ADENOSQUAMOUS CARCINOMA OF THE ILEUM - A CASE REPORT

F C Ng, H K Ang, H C Chng

ABSTRACT

Small bowel malignancies are very uncommon and adenosquamous carcinomas of the small intestine are extremely rare. We report the second case of an adenosquamous carcinoma of the ileum in a 77-year-old Chinese male.

Keywords: adenosquamous carcinoma, small intestine

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INTRODUCTION

Primary malignant neoplasms of the small intestine are very uncommon, constituting only 1% to 3% of all gastrointestinal malignancies. The various types of malignant tumours include endocrine cell tumours, lymphoma, adenocarcinoma and leiomyosarcoma and others[1].

Adenosquamous carcinomas of the gastrointestinal tract are rare with previously reported tumours found mainly in the large intestine, especially in the caecum. Only 3 such cases were found in the small intestine viz 2 in the jejunum and one in the ileum[2].

CASE REPORT

CH, a 77-year-old Chinese male, presented to our hospital with symptoms of non-specific abdominal colic, lethargy and loss of weight for the duration of one month. Physical examination did not reveal any significant abnormality. He was scheduled for an elective colonoscopic examination. Prior to the colonoscopy, however, he was admitted to hospital with evidence of generalised peritonitis and an erect pain radiograph of his chest showed free intraperitoneal air. An emergency exploratory laparotomy was performed and the operative findings were that of a 3-cm tumour in the terminal ileum with a 5-mm central perforation the tumour mass. There were extensive peritoneal seedlings and multiple enlarged mesenteric lymph nodes were also seen. The serosal surface of the ileum was extensively coated with metastatic nodules necessitating a fair length of resection in order to effect a healthy anastomosis. Postoperative recovery was complicated by bronchopneumonia which resolved with antibiotic therapy. He was discharged from hospital in the third postoperative week. A month later, he was readmitted for abdominal pain and distension; physical examination showed the presence of ascites and tumour masses in the abdomen. He finally succumbed to progression of the disease about 3 months after the initial admission.

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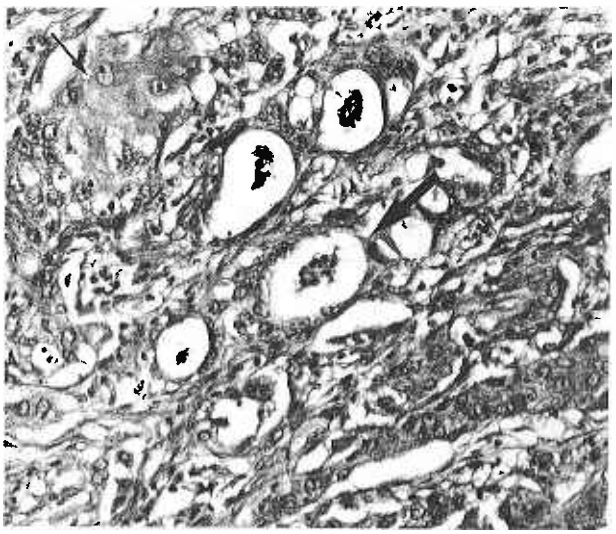
Fig 1- Closely intermixed squamous and glandular components of the tumour. Small arrow- squamous component; big arrow- glandular component. (H & E, magnification 200 X).



MATERIALS AND METHODS

The tissue was formalin-fixed, embedded in paraffin and sectioned with the microtome at 4 micrometre intervals. All sections were stained with haematoxylin and eosin. Periodic acid Schiff's reagent with and without diastase digestion and mucicarmine stain were done on the tumour sections. Immunoperoxidase staining for presence of cytokeratin and carcinoembryonic antigens were carried out using the avidin-biotin peroxidase method.

PATHOLOGY

The resected bowel consisted of a 48-cm length of the terminal ileum with its attached mesentery. There was a 3-cm hard, whitish tumour with a 5-mm central perforation; the tumour had infiltrated all the layers of the ileum and there was a stenotic occlusion of the ileal lumen. Multiple firm enlarged mesenteric lymph nodes were present together with numerous peritoneal seedlings and nodules on the serosa of the bowel wall.

The histological examination of the tumour showed the presence of a mixed squamous and glandular picture, with both elements closely intermixed (Fig 1). Features of the squamous component were sheets of eosinophilic polygonal cells with keratinised cytoplasm and intercellular bridges (Fig 2). Immunostaining test with wide spectrum cytokeratin antibody showed the presence of keratin in the cells. The glandular component showed luminal and intracytoplasmic globules that reacted positively with Periodic acid Schiff's reagent and mucicarmine (Fig 3). These glandular areas were also

Fig 2- Squamous areas show keratinised squamous cells with ample cytoplasm. These cells stain positively with cytokeratin antibody. (H & E, magnification 600 X).

