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NEWS LETTER

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EDITORIAL

Dear Colleague,

It gives me immense pleasure to welcome each one of you to the 23rd Annual meeting of Trivandrum OBGYN club from May 12-14th 2023. After two years of Covid attack we are getting back to normalcy and newer strains are causing threat in other parts of world.

This time we have moved into Hyatt Regency which was our base for more than 10 years as Vivanta by Taj. Year after year it has become my passion to hold the meeting with most recent progress in Foetal medicine, Obstetrics, Gynaecology and Reproductive Medicine with top of International speakers National speakers and State faculty.

Dr Justin Clark from Birmingham UK will be the overseas speaker along with Dr Alex Varghese from News Zealand who will be connecting us live.

Dr Mridhubashini from Kovai will deliver the Advances 2023 Oration and Dr Sonia Malik from New Delhi will deliver the Dr Thomas Chandy Memorial oration.

Apart for the two orations we will have eight Keynote address and more than 8 panel discussions.

The free paper presentations for delegates and Dr VI Nalini Memorial presentations will continue along with Dr Meera Nair Memorial Video Quiz.

I am sure that Advances 2023 will be different this time sharing a plethora of knowledge in Obgyn and Reproductive Medicine along with evenings to relax, refresh and renovate.

Do come and enjoy this meeting with a difference in Gods own country.

Thank you all who have helped me in this journey and inviting you to this academic extravaganza.

Dr K Jayakrishnan
Organising Chairperson
Advances 2023

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Always expected the unexpected

A endometrioid adenocarcinoma of the ovary was an incidental finding in a postmenopausal women

Dr SHARDA SHARMA MBBS, DNB
Fellow in Reproductive Medicine

INTRODUCTION

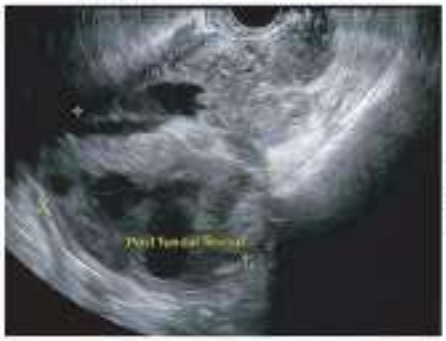
Ovarian cancer is oneof the most common gynaecologic malignancy, with over 90% of cancers arising from epithelial cells. Endometrioid carcinomas account for 8-15% of all ovarian carcinomas. They are considered the second most common malignant ovarian neoplasm. More than 70% of Endometrioid Ovarian Carcinoma are diagnosed at Federation International of Gynecology and Obstetrics (FIGO) I-II according to Ovarian Cancer statistics in the United States in 2018. Mean patient age is 55 years at the time of presentation. Usually unilateral; only 5% bilateral. Associated with endometriosis/endometriotic cyst (15%), endometrioidadenofibroma, synchronous endometrial endometrioid adenocarcinoma or endometrial hyperplasia. Risk factors: endometriosis, hormone replacement therapy, first degree family history of breast carcinoma. May occur in the setting of Lynch syndrome.

CASE REPORT

A 52 year P1L1 postmenopausal female with complaint of postmenopausal bleeding 1 month, associated with dysmenorrhoea, diagnosed with multiple fibroids with degeneration. BMI 32 kg/m2. There was no other altered bowel and bladder habbits. No history of nausea, vomiting, anorexia or weight loss. Per abdomen a mass of 20-22 weeks size was palpable. Per speculum examination cervix appeared healthy, smear was taken. On bimanual examination uterus was found to be enlarged 22-24 weeks size.

USG

Ultrasonography was done which showed markedly enlarge uterus with multiple fibroids with posterior SSF close to cervix 4.5 x 4.6 cm, posterior SSF 5 x 4.6 cm, Posterior fundal SSF 10.9 x 11.2 cm with cystic degeneration. Right ovary not visualised and left ovary appears to be normal. Endometrial thickness 7 mm.



Biochemical marker:
CA-125 106 U/ml
LDH raised 361 IU/L

The patient was then taken for hysterectomy with bilateral salpingo-oophorectomy. Peritoneal washings were also taken.

An endometrioid tumor of the left ovary was an incidental finding, which is a very rare occurrence.

GROSS (Macroscopy)

Total hysterectomy specimen with right tubes/ovary and partly collapsed left tubo-ovarian soft tissue. Uterus with cervix measures 13 x 8 x 7 cm. Section show distorted and minimally dilated endometrial cavity containing an endometrial polyp measuring 2 x 1 cm. Endometrium otherwise with in normal limits. Myometrium shows an irregular thickening (maximum thickness of 5 mm) seen towards the attached soft tissue of left tubo-ovarian side. Cervix unremarkable. Right tube 6 cm in length section normal, that ovary measure 3 x 2 x 2 cm section normal. Another fibroid received 5 cm in diameter. Section whitish whorled appearance. Left tubo-ovarian area (possibly capsular part of shelved out lesion) show brownish edematous soft tissue whole measuring 11 x 8 cm. Another irregular oval grey brown soft tissue lesion received weighing 325 gm and measuring 14 x 13 x 8 cm.Surface greyish white fleshy and bosselated section show a partly solid and partly cystic lesion with peripheral greyish white fleshy cut structure and central attempted central cystic changes.

