave; ST segment depression; prominent U wave. These abnormalities may appear with potassium depletion before the serum level of potassium decreases. To lessen the possibility of potassium deficiency, the diet, in addition to meat and vegetables, should include potassium-rich foods such as citrus fruits and bananas. If significant potassium depletion should occur during therapy, oral potassium supplements in the form of potassium chloride (3 to 4.5 gm/day), fruit juice and bananas should be given.
Adverse Reactions: The most common reactions are dry mouth, drowsiness and sedation. Constipation, dizziness, headache, and fatigue have been reported. Generally these effects tend to diminish with continued therapy.
Clonidine hydrochloride: Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities in liver function tests; one case of possible drug-induced hepatitis without icterus and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlorthalidone and papaverine hydrochloride. Weight gain, transient elevation of blood glucose, or serum creatine phosphokinase; congestive heart failure, Raynaud's phenomenon; vivid dreams or nightmares, insomnia, other behavioral changes, nervousness, restlessness, anxiety and mental depression. Also rash, angioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associated with a rash; impotence, urinary retention; increased sensitivity to alcohol, dryness, itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomastia, weakly positive Coombs' test, asymptomatic electrocardiographic abnormalities manifested as Wenckebach period or ventricular trigeminy.
Chlorthalidone: Symptoms such as nausea, gastric irritation, anorexia, constipation and cramping, weakness, dizziness, transient myopia and restlessness are occasionally observed. Headache and impotence or dysuria may occur rarely. Orthostatic hypotension has been reported and may be potentiated when chlorthalidone is combined with alcohol, barbiturates or narcotics. Skin rashes, urticaria and purpura have been reported in a few instances.
A decreased glucose tolerance evidenced by hyperglycemia and glycosuria may develop inconsistently. This condition, usually reversible on discontinuation of therapy, responds to control with antidiabetic treatment. Diabetics and those predisposed should be checked regularly.
As with other diuretic agents hypokalemia may occur (see Precautions). Hyperuricemia may be observed on occasion and acute attacks of gout have been precipitated. In cases where prolonged and significant elevation of blood uric acid concentration is considered potentially deleterious, concomitant use of a uricosuric agent is effective in reversing hyperuricemia without loss of diuretic and/or antihypertensive activity.
Idiosyncratic drug reactions such as aplastic anemia, thrombocytopenia, leukopenia, agranulocytosis, and necrotizing angiitis have occurred, but are rare.
The remote possibility of pancreatitis should be considered when epigastric pain or unexplained gastrointestinal symptoms develop after prolonged administration.
Other adverse reactions which have been reported with this general class of compounds include: jaundice, xanthopsia, paresthesia and photosensitization.
Overdosage: *Catapres (clonidine hydrochloride):* Profound hypotension, weakness, somnolence, diminished or absent reflexes and vomiting followed the accidental ingestion of Catapres by several children from 19 months to 5 years of age. Gastric lavage and administration of an analeptic and vasopressor led to complete recovery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minute intervals abolishes all effects of Catapres overdosage.
Chlorthalidone: Symptoms of overdosage include nausea, weakness, dizziness, and disturbances of electrolyte balance. There is no specific antidote, but gastric lavage is recommended, followed by supportive treatment. Where necessary, this may include intravenous dextrose and saline with potassium administered with caution.
How Supplied: Combipres® 0.1 (Each tablet contains clonidine hydrochloride 0.1 mg + chlorthalidone 15 mg). It is available as pink, oval, single-scored compressed tablets in bottles of 100 and 1000.
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For complete details, please see full prescribing information.

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