colon showed active inflammation with pronounced mucosal fissuring ulceration that extended through the bowel wall. The appearances were consistent with the diagnosis of pseudomembranous colitis.

Comment

Pseudomembranous colitis is a well recognised complication of treatment with oral antibiotics and causes severe and persistent diarrhoea. The diagnosis is confirmed by sigmoidoscopy and rectal biopsy and by isolation of the *Clostridium difficile* organism or toxin. Treatment consists of maintaining fluid balance and administering oral vancomycin 500 mg four times daily. In cases which fail to respond to these measures, diarrhoea may persist and toxic megacolon or septicaemia may develop. In these circumstances surgical resection of the colon may be necessary.[1-3] There have been only two cases reported of perforation of the colon secondary to pseudomembranous colitis.[4,5] Both patients were extremely ill and had obvious signs of generalised peritonitis.

There has been no previous report of a patient with pseudomembranous colitis developing pneumoperitoneum without evidence of generalised peritonitis, systemic toxicity, or frank perforation. We assume that in our patient the very thin wall of the inflamed colon allowed the transudation of gas but not the escape of liquid faeces. In the absence of faecal contamination of the peritoneal cavity signs of peritonitis did not develop.

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(*Accepted 2 February 1984*)

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Carcinomatous meningitis diagnosed with monoclonal antibodies

The diagnosis of neoplastic meningitis is confirmed by the demonstration of malignant cells in the cerebrospinal fluid, but such cells can be difficult to identify in conventional cytological preparations. By using monoclonal antibodies as markers for tumour cells in cerebrospinal fluid we successfully diagnosed carcinomatous meningitis in a patient in whom previous investigations had given negative results.

Case report

A 57 year old woman presented with a six month history of anorexia and weight loss followed by a deep vein thrombosis and bilateral pleural effusions that were drained but did not show malignant cells on cytological examination. A computed tomogram of the body obtained at that time was considered to be normal. She subsequently became confused and ataxic, at which time a computed tomogram of the brain showed hydrocephalus. An initial lumbar puncture showed clear cerebrospinal fluid at a pressure of 37 cm H_2O containing 10^6/l white cells (1/mm^3), a normal protein concentration of 0·35 g/l, but a low glucose concentration of 0·5 mmol/l (9 mg/100 ml) (blood glucose concentration 8·2 mmol/l (148 mg/100 ml)). No malignant cells were seen on cytological examination. A second specimen of cerebrospinal fluid gave almost identical results. By this time physical examination showed a possible pelvic mass, and neoplastic meningitis was diagnosed. She was admitted to the department of neurosurgery at Frenchay Hospital, Bristol, where a third lumbar puncture was performed.

Cytological examination showed a scanty deposit of degenerate cells, which were interpreted as being reactive rather than neoplastic. Monoclonal antibody testing was carried out on this sample using indirect immunofluorescence on unfixed cytospin preparations. We used a panel of antibodies comprising markers for epithelial cytokeratin, neuroectodermal cells, leucocytes, and carcinoembryonic antigen. The table shows the results. Immunostaining with LE61 showed scattered cells of epithelial origin, some of which were vacuolated. These cells contained carcinoembryonic antigen as shown by L11/285/14. All other cells were confirmed as being leucocytes, mostly T lymphocytes. No cells of neuroectodermal origin were found.

These findings led us to suggest several further investigations including gastrocopy, which resulted in a diagnosis of gastric carcinoma. When she ultimately died necropsy showed linitis plastica with ovarian metastasis and widespread meningeal infiltration.

Results of immunofluorescence tests with a panel of monoclonal antibodies

Antibody	Reference	Antigen	Result
LE61	1	Epithelial cytokeratin	Positive
L11/285/14	Unpublished	Carcinoembryonic antigen	Positive
2D1	2	Panleucocyte	Positive
UCHT1	3	T cell membrane	Positive
UJ13A	4	Pan-neuroectodermal	Negative

Comment

The difficulties of making a swift and accurate diagnosis of neoplastic meningitis are shown by this case, in which repeated cytological examination of the cerebrospinal fluid gave negative results. The use of appropriate monoclonal antibodies in a straightforward immunofluorescence test resulted in the confident identification of carcinoma cells, which were also shown to contain carcinoembryonic antigen. Cytological appearances of the cerebrospinal fluid generally deteriorate rapidly, but use of the marker LE61 for cytokeratin confers the advantage that the cerebrospinal fluid can be tested at leisure or after transport to a specialised laboratory as the antigen remains stable for at least three days (personal observations).

Monoclonal antibodies that recognise tissue differentiation antigens and tumour associated antigens appear to be ideal markers for identifying cells in any cytological preparation but have only recently been used in the cytological examination of cerebrospinal fluid.[5] Our experience with this patient and others examined in the past two years suggests that immunofluorescence testing with monoclonal antibodies will greatly increase diagnostic accuracy in neoplastic meningitis, with resulting impact in clinical management.

We thank Mr H B Griffith for permission to report this case and Drs J T Kemshead, P C L Beverley, and C H Ford for generously providing antibodies. We acknowledge the support of the Bristol Brain Tumour Research Fund, the Francis and Augustus Newman Foundation, and the Medical Research Council.

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(*Accepted 25 January 1984*)

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