MICROSCOPY

Section of the bits from the lesion shows an invasive neoplasm composed of sertoliform tubules/ complex glands/solid islands of oval or round cells having varying faintly eosinophilic cytoplasm and pleomorphic vesicular nuclei. Also seen cell islands with marked nuclear atypia, attempted differentiation and focal necrosis. This lesion is seen infiltrating the soft tissue of the left tube, tubo ovarian area and adjacent myometrium. Uterus otherwise shows simple cystic hyperplasia of endometrium with polyp, leiomyoma and cervix with moderate chronic cervicitis. Right fallopian tube and ovary are with in normal limits.

Smear from peritoneal fluid- showed no atypical cells.

DIAGNOSIS

Moderately differentiated (FIGO- Grade 2) Endometrioid adenocarcinoma - (Predominant sertoliform type) involving left ovary, left tubo-ovarian soft tissue and adjacent myomertium (FIGO-Stage 2A).

She was referred to regional cancer center for further management.

TREATMENT AND PROGNOSIS

Endometrioid histology may carry a slightly better prognosis than a serous or mucinous cystadenocarcinoma of the ovary independent of stage. Pure endometrioid tumors carry a far better outcome than a mixed variety.observation is encouraged for stage IA/IB patients, similar to Low Grade Serous Ovarian Carcinoma; for patients with stage IC, observation or intravenous platinum-based therapy seems preferable; additionally, for women with high ER/PR expression of tumor cells, options to use hormonal therapy (tamoxifen, aromatase inhibitors); and patients with stage II-IV could be considered to accept systemic adjuvant chemotherapy following surgery. However, the treatment recommendations for grade 2/3 Endometrioid Ovarian Carcinoma are the same as High Grade Serous Ovarian Carcinoma, employing paclitaxel and platinum-based chemotherapy after surgical resection as the mainstay of primary treatment.

Although high-grade Endometrioid Ovarian Carcinoma and High Grade Serous Ovarian Carcinoma have equally high response rates to platinum-based chemotherapy, high-grade Endometrioid

Ovarian Carcinoma seems to develop chemoresistance easily at recurrence, indicating the significance for novel therapeutics in this subtype.The prognosis of Endometrioid Ovarian Carcinoma is satisfactory compared with other histological subtypes, there are still big differences present when Endometrioid Ovarian Carcinoma is further classified molecularly. In the analysis of prognosis, the TP53abn group has a poor prognosis, with a 10- year disease-specific survival (DSS) of lower than 40%; whereas the POLEmut group has the best prognosis among the four molecular subtypes.

Generally, epithelial ovarian cancer grows rapidly and presents as advanced disease at the time of diagnosis because patients do not experience symptoms in the early stages. It exhibits various genetic mutations, and a subdivision in two types has been introduced based on genetic and pathohistological characteristics: type 1, which comprises serous low grade, endometrioid, mucinous and clear cell epithelial tumors of the ovary, and type 2, which comprises high grade serous, high grade endometrioid, malignant combined mesodermic and undifferentiated ovarian tumor. The serous pathohistological type is characterized by a mutation on the TP53 gene, in clear cell mutation it occurs on ARID1, PIK3Ca, which is also characteristic of the endometrioid carcinoma, although they also present a mutation of the CTTNB1 gene. On the other hand, in mucinous ovarian carcinoma, the KRAS gene mutation is dominant. It is believed that tumors belonging to type 1 originate from the same forms of benign or borderline lesions or appear in the field of endometriosis, such as the endometrioid and clear cell histological type of the tumor. Those belonging to type 2 are more aggressive, and it is believed that they most likely originate from the oviduct fimbriae and that their manifestation on the ovaries is primary tumor metastases. If diagnosed at an early stage, they usually belong to type 1 as opposed to type 2, which is detected at a late advanced stage.

OVARIAN CYSTADENOFIBROMA - A TUMOR IN DISGUISE

Dr MEENAKSHI A. MBBS, DNB
JUNIOR CONSULTANTS IN OBSTETRICS & GYNAECOLOGY

CASE REPORT

Miss. X, 23 years old unmarried woman, came with complaints of heavy menstrual bleeding. She had attained menarche at 13 years of age. She had regular cycles once in 28 - 30 days with flow lasting for 4 -5 days without dysmenorrhea. Her LMP was on 04/11/2022. Bleeding lasted for >24days, associated with passage of clots which required changing 3 pads/day. She had no significant medical or surgical history.

