Gallstone Embedded within a Port Site Metastasis – Report of a Case

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ABSTRACT

The occurrence of port site metastasis after laparoscopic cholecystectomy for an incidental gallbladder carcinoma is well-documented but the underlying aetiology is not clear. Several mechanisms including tumour implantation to the effects of carbon dioxide usage have been implicated. Here, we describe an unusual case of a late port site recurrence in a 60-year-old woman where a gallstone was found embedded within the heart of the recurrence. We critically review the basic and clinical evidence that contributes to the pathophysiology of this phenomenon and the surgical strategies employed.

Keywords: gallbladder carcinoma, port site metastases, laparoscopic cholecystectomy, pneumoperitoneum and implantation

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INTRODUCTION

The phenomenon of port site metastasis from an intra-abdominal malignancy after laparoscopic surgery is well documented[1-4]. The number of reports in the literature has risen with the increasing use of minimally invasive procedures for management of both benign and malignant disease. However, the aetiology of this form of metastasis remains unclear. Tumour implantation at the port site during removal of the abdominal viscera is one widely accepted theory[5]. Incidental gallbladder carcinomas removed at the time of laparoscopic cholecystectomy are subjected to this complication. Here, we describe an unusual case of a late port site metastasis where a gallbladder calculus was implanted at the port site that formed the heart of the recurrence and consider the pathophysiological mechanisms that underlie this phenomenon. To our knowledge, this is the first case reported where a coexistent gallstone was found embedded within the port site metastasis.

CASE REPORT

A 60-year-old Indian lady presented to us with a one-month history of an abdominal wall lump that was increasing in size. Significantly, she had a laparoscopic cholecystectomy done two years and nine months ago. The gallbladder was removed through the infra-umbilical port with no record of gallbladder perforation or bile spillage. The histology at that time was of a moderately differentiated adenocarcinoma of the gallbladder that had infiltrated the mucularis propria (stage 2). She refused further treatment despite the initial diagnosis and defaulted follow-up. A computed tomography of the abdomen showed a 7 X 5.5 cm heterogeneous mass arising from the substance of the anterior abdominal wall. Within the mass, a sub-centimeter calcification consistent with a gallstone was seen (Fig. 1). There was no evidence of local recurrence at the tumour bed or of metastatic spread.

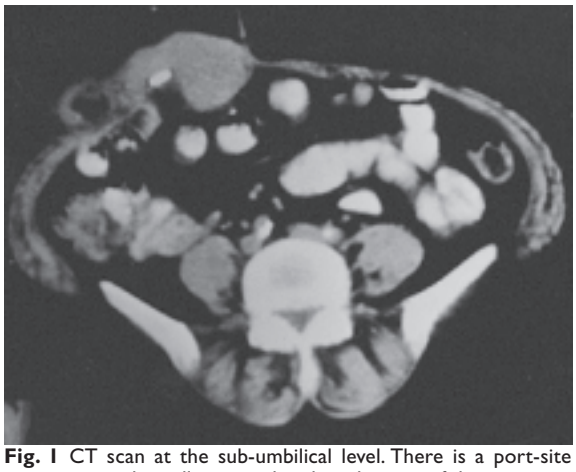

Fig. 1 CT scan at the sub-umbilical level. There is a port-site recurrence with a gallstone within the substance of the mass.

A wide excision of the anterior abdominal wall mass was undertaken with a polypropylene mesh repair for the defect. Cut section of the mass identified the gallstone and histology confirmed adenocarcinoma arising from the gallbladder. Her recovery was unremarkable and the patient remains disease free 18 months after surgery.

DISCUSSION

Since the original description of cutaneous seeding of gallbladder cancer after laparoscopic cholecystectomy[1],

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the report of this phenomenon is increasing. The actual incidence of port site metastasis after laparoscopic cholecystectomy for gallbladder cancer is estimated to be 12.5% to 29%[4,6-8]. The aetiology of port site metastases is not known but there are several postulated pathophysiological mechanisms.

One theory for port site metastases is that of "implantation". Direct implantation of tumour cells presumes a mechanical origin for metastases when the gallbladder is extracted through a small incision with direct tumour to wound contact. The specimens are often forcibly extracted with considerable trauma and manipulation. Perforation of the gallbladder is a common finding during laparoscopic cholecystectomy and may occur at the time of the extraction with deposition of tumour cells at the port site. In our patient, perforation of the gallbladder must have occurred at the time of extraction with implantation of a spilled gallstone within the laparoscopic tract. Simultaneously, tumour cells from the perforated gallbladder seeded the tract that has led to this recurrence. Anecdotal evidence from our patient's findings supports the direct implantation theory.

