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# A CASE OF LEFT SEROUS CYSTADENOMACARCINOMA OF OVARY PRESENTING AS SPLENIC FLEXURE GROWTH CAUSING CLOSEDE LOOP OBSTRUCTION – A CASE REPORT

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**ABSTRACT** Ovarian cancer is the most lethal of all gynecologic malignancies and has witnessed minimal improvements in patient outcomes in the past three decades. About 70% of ovarian cancer patients present with disseminated disease at the time of diagnosis. The standard of care remains a combination of debulking surgery and platinum- and taxanes-based cytotoxic chemotherapy. Even though metastasis is the leading cause of ovarian cancer related fatalities, our understanding of the process remains limited. Ovarian cancer has a unique pattern of metastasis where the hematogenous spread is less common. Ovarian cancer cells mainly metastasize within the peritoneal cavity, which involves exfoliation from the primary tumor, survival, and transport in the peritoneal fluid followed by metastatic colonization of the organs within the peritoneal cavity. A key step for successful metastasis is their attachment and productive interactions with the mesothelial cells covering the metastatic organs for the establishment of metastatic tumors.

**KEYWORDS :** ovarian cancer, bowel obstruction,metastasis,microenvironment

## INTRODUCTION

Serous cystadenocarcinoma is most common ovarian malignancy ,usually occuring in 5th decade with most common metastasis to liver followed by intestine. In this case patient presented to casualty with closed loop obstruction due to a growth in the splenic flexure with intact ileocacal valve. Later was planned for emergency laparotomy and subtotal colectomy with ileostomy was done. Only palliative procedure could be done because of extensive metastasis to peritoneum and omentum with deposits over left ovaries. The detail case report shows the unusual presentation of closed loop obstruction with dilated large bowel loops.

## CASE PRESENTATION

A 70 year old female was brought to the casualty with complaints of vomiting, constipation for 1 month and obstipation for 10 days. On examination, abdomen distended with guarding and absent bowel sounds. She was taken to private hospital and was advised admission and surgery, CECT was taken and it suggested a growth in splenic flexure causing obstruction. She was not willing for surgery and was taken to home and now presented after 10 days. She was planned for emergency laparotomy and intraoperative findings showed large bowel loops proximal to splenic flexure dilated upto the cecum, with collapsed descending colon (Fig1). Growth adherent to lateral pelvic wall and spleen in the splenic flexure. Deposits all over omentum, peritoneum, left ovary (Fig.2). Planned for subtotal colectomy with terminal ileum brought out as ostomy, multiple peritoneal ovarian mesentery biopsies were taken. Palliative resection to relieve obstruction was done. HPE of the case suggested metastatic carcinomatous deposits in the omentum, mesentery, splenic flexure from high grade left cyst adenocarcinoma of ovary (Fig3, 4, 5&6)

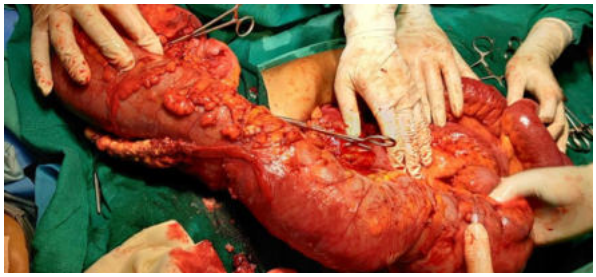


*Fig1: intraoperative picture showing dilated large bowel loops*

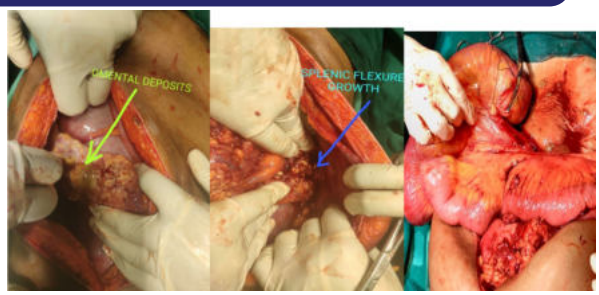



*Fig 2: Intraoperative pictures showing Omental deposits, splenic flexure growth, and dilated bowel segment*