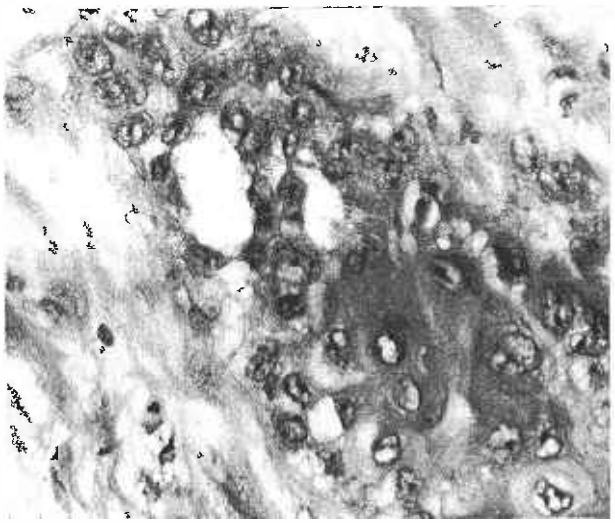
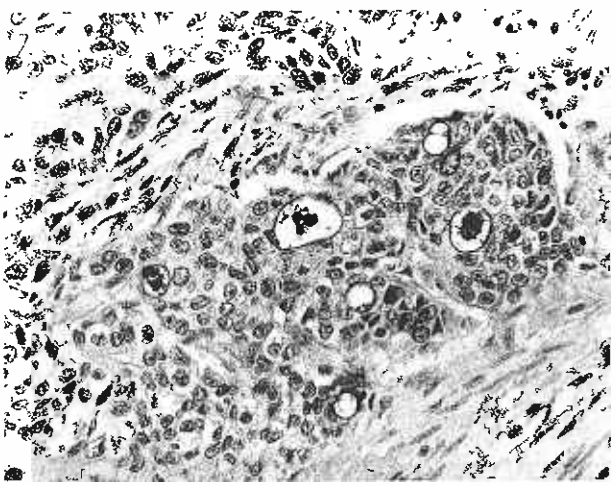


Fig 3- Cytoplasmic and luminal mucin are present in the glandular areas. (Mucicarmine stain, magnification 200 X).



immunoreactive to antibodies to carcinoembryonic antigen. The metastases in the mesenteric lymph nodes and the omental nodule consisted predominantly of the squamous component.

DISCUSSION

Adenosquamous carcinomas of the bowel are very uncommon with a small number of cases previously reported in the large intestine. Such tumours affecting the small intestine are exceedingly rare and only one case located in the ileum had previously been reported[2].

By definition, an adenosquamous carcinoma must contain both adenocarcinomatous and malignant squamous elements. Adenosquamous carcinoma of the bowel have an obscure origin. Wood(1967) suggested that these tumours may have evolved from undifferentiated basal cells of the gut epithelium. In the large colon, Williams (1979) had provided substantial evidence to support the proposal that, in rare instances, adenomas of the colorectum do contain areas of squamous differentiation; squamous and adenosquamous carcinoma may arise from such foci[3].

The presentation of tumours of the small intestine is often vague and nonspecific; in addition, symptoms are often absent until a complication viz perforation, obstruction or bleeding occurs[4]. As a result, survival figures for small intestinal malignancies have not improved over the past few decades despite advances in surgical technology. Radiological contrast studies of the small intestine and the use of sophisticated techniques such as arteriography and Technetium labelled erythrocyte scintigraphy have a low sensitivity rate with a correct preoperative diagnosis in only 31% of patients[5]. Radical resection at an early stage appears to offer the only hope for improved survival.

Our case report and review of the literature reinforce the need for a high index of suspicion in the diagnosis of tumours of the small intestine especially in the older patient with persistent, atypical symptoms with normal upper gastrointestinal and colonic studies[6].

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ANSWER TO THE ELECTROCARDIOGRAPHIC CASE

Diagnosis : Hypokalemia

DISCUSSION

The electrocardiogram in Fig 1 shows marked T wave inversion, prominent U waves, especially in leads aVF and V2 to V6. There is also slight ST depression in V5 to V6. The second complex in leads I, II and III shows abnormally tall and wide P waves. Electrolyte disturbances such as hypokalemia or hypomagnesemia should be suspected. Predisposing causes of hypokalemia should be looked for, namely abnormal gastrointestinal losses due to inflammation, malabsorption or carcinoid syndrome, urinary losses as in renal tubular defects from various causes, use of drugs such as diuretics, laxatives, liquorice containing drugs, and endocrine causes such as hyperaldosteronism, Bartter's syndrome, Cushing's syndrome. On further questioning, the patient admitted having diarrhoea and vomiting for the past three days. The serum potassium was 1.9 mmol/l. Fig 2 shows the reversion of the electrocardiogram to normal when the serum potassium was corrected to 4.0 mmol/l.