However, there are several criticisms of this theory. Firstly, if implantation was the only mechanism of spread, it is expected that there should be reports of incision or scar recurrence after open cholecystectomy. Yet in our literature search, this appears to be a rare event with only a single study by Lundberg et al[9] addressing this issue. In this study of 186 patients from the Swedish registry, 12 (6.5%) patients had wound recurrence from open cholecystectomy compared to 16% from laparoscopic cholecystectomy. Secondly, there are reports of port site metastases in patients undergoing laparoscopic cholecystectomy for incidental disease confined to the mucosa where the gallbladder is excised intact and without bile spillage[10]. Thirdly, the precautionary use of a cellophane Endo-bag for extraction to prevent direct contact and implantation still yielded 8 (11.5%) cases of port site recurrence in 70 patients with non-apparent gallbladder carcinoma in a multi-centre survey by Paolucci et al[11]. Finally, there are reports of trocar site metastases from other than the extraction port[12,13]. These suggest that there are other biological causes for port site metastases to occur.

The role of laparoscopy has been implicated and investigated in light of these findings. The creation of a pneumoperitoneum produces a high-pressure system within the peritoneal cavity. When an intra-abdominal malignancy like that of an incidental gallbladder carcinoma is present, there is an increased risk of wound metastasis. This has been demonstrated using a rat model[14] where trauma to an implanted tumour was five times more likely to produce wound metastasis in a laparoscopic model than in a laparotomy model. Two mechanisms have been suggested to account for this. First is the "aerosol" theory where exfoliated tumour cells are disseminated to the port sites during the turbulence of insufflation and secondly the "chimney" effect produced by gas leaks through port sites[15]. This occurs due to the movement of gas during insufflation and the pressure gradient that exists between the peritoneal cavity and the environment. To support this, an elegant experimental rat model reported by Mathew et al[16] demonstrated the presence of wound metastases in most rats undergoing insufflation but in none of the rats in the gasless laparoscopy group. Using radiolabelled tumour cells, they were also able to show increased numbers of tumour cells in the gas vented from the insufflation group compared to the gasless laparoscopy route. However, these results have not been universally replicated and tumour cells in aerosol form have been difficult to identify in clinical and experimental models[17,18].

Carbon dioxide is used universally to create the pneumoperitoneum for laparoscopic procedures. It has been postulated that carbon dioxide may influence the growth of tumour cell growth in the laparoscopic model to account for port site recurrence. In rat models, tumour cell growth was increased significantly exvivo and in vivo after insufflation with carbon dioxide as compared to a helium or atmospheric air[19]. The mechanism of promotion of tumour cell growth by carbon dioxide is not clear. The acidic environment created by carbon dioxide may activate enzymes of the cell cycle within implanted tumour cells. Host defence mechanisms in the form of peritoneal macrophages are affected by carbon dioxide secondary to a decrease in extracellular pH[20]. Interleukin-1β, which promotes cell growth, may be increased and conversely TNF response is decreased. The use of gasless laparoscopy circumvents the use of CO2 and has been shown to reduce port site recurrence[21,22].

A third possibility for preferred spread to port sites during laparoscopy is the role of site-specific surgical trauma to the peritoneum. The peritoneum is believed to serve as an effective barrier against tumour cell invasion into the abdominal wall. Hence trauma to the peritoneum at trocar insertion sites may assist in tumour cell implantation and growth. Aoki et al[23] reported on a mouse model where cultured human gallbladder cancer cells were injected intra-peritoneally after laparoscopy. Three

arms were compared: a control arm where no surgical procedure was carried out, a peritoneal injury arm and a peritoneal injury with repair arm to address this issue. No control mice showed tumour recurrence. In the laparoscopic model, 90% showed port site recurrence. Interestingly, the arm with peritoneal injury and repair showed only 40% port site recurrence. They concluded that peritoneal injury at trocar sites enhances implantation of carcinoma cells and suggest that repair of injured peritoneum at these sites may reduce the frequency of port site recurrence.

These clinical and experimental data provide clues to understanding the development of port site recurrence. Seeding could have occurred 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 case does not answer the question of how port site metastasis occurs, our patient provides clinical evidence to support the direct implantation theory for recurrence. We have also practised the use of an endo-catch bag to decrease the chance of direct implantation through the extraction site. Tumoricidal agents like povidone-iodine and silver sulphadine have been shown in experimental models to reduce the chance of port site recurrence[24,25] and should be considered. Wide excision of the abdominal wall recurrence with close follow-up remain the mainstay of treatment and will be required in all patients.

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