loops of small bowel. No discrete lymph nodes were identified and no free air or free fluid was seen within the abdominal cavity. A colonoscopy study was scheduled in view of the CT findings, which showed a friable, stenotic tumour, 68 cm from the anal verge, which extended 10 cm proximally obstructing approximately 80% of the bowel lumen. Histological examination of a biopsy of the lesion showed adenocarcinoma. Other significant laboratory findings included decreased haemoglobin (10.6 mg/dL), elevated carcinoembryonic antigen (19.46 ng/ml) and C-reactive protein (36.9 mg/dL).

Because of the CT findings, the working preoperative diagnosis of this patient was stage IV colon cancer. Definitive surgical management in this case was guided by not only the radiological staging, but also by intra-operative findings, which showed loops of small bowel firmly adherent to the descending colon, possibly representing an advanced stage of the disease. A left hemicolecction with en-block resection of the jejunum was performed. The surgical specimen was subsequently submitted to the histopathology laboratory.

CASE REPORT
The patient was a 69-year old female with medical history of diabetes mellitus, hypertension and arthritis. Surgical history included a Caesarean section 28 years previously, laparoscopic cholecystectomy in September 2008, repair of suture sinus tract in December 2008, vaginal hysterectomy in 2010 and the most recent being a supra-umbilical hernia repair (with mesh) done in February 2013. She presented to hospital ten weeks later with abdominal pain for two days, with increasing intensity, and diarrhoea. Computed tomography (CT) imaging showed thickening and enhancement of the middle portion of the descending colon with features suggestive of a primary colonic lesion, which involved adjacent loops of small bowel. No discrete lymph nodes were identified and no free air or free fluid was seen within the abdominal cavity. A colonoscopy study was scheduled in view of the CT findings, which showed a friable, stenotic tumour, 68 cm from the anal verge, which extended 10 cm proximally obstructing approximately 80% of the bowel lumen. Histological examination of a biopsy of the lesion showed adenocarcinoma. Other significant laboratory findings included decreased haemoglobin (10.6 mg/dL), elevated carcinoembryonic antigen (19.46 ng/ml) and C-reactive protein (36.9 mg/dL).

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The postoperative recovery of the patient was adequate and she was eventually discharged, to be followed-up by the surgical and oncology teams, once diagnosis and pathological staging was complete.

The surgical specimen was received in 10% buffered formalin, and included a segment of large bowel, 20 cm in length, with a firmly adherent segment of small bowel 8.0 cm in length. External examination showed a segment of small bowel firmly attached to the serosa of the colon by firm, irregular, tan grey tissue. The colonic segment was opened along its longitudinal axis, revealing a firm, fungating, grey tumour, 6 cm in greatest dimension, arising from the mucosa, and which on transverse sections appeared to invade the bowel wall. The subjacent pericolic fat showed a poorly demarcated, firm, grey infiltrating lesion which appeared to be continuous with the invasive colon tumour, and the muscle wall of the adjacent small bowel appeared to be involved. The small bowel mucosa was elevated at that point but did not appear to be involved by the tumour. Multiple matted and discrete lymph nodes were identified in the pericolic tissue.

Representative sections were taken from the descending colon tumour, the pericolic tissues, the adherent loop of small bowel, and the large and small bowel margins. Regional lymph nodes were also sampled. These were processed routinely, and 5.0 µm sections were cut and stained with haematoxylin and eosin (H&E).

Microscopic examination of sections taken showed a well-differentiated adenocarcinoma with invasion of the wall of the colon as far as the muscularis propria without extension into the pericolic fat. Present at the leading edge of the tumour (Fig. 1) were extensive sheets of histiocytes which extended outwards in an infiltrative fashion to involve the wall of the colon adjacent to the tumour, the pericolic fat and the muscular layer of the adherent small bowel. The histiocytes were large with small round nuclei and abundant, acidophilic, granular cytoplasm.

Also present within the infiltrate but in lesser numbers were lymphocytes, plasma cells and scattered eosinophils. Many of the histiocytes contained round, basophilic intracytoplasmic concretions pathognomonic for malakoplakia – the Michaelis-Gutmann bodies (Fig. 2). Examination of the lymph nodes showed no evidence of metastatic adenocarcinoma, but malakoplakia was present. Hence, based on the histologic evidence, the patient was diagnosed with well differentiated invasive adenocarcinoma of the colon, Dukes' A, pT2 pN0, (American Joint Committee on Cancer (AJCC), 7th edition), associated with malakoplakia of the large and small bowel and lymph nodes.

**DISCUSSION**

The association of malakoplakia and colonic adenocarcinoma is rare, with the majority of cases described in the literature occurring in the rectum and sigmoid colon (1, 2) and with a male predominance of 4:1 (1). Only one other case has been reported in the descending colon. Largely, because of its rarity, the pathogenesis of the malakoplakia in cases of adenocarcinoma is uncertain. Possible causes include infections *eg* *E coli* and *Klebsiella* spp, an abnormal immune response and a disorder of macrophage response related to defective lysosomal function (3). Alteration in the normal gut flora and an unusual stromal response to tumour are other postulated mechanisms. Although there have been claims of an association between steroid use and malakoplakia (4), relatively few such cases have been reported.

Where the two conditions co-exist, malakoplakia almost always occurs adjacent to the invasive tumour and is usually a localized phenomenon, lending some support to the hypothesis of an unusual stromal response to the carcinoma. In our case, the malakoplakia extended from the invasive...
margin of the tumour, a feature which is in keeping with other reports (2, 5). Malakoplakia also involved the muscularis propria, pericolic fat and regional lymph nodes. The adherence to loops of small bowel in turn mimicked direct tumour invasion – both on CT imaging and at surgery.

Previous reports have commented on the likelihood of overstaging the cancer where there is pericolic involvement and adherence to other intra-abdominal structures eg bladder, pelvic wall or small bowel (1, 4, 6) and we would wish to emphasize this point. Overstaging may result in attempts at palliative therapy or to referral of the patient for neoadjuvant chemotherapy. Extensive examination of the tumour in our patient revealed no spread beyond the muscularis propria and no metastases to regional lymph nodes, placing it as a Dukes’ stage A tumour (pT2, AJCC).

The largest meta-analysis of cases of malakoplakia and colonic adenocarcinoma (1) shows that Dukes’ stage B tumours are overwhelmingly the most common stage at surgery and our case is one of the few reported cases of a Dukes’ stage A tumour associated with malakoplakia. Apart from the possibility of errors in staging and treatment of the cancer, malakoplakia itself does not appear to alter the prognosis of the tumour.

REFERENCES