The T wave inversion seen here differs from the symmetrical deep arrowhead appearance classically described in subendocardial infarction. In addition, prominent upright U waves are not a feature of subendocardial infarction, although inverted U waves are a sign of coronary artery disease. The effect of low serum potassium on the electrocardiogram is well documented. As the serum potassium is progressively lowered, the following changes sequentially take place: ST segment depression, decrease in T wave amplitude, increase in U wave amplitude, fusion of the T and U wave (resulting in pseudoprolongation of the QT interval), increase in QRS amplitude and duration[1,2], and T wave inversion[3]. Hypokalemia also increases the P wave amplitude and duration and causes first and second degree atrioventricular block[2]. Similar changes may also occur in hypomagnesemia[2]. Hypokalemia also predisposes to cardiac dysrhythmias. It causes early afterdepolarisations which may lead to the development of torsade de pointes, a kind of polymorphic ventricular tachycardia in which the axis seems to revolve around the isoelectric line. Hypokalemia also causes enhanced automaticity. In the presence of hypokalemia, digitalis toxicity is potentiated[4]. Digitalis and potassium occupy the same binding sites on membrane sodium-potassium ATPase and a low serum potassium enables more digitalis to bind to the cell membrane. Progressive hypokalemia decreases the negative transmembrane potential till the cell eventually becomes no longer excitable. Correction of hypokalemia is lifesaving and should be proceeded with immediately using intravenous potassium chloride 40 - 60 mEq/L at 20 mEq/hour; approximately 200 to 250 mEq/day.

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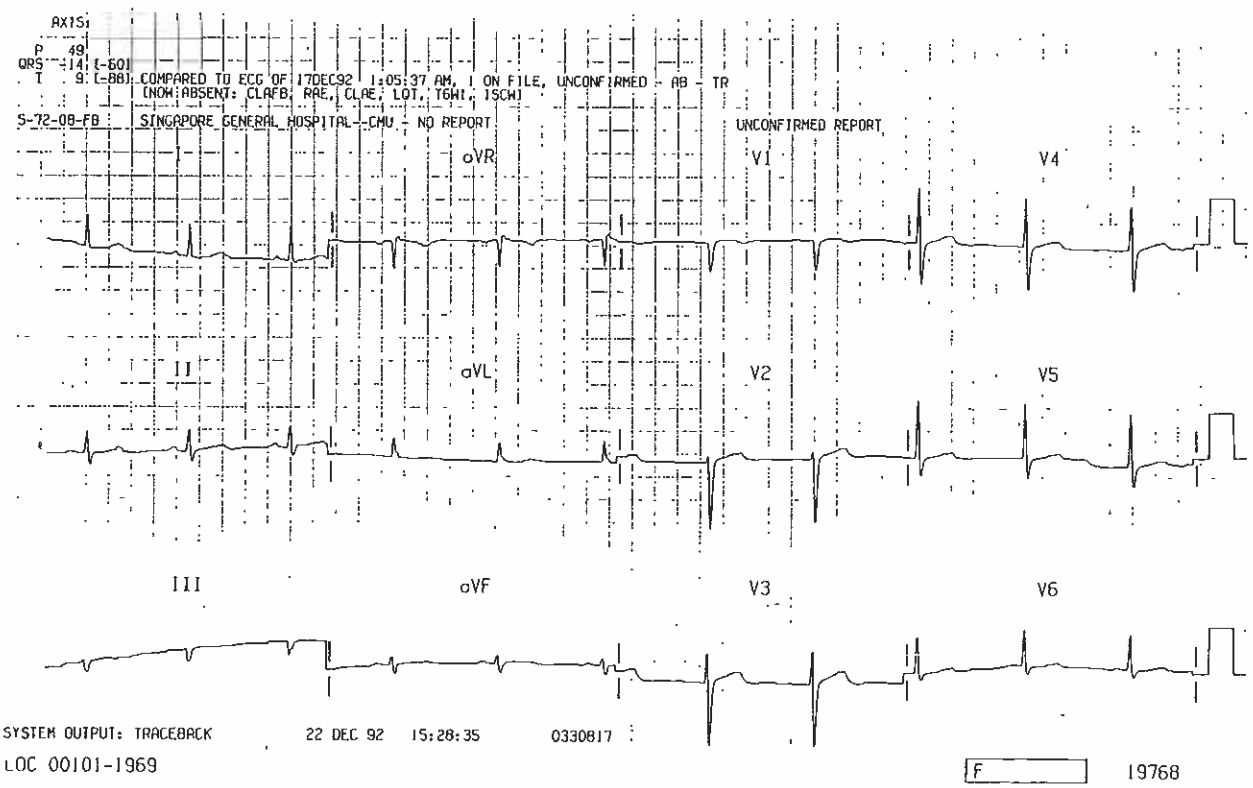
Fig 2 - Electrocardiogram repeated after correction of the serum potassium to 4 mmol/l



Erratum

Letter to the Editor - Review of Handbook of Acute Medicine Singapore Med J 1993; 34: 279

In the letter, the practice address of Dr K M Fock was printed as "Tan Tock Seng Hospital". This is incorrect. Dr K M Fock is the Senior Consultant Physician, Consultant Gastroenterologist & Head, Division of Medicine, Toa Payoh Hospital, Toa Payoh Rise, Singapore.

Our sincere apologies for the error.

-The Editor