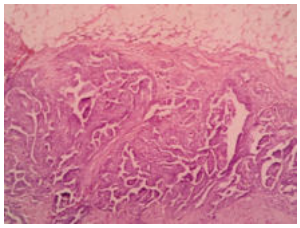


*Fig 3: Histology showing the tumour*

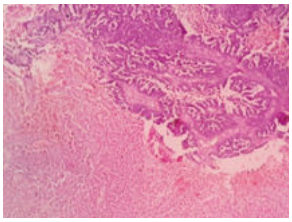


*Fig 4: Histology showing tumour with areas of necrosis*

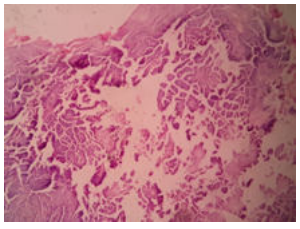


*Fig 5: Micropapillary pattern and Calcification*

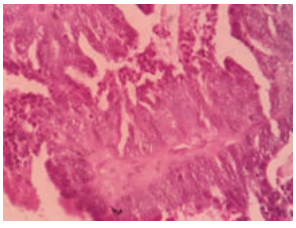
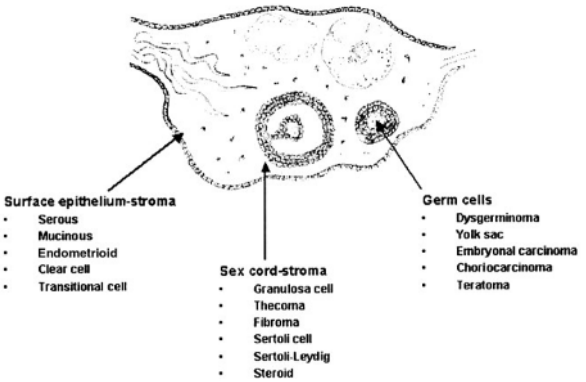


*Fig 6: Papillary pattern of malignant cells*

## DISCUSSION

Ovarian cancer is the most lethal of all gynecologic malignancies About 90% of all ovarian cancers are epithelial in origin, which are classified into high-grade serous, low-grade serous, endometrioid, clear cell, and mucinous subtypes [1]. Of these, the high-grade serous ovarian cancer (HGSOC) is the most common subtype and is characterized by mutations in p53 and genomic instability [2]. In the past, these tumors were thought to originate from the ovarian surface epithelium. However, more recently researchers have started to believe that they may actually originate from the fallopian tube fimbriae based on the analysis of prophylactic

salpingo-oophorectomy samples [3].




One of the reasons for the poor prognosis of ovarian cancer is the fact that most patients are diagnosed late [4]. It is a highly metastatic cancer and more than 70% of ovarian cancer patients are diagnosed with metastasis [5]. As the tumor grows within the peritoneal cavity, the symptoms produced are abdominal pain or bloating and may be confused with other bowel diseases like irritable bowel syndrome [6,7]. Ovarian cancer is often called 'the silent cancer' or 'the disease that whispers' because of these diffuse symptoms. The presence of high levels of cancer antigen 125 (CA-125) is used as a diagnostic marker of disease progression. Pelvic ultrasound, MRI, and CT scanning is also used to determine the extent of the disease. Patients undergo a 'debulking' surgery usually conducted by a gynecologic oncologist with a goal to remove as much of the tumor masses as possible from the abdomen [8]. The surgery is followed by adjuvant cytotoxic chemotherapy consisting of a combination of carboplatin and paclitaxel. The response to therapy is determined by measuring the serum CA-125 levels and by imaging techniques [3]. If the disease relapses within 6 months, it is considered chemoresistant and if relapse occurs after 12 months, it is considered chemosensitive. While a majority of the patients respond well initially to chemotherapy, most eventually end up developing chemoresistance [9]. Bowel obstruction by the metastatic tumors is the predominant cause of ovarian cancer-related mortality [10]. Since many parts of the bowel get affected, it becomes extremely difficult to surgically treat this condition. In addition, extensive ascites is a cause for major discomfort. Palliative measures such as control of nausea, abdominal pain, draining ascites, and modified diet are typically resorted to [3]. Since most of the ovarian cancer patients are diagnosed with advanced disease, in effect, it is metastasis that is being treated [6]. Therefore, a greater understanding of the process and regulation of ovarian cancer metastasis is essential.

