

# Synchronous abdominal tuberculosis and adenocarcinoma of the colon

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## Abstract

This case report describes a young man in his early 20s who was found to have synchronous adenocarcinoma of the colon and abdominal tuberculosis. This is a rare finding and it is discussed whether a dampened immune response by the cancer allowed a latent tuberculosis infection to become active or vice versa.

## Keywords

Tuberculosis; adenocarcinoma; colonic adenocarcinoma; colorectal cancer.

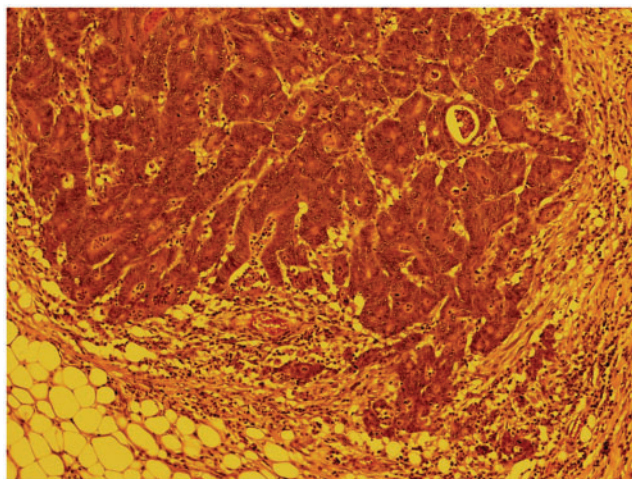
## Introduction

Synchronous abdominal tuberculosis and adenocarcinoma of the colon is a rare finding with only a few reports in the literature coexisting<sup>[1–3]</sup> and none from the United Kingdom. We describe the case of both of these pathologies found in a 21-year-old man and discuss the possible pathogenesis of these 2 lesions.

## Case report

A 21-year-old Asian gentleman had presented to his general practitioner for almost 2 years with altered bowel habit and anaemia before an endoscopic evaluation was performed. He was born in the United Kingdom but he had travelled twice to Mauritius (for 2 weeks at a time) and his father had had tuberculosis 2 years previously after one of the trips. He had no family history of colorectal carcinoma. At colonoscopy an advanced partially obstructing neoplasm in the descending colon was found and this was confirmed on histology to be an adenocarcinoma.

Staging computed tomography (CT) showed (as well as the colonic tumour) some adenopathy of the mediastinum particularly the retrocaval, pretracheal and subcarinal regions. There was also right hilar adenopathy. The lungs were clear apart from a small triangular subpleural bleb. There was definite adenopathy of the right internal iliac region and possibly the para-aortic region. The liver was free of metastatic disease. There was some discussion at the multidisciplinary meeting



**Fig. 1.** Typical colorectal carcinoma with fat in the bottom left corner indicating Dukes B (magnification  $\times 25$ ).



**Fig. 2.** Several granulomata, including multinucleate giant cell, with early central necrosis (magnification  $\times 10$ ).

as to whether he should have a mediastinoscopy first to get a biopsy of one of these nodes but it was decided to go straight to surgery to avoid any delay in treating the colorectal tumour.

At operation, an obstructing descending colon tumour with associated small node lymphadenopathy was found. Surprisingly there were also 2 distinct areas of probable neoplastic tissue in the ileum associated with massive lymphadenopathy to the root of the mesentery. There was no liver lesion. A left hemicolectomy and ileal mesenteric lymph node excision was performed.

A left hemicolectomy and separate ileal lymph node were received by the pathologists. Thirty-five millimetres from one resection margin of the colon was an annular tumour infiltrating into subserosal adipose tissue. Appearances were typical of a carcinoma. The lymph node measured 20 mm in maximum dimension and was necrotic.

Microscopy confirmed an adenocarcinoma of colon infiltrating subserosal adipose tissue to a depth of 10 mm. None of the 18 lymph nodes identified were involved by tumour nor did they show any evidence of granuloma formation. Overall stage was Dukes B, G2 T3 N0 MX (Fig. 1).

The separate lymph node showed numerous granulomata which were becoming confluent in areas. Langhans type giant cells were present. Non-suppurative necrosis, including caseation necrosis, was present. Stains for microorganisms, including acid-alcohol fast bacilli (AAFBs), were negative. The appearances were thought to be most likely due to tuberculosis (Figs. 2 and 3). He had a quantiferon tests and the results were inconclusive. It was decided on balance to treat this as tuberculosis.

