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Case Report

Mixed germ cell tumour complicated by pulmonary thromboembolism: a case report

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ABSTRACT

Malignant ovarian germ cell tumours (MOGCTs) are a heterogeneous group of tumours that have several histological different types derived from primordial germ cells of the embryonic gonad. They account for less than 5% of all ovarian malignancies, and are seen in the second and third decade of life. The majority of germ cell tumours are diagnosed in the early stages. Histology, FIGO stage and residual tumour after surgery are the most important prognostic factors. Recent multimodality therapy with staging laparotomy and conservative surgery, followed by platinum based chemotherapy, is associated with survival rates of 60 - 80%, even in patients with advanced disease. Mixed germ cell tumours are extremely rare and the prognosis depends on the size of each component of the tumour. The exact incidence of thrombo embolic events in patients with malignancy is difficult to determine. However ovarian malignancy is strongly associated with venous thrombo embolism. We report a case of a fourteen year old girl with a mixed GCT, with elements of yolk sac tumour and embryonal carcinoma, who succumbed to pulmonary thromboembolism.

Keywords: MOGCT, Pulmonary thromboembolism

INTRODUCTION

Ovarian germ cell tumours are a challenge to the Gynaecologist. The majority of malignant ovarian germ cell tumours are diagnosed early, and complete remission is usually achieved, with future fertility rates up to 70%.[1] However, the most malignant tumours often progress rapidly, and are associated with high mortality rates. Histology of the tumour is often the most important factor determining survival.[1]

CASE REPORT

Ms. X, a fourteen year old girl presented to the Gynaecology OPD with pain abdomen and abdominal distension for one month. She also had progressive difficulty in breathing, vomiting and loss of appetite for two weeks. She had attained menarche two months back, and was having regular cycles. There was no family history of breast, uterine, gastro intestinal or ovarian malignancies.

On examination she was of average built, with a height of 158 cm, weight of 57 kg and a BMI of 22.8 kg/m2. Her vital signs were stable, and she appeared pale and mildly dehydrated. There was no icterus, clubbing, cyanosis pedal oedema or palpable lymphadenopathy. The breast and thyroid examination was normal. On inspection the abdomen was distended from the pubic symphysis to the xiphisternum. There was no local rise of temperature. Diffuse tenderness was present all over the abdomen. The lower border of the mass could not be made out. On per rectal examination, the rectal mucosa was free.

Ultrasonography showed a heterogeneous solid cystic intra peritoneal mass extending from the pelvis to the epigastric region. Moderate to severe ascites was present.

The uterus was normal size, and ovaries were not separately visualised.

CECT of the abdomen and pelvis showed a pelvic mass of 20 x 22 x 12 cm displacing the small bowel cranially, and the rectum and sigmoid colon posteriorly. Omental stranding was seen. There was moderate ascites with minimal right pleural effusion. No liver metastases or significant lymphadenopathy was noted.

Serum B Hcg was 6270 IU, LDH 3383 IU, αFP was 1000 IU. Ca 125 was 388 IU. A provisional diagnosis of malignant ovarian germ cell tumour was made, and the patient was posted for a staging laparotomy after anaemia correction with two pints of packed cell transfusion.

Under general anaesthesia, the abdomen was opened by a midline vertical incision extending from the pubic symphysis to the xiphisternum. Approximately 1.5 litres of haemorrhagic ascitic fluid was drained and sent for cytology analysis. A solid cystic mass of 20 x 25 cm arising from the left ovary was removed along with the left fallopian tube, which was stretched over it. The uterus, right tube and ovary were inspected and found to be grossly normal. The abdomen was explored. There were no gross peritoneal deposits. The liver, under surface of the diaphgram, stomach, omentum, small and large bowel appeared grossly normal. Infra colic omentectomy was done.

Ascitc fluid cytology showed predominant neutrophils and mesothelial cells in a haemorrhagic background. Histopathology showed a malignant mixed germ cell tumour (yolk sac tumour with embryonal carcinoma) with 18 – 20 mitotic figures per high power field. The fallopian tube and omentum were free of tumour. FIGO staging was done and the tumour was allotted stage IA Grade 2.

The patient was planned for post-operative chemotherapy with Bleomycin, Etoposide and Cisplatin. Prior to chemotherapy her chest x-ray, blood counts, renal and liver functions were normal. B Hcg fell to 41.6 and α FP was 794.3. On day 6 of the first cycle she received G – CSF 6 mg subcutaneously.

However on day 8 she developed fever with vomiting and loose stools. She developed pancytopenia with a sudden fall in haemoglobin to 5.6 g%, TLC of 590 cells/cu mm and platelets dropping to 12,000 cells/cu mm. The patient developed ecchymotic patches all over. She also complained of sudden pain in her calf muscles. D Dimer was found to be positive. An emergency transfusion of four pints of plasma rich platelets was arranged. Even before a venous Doppler of the lower limbs could be done to confirm the diagnosis of Deep vein thrombosis, the patient had sudden onset of breathlessness, and her SpO2 dropped to 82%. As resuscitative measures were started, she had a massive cardio respiratory arrest and could not be revived. A diagnosis of massive pulmonary embolism secondary to deep vein thrombosis leading to death was made.