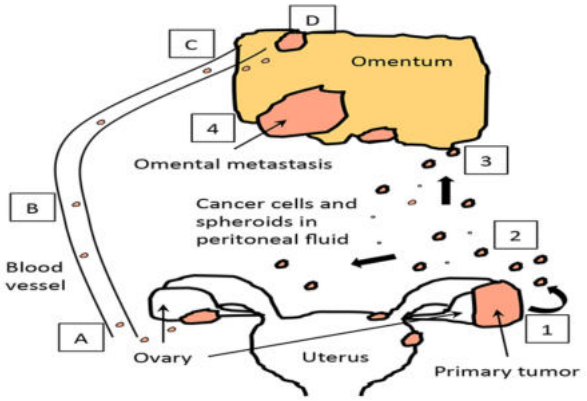
The biology of ovarian carcinoma differs from that of hematogenously metastasizing tumors because ovarian cancer cells primarily disseminate within the peritoneal cavity and are only superficially invasive. However, since the rapidly proliferating tumors compress visceral organs and are only temporarily chemosensitive, ovarian carcinoma is a deadly disease, with a cure rate of only 30%. There are a number of genetic and epigenetic changes that lead to ovarian carcinoma cell transformation. Ovarian carcinoma could originate from any of three potential sites: the surfaces of the ovary, the fallopian tube, or the mesothelium-lined peritoneal cavity. Ovarian cacinoma tumorigenesis then either progresses along a stepwise mutation process from a slow growing borderline tumor to a well-differentiated carcinoma (type I) or involves a genetically unstable high-grade serous carcinoma that metastasizes rapidly (type II). During initial tumorigenesis, ovarian carcinoma cells undergo an epithelial-to-mesenchymal transition, which involves a change in cadherin and integrin expression and up-regulation of proteolytic pathways. Carried by the peritoneal fluid, cancer cell spheroids overcome anoikis and attach preferentially on the abdominal peritoneum or omentum, where the cancer cells revert to their epithelial phenotype. The initial steps of metastasis are regulated by a controlled interaction of adhesion receptors and proteases, and late metastasis is characterized by the oncogene-driven fast growth of tumor nodules on mesothelium covered surfaces, causing ascites, bowel obstruction, and tumor cachexia.

The lack of an anatomic barrier allows the ovarian cancer cells to very conveniently spread into the peritoneal cavity. The cancer cells on the surface of the primary tumors start loosing cell–cell contact and become loosely attached to each other. As a result of this, they become prone to exfoliation into the peritoneal cavity. Exfoliation is promoted by the mechanical forces like rubbing of neighboring peritoneal organs during respiratory movements and flow of the peritoneal fluids. The cancer cells may come off as single cells or as clumps. This is a passive mode of dissemination unlike the typical invasion followed by intravasation observed in tumors undergoing hematogenous metastasis [10]. The peritoneal fluid naturally flows within the peritoneal cavity upward, toward the head, and then back downward, toward the feet, as a result of the diaphragm movement during respiration and gravitational pull, respectively . The exfoliated ovarian cancer cells from the primary tumor are disseminated throughout the peritoneal cavity by this natural flow of the peritoneal fluid . Since normally there is only a small volume of the peritoneal fluid present, dissemination is predominantly limited to the organs in the vicinity of the primary tumor . As the disease progresses, more and more ascites is produced and this enables the spread of the cancer cells to more distant sites in the abdomen. One of the predominant sites of ovarian cancer metastasis is the omentum which is a fatty double fold of the peritoneal membrane, about 8 by 8 inches in size, covering the bowels [10]. Epithelial cells tend to undergo anoikis in the absence of attachment to a substratum. Therefore, the main challenge faced by the cancer cells floating in the peritoneal fluid is overcoming anoikis and surviving floatation. In addition, they have to avoid immune surveillance. The cancer cells either form aggregates or spheroids or exist as single cells . The spheroids may also contain embedded cancer-associated fibroblasts as well as activated mesothelial cells, which contribute to the development of the ascetic microenvironment . The subsequent challenge for these floating cancer cells is to successfully attach to the surface of the organs in the peritoneal cavity. Debulking surgery often reveals such spheroids loosely attached to the peritoneum. The mesothelial cells covering the peritoneum and the bowels secrete mucus like substances, which help in reducing friction between surfaces as they brush against each other during the course of the organs' natural movements. The same also helps in preventing attachment of the cancer cells to some extent. However, the integrins expressed by the metastasizing cells help them to attach to the extra cellular matrix proteins (ECMs) secreted by the mesothelial cells. Thereafter, the cancer cells are able to push apart the mesothelial cells forming the protective barrier and invade into the organ.

The process of attaching to and developing metastatic tumors in the new organ is known as metastatic colonization . It is considered the least efficient step in the whole process of metastasis.

Mechanisms of ovarian cancer metastasis: Transcoelomic dissemination. (1) The cancer cells loose cell–cell contact and exfoliate into the peritoneal cavity. (2) They float in the peritoneal fluid and are carried all over the peritoneal cavity. (3) Attachment to the peritoneal organs like the omentum. (4) Formation of the metastatic tumor. Hematogenous metastasis.

(A) Invasion and intravasation. (B) Transport of circulating cancer cells through the blood vessels. (C) Extravasation from the omental capillaries. (D) Formation of the metastatic tumor in the omentum.




## CONCLUSION

Unusual presentation of ovarian carcinoma as large bowel obstruction is rare and has poor outcome .Most being terminal stage needing palliative resection and palliative care.

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