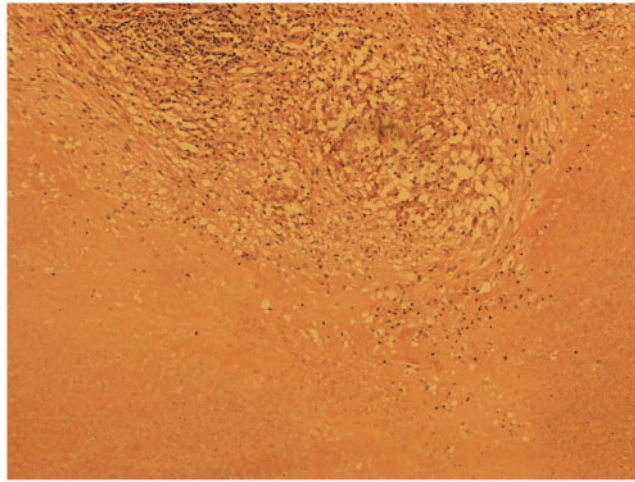


Fig. 3. Caseation necrosis in the bottom half with granulomata in the top (magnification  $\times 40$ ).

On further questioning of the patient he reported a long-standing history of sweats, fevers and weight loss of 12.7 kg. He was referred for anti-tuberculosis therapy and then on for adjuvant chemotherapy. After seeing the oncologists and discussing chemotherapy, it was decided that he would only have small benefit from chemotherapy so he decided against it. He went straight on to have treatment for his tuberculosis and this lasted for 6 months. He was offered human immunodeficiency virus (HIV) testing but as he did not have any risk factors he declined.

## Discussion

The abdomen is one of the commonest sites of extrapulmonary tuberculosis and abdominal tuberculosis tends to be a disease of young adults. It typically occurs in the terminal ileum or ileocaecal junction where there is an abundance of lymphoid tissue and a high rate of absorption and prolonged stasis, although it has multiple ways of manifesting itself and can affect lymph nodes, any part of the intestinal tract, peritoneum and solid organs<sup>[1,4]</sup>.

It has been proposed that it can be spread by ingestion of contaminated food, by swallowing infected sputum, by haematogenous spread from the primary lung focus in childhood with later reactivation, by retrograde lymphatic spread or direct spread from infected organs. Gross pathologic findings may include transverse ulceration, fibrosis, thickening and structuring in the bowel wall as well as mesenteric lymphadenopathy, omental thickening and peritoneal tubercles. Clinical features include pain, fever, weight loss, diarrhoea and constipation, anorexia and malaise but depend on the affected site<sup>[4]</sup>.

The coexistence of the abdominal tuberculosis and adenocarcinoma of the colon is rare and it is unclear if it is coincidence or if they predispose to each other. This patient was referred late as his symptoms, although typical of colon cancer, were in an atypical age group. It has been previously noted that the age characteristics of patients with these coexisting pathologies occurs in the younger age group<sup>[1]</sup>. This raises the possibility that the tuberculosis is causing an immunosuppressed state allowing the development of colorectal neoplasia. Indeed, it is very unusual for someone of this age to have a colorectal cancer, which was a significant factor in his delayed diagnosis.

An alternative hypothesis is that he had a latent tuberculosis infection that became active when he got his colorectal cancer as this made him immunosuppressed. His quantiferon test was inconclusive and this result tends to occur in an immunosuppressed state.

## Teaching points

- Patients born in high incidence parts of the world have a higher risk of latent and therefore active tuberculosis, more so the latter if they become immunocompromised. We should be aware of this when treating cancer patients.

- This has a bearing on giving chemotherapy to patients as this too increases their immunocompromise.
- Cancer patients who have risk factors for tuberculosis should be screened with a quantiferon test first and consider tuberculosis chemoprophylaxis for those who are positive or have an inconclusive result.

## References

1. Husain M, Sachan PK, Khan S, Chauhan N, Kishore S. Coexisting abdominal tuberculosis and colon carcinoma: case report with review of the literature. *Internet J Surg* 2007; 13.
2. Tanaka K, Kondo S, Hattori F, *et al.* A case of colonic carcinoma associated with intestinal tuberculosis and an analysis of 26 cases reported in Japan. *Gan No Risho* 1987; 33: 1117-23.
3. Jain BK, Chandra SS, Narisimhan R, Ananthkrishnan N, Metha RB. Coexisting tuberculosis and carcinoma of the colon. *Aust NZ J Surg* 1991; 61: 828-31. doi:10.1111/j.1445-2197.1991.tb00166.x.
4. Sharma MP, Bhatia V. Abdominal tuberculosis. *Indian J Med Res* 2004; 120: 305-15.