Does omentectomy prevent malignant small bowel obstruction?

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Because the omentum collects and disseminates cancer cells, omentectomy is an integral part of ovarian cancer surgery. We postulate that the omentum serves a similar function in colon cancer and may contribute to post-operative malignant small bowel obstruction (S.B.O.) and that routine omentectomy during colectomy would reduce the incidence of S.B.O. Fischer 344 rats and a transplantable carcinogen-induced rat colon cancer were used to test: (1) whether the omentum is a unique site of intra-abdominal colon tumor implantation which contributes to S.B.O.; and (2) whether omentectomy at the time of tumor implantation would reduce the incidence of S.B.O. Statistical analysis confirmed that animals undergoing omentectomy had a significantly lower incidence of omental tumors and malignant S.B.O. (26 per cent and 16 per cent respectively) when compared with sham operated animals (75 per cent and 85 per cent respectively, $P < 0.001$). These data suggest that the omentum is a source of bowel obstruction from implantation and growth of tumour cells in the rat model. Although this could be tested in other animal systems, the addition of routine omentectomy to colectomy is simple, not time-consuming, and may reduce postoperative morbidity.

Introduction

Omentectomy is considered to be an important part of debulking procedures for patients with ovarian cancer, because the omentum collects and disseminates tumor cells. Frequently, large tumor deposits are found in the omentum which can be responsible for episodes of small bowel obstruction (S.B.O.), because of tumor adherence to viscera and the abdominal wall. We postulate that the omentum behaves similarly in colon cancer and may contribute to postoperative malignant S.B.O. We further postulate that routine omentectomy during colectomy will reduce the incidence of S.B.O. in colon cancer patients. These postulates were tested using a rat colon cancer model system.

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Methods

Tumor production

The tumor used in these studies is a rodent, dimethylhydrazine (DMH) induced, poorly differentiated adenocarcinoma originally developed from a mesenteric metastasis in a colon tumor-bearing rat [10]. It is maintained in our laboratory in subcutaneous (s.c.) passage in Fischer 344 (F344) rats. For these experiments, a s.c. tumor was removed and necrotic areas were resected. The tumor was cut into small pieces with scissors and passed through stainless steel screens to generate single cell suspensions. Tumor cells were washed three times in Roswell Park Memorial Institute (RPMI) 1640 medium supplemented with 20% heat-inactivated fetal bovine serum (FBS) and HEPES buffer, stained for viability with trypan blue dye, and counted. Tumor cells were re-suspended at a concentration of $1 \times 10^7$ viable cells in 1 ml of complete medium.

Animal groups

Four groups of 100 g F344 male rats were studied in three separate experiments. All rats were anesthetized with 3.5% chloral hydrate (1 ml/100 g given intraperitoneally) (i.p.). Rats in Group 1 were injected with $10^7$ viable tumors cells i.p. Rats in Group 2 underwent abdominal exploration through a midline incision and the viscera were removed from the abdominal cavity, left exposed for approximately 15 minutes, and then returned to the abdominal cavity. Prior to closure with stainless steel clips, animals were injected with $10^7$ tumor cells i.p. Rats in Group 3 were explored through a midline incision, and the dependent portion of the omentum and the fatty appendages on the epididymis were removed using titanium clips for hemostasis. Prior to closure with stainless steel clips, these animals were also injected with $10^7$ tumor cells i.p. Group 4 rats underwent a flank incision into the paraspinous muscles without entering the peritoneal cavity prior to i.p. injection of $10^7$ tumor cells. Surviving animals were sacrificed at 26 weeks (by overdosage with chloral hydrate), and underwent complete autopsy. Those dying prior to this time also underwent complete autopsy. The incidences of omental metastases (omento remnant metastases in Group 3) and of malignant S.B.O.s were determined.

Results

The incidences of omental tumor and malignant S.B.O. in the four groups of animals is shown in the table. Omental tumor was easily visualized as a large mass infiltrating and, in many cases, replacing the omentum. Thirty-five out of 47 (75 per cent) animals in the sham-operated group and 10 out of 13 (77 per cent) rats in the flank incision group developed omental metastases compared to 19 out of 43 (44 per cent) in the non-operated, control group. Eleven of 43 (26 per cent) rats in the omentectomy group had tumor deposits in either the lesser omentum (superior to the stomach) or in the omental remnant.

At autopsy, an animal was considered to have a malignant S.B.O. if an obstructing tumor mass was identified with a dilated proximal segment of small bowel and non-distended small bowel distal to this obstruction. Forty of 47 (85 per cent) sham-operated animals and 8 of 13 (62 per cent) flank incision rats developed malignant S.B.O.s, whereas only 13 of 43 (30 per cent) non-operated animals and 7 of 43 (16 per cent) omentectomy animals were noted to have malignant S.B.O. Six of the seven omentectomy rats with S.B.O. had tumor growing in the omental remnant;