Her chest was clinically clear. The abdomen was flat, with no organomegaly. Vaginal examination showed a firm uterus irregularly enlarged to the size of a ten week pregnancy. A clinical diagnosis of multiple uterine fibroids with chronic pelvic inflammatory disease was made. Investigations included PCV 29, WBC 2,700/dl, with normal differential count, platelets 206x10^5, ESR 120mm/hr, Urinalysis was normal and no pathogens were grown on culture. The retro viral test was negative. Plain abdominal X-ray and intravenous pyelogram were normal. An initial ultrasound scan was reported thus:

1. Intestine: Thickened loop of intestine 25mm thickness and 61mm in diameter is seen below the liver and adjacent to the right kidney.
2. Liver, gallbladder, pancreas, kidney and spleen normal.
3. Bulky anteverted uterus 132 x52 x38mm in longitudinal, transverse and antero posterior diameters with a subserous fundal fibroid 48mm in diameter and normal endometrium.
4. Bilateral solid ovarian masses, right 110x90mm and Left 125x95mm.
5. No fluid collection

A repeat scan on the 4th of May reported only huge multiple interstitial fibroids.

The patient was booked for exploratory laparotomy on the 16th of May and the findings were as follows:

1. 2 litres of ascitic fluid, diffuse peritoneal seedlings and omental metastases seen. There was a tubular mass 100mm in length involving the hepatic flexure of the colon. There were bilateral ovarian tumours, right 120x110mm and left 90x75mm. There was also a fundal fibroid 5x80mm. A diagnosis of cancer of the colon with secondaries to the ovaries and peritoneum was made. A right hemicolectomy with ileo transverse anastomosis, left partial ovariotomy , right ovariotomy , and myomectomy were done. The blood loss was estimated at 250mls. The abdomen was closed in layers and a drain was inserted.
All specimens were sent for histology and reported thus:

**GROSS.** Right Ovary 9.5x9x3.5cm. Greyish white firm and nodular. Cut surface shows a greyish white brownish and dark surface with variable size cystic areas. Focal necrosis noted.

Left Ovary 8.5x6.5x3cm. same as the right ovary but for size.

**Intestine:** 43 cm, with proximal end about 17cm from the ileo caecal junction. The mesentry and entire segment including the appendix are matted with variable size enlarged greyish white nodules. A huge firm nodular greyish white colonic mass (ascending colon) is seen about 12cm from the ileo caecal junction. Its cut surface is greyish white.

**MICROSCOPY:** Fig 1

Sections of the intestinal tissue show a non-encapsulated mass invading the muscular layer. The mass in most areas is composed of variable sized ragged glands invading the muscle layer. Also seen are foci of infiltrating sheet and nest of loosely cohesive malignant epithelial cells. Some of the glands are swimming in a lake of mucin. This same tumour is seen invading the mesentery, venniform appendix, ileum and adjacent caecum. The proximal and distal resection margins are spared. Both ovaries are seen to have been diffusely invaded by these malignant glands.

A histological diagnosis of Mucinous adenocarcinoma of the colon with bilateral Krukenberg’s tumours of the ovaries was made.

The post operative period was very stormy and was complicated by severe hypertension BP 200/110mm of mercury and pulmonary oedema which were successfully controlled with hydralazine, intravenous frusemide, aminiophyline and oxygen. The patient thereafter slowly recovered and was discharged on the 12th post-operative day. She was worked up for chemotherapy which was started on 15/6/07 with Leucovorin and 5-fluorouracil. She developed malignant intestinal obstruction secondary to diffuse peritoneal metastases and bilateral pleural effusion, gradually went downhill and died on 6/8/07.

**DISCUSSION**

Malignant ovarian tumours of the ovary may be primary or secondary. Secondary tumours are rare and when bilateral and mucinous are named Krukenberg tumours after Friedrich Krukenberg who described them in 1896. They constitute about 40% of all ovarian malignancies. Secondaries to the ovaries and originate from gastric carcinoma in 70-80% of cases. Sites of primaries are the colon, kidney or the gall bladder. Their incidence varies with that of gastric cancer in the environment and is said to be about 2% of all ovarian malignancies.

The age of presentation of this patient is close to the mean age of presentation of 43.3 years and is also consistent with the fact that most (71.5%) cases present before menopause. The prognosis of these patients is that of the primary tumour most patients dying within a year of diagnosis as was the case here.

This case serves to alert practitioners to the fact that all nodular masses in the pelvis are not necessarily fibroids and all pains associated with menstruation are not always the result of pelvic inflammatory disease. Such symptoms may indeed as illustrated here be the result of more serious problems. Although this patient had fibroids it was relatively small and insignificant in comparison to the very large ovarian secondaries and was not responsible for the patient’s symptoms. It is possible that the premature labour the patient had was precipitated by the tumour while the blame was inadvertently heaped on the fibroids. This case also suggests that when premature labour occurs careful investigations should be done to try to elicit the cause of the premature labour. Had this been done the lesion may have been discovered when it was still amenable to treatment.

Also demonstrated in this case is the fact that ultrasound is highly observer dependent. Both scans failed to show the presence of ascitis while the second scan completely missed the colonic mass. This possibility of an error should always be borne in mind in the evaluation of imaging results of patients.

**REFERENCES**


Krukenberg Tumour Ezem et al