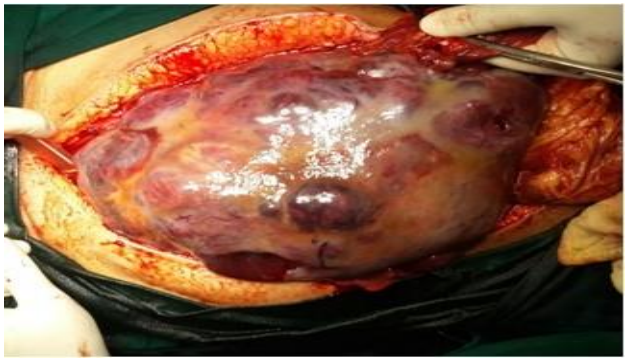

Figure 1: Staging laparotomy.

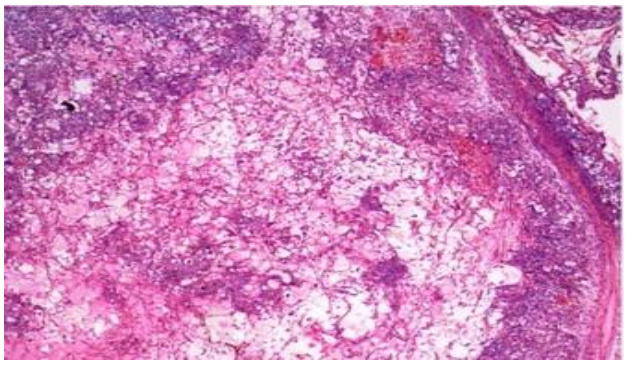

Figure 2: Embryonal cell component.

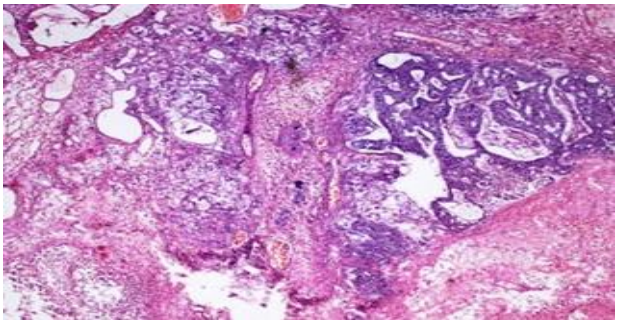

Figure 3: Yolk sac component.

DISCUSSION

The principles of cytoreductive surgery in ovarian cancer must be applied to the management of OGCTs. But, for a young girl desiring a pregnancy, the surgical treatment must be conservative, with an attempt to preserve fertility. Weinberg et al described fertility rates of up to 80%, following post-operative chemotherapy.[1]

For both dysgerminomas and non dysgerminomatous germ cell tumours, after a surgical staging and treatment, the first line chemotherapy is BEP (cisplatin, etoposide, bleomycin). The choice of post-operative chemotherapy

is determined by the surgical stage and the histologic type of malignancy.

Mixed germ cell malignancies of the ovary contain two or more germ cell elements. The most common component of a mixed malignancy is dysgerminoma in 80%, followed by EST in 70%, immature teratoma in 53%, choriocarcinoma in 20%, and embryonal carcinoma in 16%. The mixed lesions may secrete AFP, hCG, or both or neither of these markers.

These lesions should be managed with combination chemotherapy, preferably BEP. The serum marker, if positive initially, may become negative during chemotherapy, but this finding may reflect regression of only a particular component of the mixed lesion. Therefore, for these patients, a second-look laparotomy may be indicated to determine the precise response to therapy if macroscopic disease was present at initiation of chemotherapy.

The most important prognostic features are the size of the primary tumour and the relative size of its most malignant component. For stage Ia lesions smaller than 10 cm, survival is 100%. Tumours composed of less than one third EST, choriocarcinoma, or grade III immature teratoma also have an excellent prognosis, but it is less favourable when these components constitute most of the mixed lesions.

The incidence of venous thrombosis for cancer patients is increased compared with patients without cancer, but estimations of the incidence for different types of cancer have rarely been made because of the low incidence of various types of cancer. Patients with tumours of the bone, ovary, brain and pancreas had the highest cumulative incidence of DVT of the leg or arm or PE in the first six months after diagnosis of cancer.[2] The risk of a VT within 6 months after this first thrombotic event was 4.6-fold increase compared to cancer patients who did not have a thrombotic event in the 6 months after cancer diagnosis. Patients with distant metastases and those undergoing chemotherapy had a 2-fold increased risk compared with those without metastases or not using chemotherapy.[2]

Multiple biomarkers have been linked to cancer-associated thrombosis. The highest level of evidence currently exists for components of the complete blood count: prechemotherapy elevated platelet counts, elevated leukocyte counts, and low haemoglobin levels are all associated with chemotherapy-associated VTE.[3] Elevated D-dimer was also associated with 1.8 fold increased risk of VTE in the Vienna Cancer and Thrombosis Study (CATS) registry.[4] Thromboprophylaxis is currently recommended for cancer inpatients without contraindications by the American Society of Clinical Oncology (ASCO) and National Comprehensive Cancer Network (NCCN) guidelines. The Randomized Comparison of Low Molecular Weight Heparin versus Oral Anticoagulant Therapy for Long-Term Anticoagulation in Cancer Patients with Venous Thromboembolism (CLOT) study, showed that low molecular weight heparin is the best anticoagulant. On the basis of long-term follow-up data on patients with thrombosis, those with cancer have a 4- to 8-fold higher risk of dying after an acute thrombotic event than patients without cancer. This high mortality probably reflects deaths due to both thromboembolism and a more aggressive course of malignancies associated with VTE. Further epidemiological research will provide more reliable estimates of the thrombotic risk associated with different types of tumours, stages of disease, and antitumour treatments.

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