Post-Laparoscopic Cholecystectomy Port-Site Metastases from Previously Undiagnosed Upper Gastro-Intestinal Malignancies - Our Experience of Six Cases
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INTRODUCTION

With the advent of minimal invasive surgery in a big way there is an increased enthusiasm among surgeons for laparoscopy even in cases of intra-abdominal malignancies. Recurrence of the tumor in abdominal incision is more common after laparoscopic surgeries in comparison to open surgeries[1] although it is well documented but not supported by many in the surgical fraternity.

In the last one and a half years, we have received six cases of port-site metastases who had undergone laparoscopic cholecystectomy for cholelithiasis with symptoms of recent onset dyspepsia. Port-site metastases occurred within 1-3 months postoperatively. There was no evidence of malignancy in the gall bladder peroperatively and in the histopathological specimen. All these cases were subsequently proved to have upper gastrointestinal malignancies, missed or undiagnosed at the time of laparoscopic cholecystectomy.

MATERIAL AND METHODS

In the last one and a half years we have received six cases of port-site metastases who had undergone laparoscopic cholecystectomy for cholelithiasis with symptoms of recent-onset dyspepsia at various private hospitals. Preoperative ultrasonography of the abdomen of all the patients was suggestive of cholelithiasis.

After surgery, all the patients had complaints of progressive dysphagia, anorexia and pain in the epigastrium. All the patients developed hard lumps at the port sites within 1-3 months after surgery (figure 1, 2). Thereafter, they were referred to our hospital for further management.
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**RESULTS**

A detailed history, clinical examination and investigations were carried out in all six patients. Clinical examination revealed hard, erythematous, ill-defined, and tender lumps fixed to underlying skin. The overlying skin was ulcerated in two of the patients. The investigations done in the patients were fine-needle aspiration cytology and biopsy from port-site lump, previous slide review, and CECT of the whole abdomen (figure 3, 4), upper gastro-intestinal endoscopic biopsy and tumour markers.

All patients were diagnosed to have upper GI malignancy, which was undiagnosed or missed at the time of laparoscopic cholecystectomy. A repeat histopathological examination of the GB specimen and review of previous slides in all the six cases at our hospital confirmed chronic cholecystitis with no evidence of malignancy in gall bladder specimen.

FNAC from the lumps in all patients showed metastatic adenocarcinoma (Figure 5).

These patients were subjected to UGIE that revealed a growth at the antrum in four patients and at the GE junction in two patients.

Biopsy of all patients was suggestive of adenocarcinoma. There were liver metastases and ascites in three of the patients and we failed to understand if these were missed or absent at the time of laparoscopic cholecystectomy. If that be the case, the tumour was very aggressive.

**Figure 1**

Fig. 1: Clinical photograph of a patient having port site metastases with overlying skin ulceration and erythema

**Figure 2**

Fig. 2: Clinical photograph of a patient having metastases at all the four port sites

**Figure 3**

Fig. 3: CECT of the abdomen showing a port-site lump

**Figure 4**

Fig. 4: CECT of the abdomen showing an anterior abdomen wall lump and a gastric growth
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DISCUSSION

The implantation of tumor cells at the trocar insertion or incision site is variously termed as port-site tumor recurrence, port-site or incisional metastases, port-site or trocar-site wound recurrence, wound or port-site tumor implantation and wound recurrence. The incidence of port-site metastasis after laparoscopic cholecystectomy for gallbladder cancer is estimated to be 12.5% to 29%.[2,3,4]

Since the earliest published reports on tumor growth at trocar insertion ports after laparoscopic surgery for intra-abdominal malignancies, a large body of literature has been published on various aspects and possible mechanisms of tumor seeding during laparoscopic surgery of the gastrointestinal tract.[5]

Although some data suggest that hematogenous spread may be one possible mechanism of tumor implantation into the operative wound, most clinical and experimental studies agree that direct tumor cell implantation is indeed the major contributor to port-site tumor growth.

Etiology of port-site metastases is not fully understood. Some postulated pathophysiological mechanisms are:

Direct implantation of tumor cells during laparoscopic handling of the tumor; perforation of a viscus with tumor during extraction or spillage of its contents; creation of pneumoperitoneum produces a high pressure system within the peritoneal cavity; during gas insufflation there exists a pressure gradient between peritoneal cavity and the environment; and when there is intraabdominal malignancy there is an increased risk of wound metastases. Aerosol theory and chimney effect during turbulence of insufflation have been proposed as causative mechanisms for tumor cell implantation at port sites.

Carbon dioxide may influence growth of tumor cells by providing an acidic environment which may activate the enzymes of cell cycle within implanted tumor cells.[6] Peritoneal trauma may also predispose to tumor cell invasion into the abdominal wall.[7]

Some measures can be adopted in order to decrease the incidence of PSM such as: avoiding contamination of port sites or spillage from the viscus; the use of an Endo-Catch bag[8] or similar devices is to be encouraged; tumour implantation following laparoscopy is promoted by the presence of intra-peritoneal blood and that this effect may be reduced by the use of intra-peritoneal heparin.[9]
The incidence of wound metastases may be decreased by gasless laparoscopy,[10,11] or helium insufflation, closure of the peritoneal defect, intraperitoneal irrigation with chemotherapy solution and topical application of povidone iodine (tumoricidal).[12,13]

Although the port-site metastases are reported all over the world, this clinicopathologic case series was unique as there was no direct tumour handling at the time of laparoscopy. The probable mechanism must have been the contamination of port sites due to floating tumour cells in the peritoneal cavity at the time of surgery.

All six patients in our series had dyspepsia as the major symptom. This was attributed to gall stones as gall stone patients usually present with dyspepsia. But occurrence of port-site metastases from undiagnosed UGI malignancies suggests that care should be exercised in attributing dyspepsia to gall stone disease only, and an UGI endoscopy may be included as an essential preoperative investigation tool, especially in middle-aged and elderly patients.

This clinical data provides clues to understanding the development of port-site metastases. Seeding can occur through any one of the above mechanisms. As the mechanisms for tumour recurrence at port and trocar sites are still poorly understood, practical recommendations are difficult to make. Although this clinicopathologic case series does not answer the question of how port-site metastases occur, our patients provide strong clinical evidence to support the direct implantation theory for developing port-site metastases.

References

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