Transabdominal ultrasound showed normal uterus and right ovary. Left adnexa showed a 7.5 x 7.4 cm cyst with internal echoes and hyper echoic areas with papillary projections and normal vascularity. MRI of pelvis was done which showed a well defined cystic space occupying lesion in the midline of the pelvis extending from supravesical region to pelvic inlet with mildly high density fluid contents inside. T2W images showed hypo intense signals. There were no septations, haemorrhage or calcifications. Both ovaries were visualised separately and appeared normal. There was no ascites, no omental/mesenteric lesions, no lymphadenopathy. Findings favoured a cystic neoplasm of most likely paraovarian origin with O-RADS 4. Tumor markers were normal. Routine pre-operative blood investigations were within normal limits. Tumor markers were normal. Proceeded with laparoscopy. Intra operatively she was found to have a left paraovarian cyst of size 8 x 8 cm arising medial to left ovary. Paraovarian cystectomy was done without spillage of contents. Histopathology showed benign serous cystadenofibroma of ovary.

DISCUSSION

Ovarian cystadenofibroma is a rare slow growing epithelial tumor that contains both epithelial and fibrous stromal components. It accounts for 1.7% of all benign ovarian tumors. The causal factors are unknown. Lesions can be bilateral in 15% of cases. Recognized histological subtypes includes (i) serous cystadenofibroma of the ovary (ii) mucinous cystadenofibroma of the ovary (iii) endometrioid cystadenofibroma of the ovary (iv) clear cell cystadenofibroma of the ovary (v) mixed cystadenofibroma of the ovary.

Ovarian cystadenofibroma is seen in women aged 15 - 65 years. Serous cystadenofibroma of the ovary usually presents with signs and symptoms such as abdominal pain, vaginal bleeding and mass in abdomen. Many such tumors are usually detected incidentally during an abdominal ultrasound. The complications due to these ovarian tumors are rare, but may include rupture of cystic portion of the tumor within the abdomen, ovarian torsion of the affected ovary.

DIAGNOSIS

Almost all cystadenofibromas are predominantly cystic on ultrasound with septations seen in 30% of cases. Papillary projections or solid nodules have been sonographically seen in just over 50% of cases. Vascularization can be present in just under 50% of cases with a typical pattern of peripheral vascularization with scattered vessels of high blood flow impedence. The appearance of cystadenofibroma on imaging is often complex; cystic to solid mass may be visualized and it often resembles a malignant tumor.

Owing to the fibrous component of this tumor, MRI scanning shows low signal intensity on T2W images and this may help a radiologist to make a pre-operative diagnosis of this tumor and thus perhaps avoid aggressive surgical management. Other tumors with similar T2 characteristics due to a fibrous component are fibroma, fibrothecoma and Brenner tumor, which are all benign tumors. Malignant ovarian tumors with a fibrous component and low T2 signal intensity are likely to be metastases from the gastrointestinal tract and struma ovarii.

MANAGEMENT

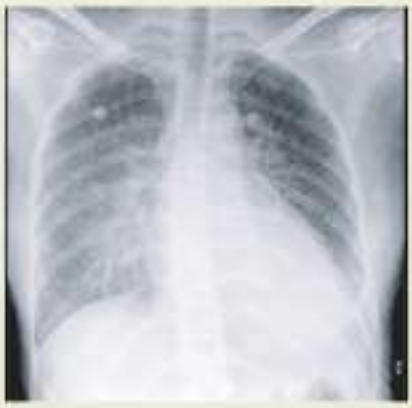
Ovarian cystadenofibroma generally tend to be benign tumors although the degree of epithelial proliferation and its relation to the stromal component of the tumor can be used for their classification as benign, borderline or malignant. The treatment of choice is complete surgical removal of the tumor. The prognosis is generally excellent with prompt and appropriate treatment. As these tumors resemble the gross appearance of malignant tumors perioperatively, when available, frozen sections should be performed to direct the surgeon and prevent the patient from unnecessary extensive surgery. They have a very low recurrence risk on complete removal through surgery.

PERIPARTUM CARDIOMYOPATHY: AN INTRIGUING CHALLENGE IN OBSTETRICS

Dr Silpa P. MBBS, DGO
Jr. Consultant Obs. Gyn.

CASE REPORT

40 year old G2A1, ICSI Conception, DCDA Twin, GDM on OHA at 33 week 6 days gestation came to our OPD with low grade fever, myalgia, cough with expectoration and headache. No complaints of running nose, sore throat, dysuria, diarrhea, or dyspnoea. Patient was conscious, oriented, Temperature - 99 degree F, PR-99/mt, BP-130/80 mm Hg, Respiratory rate - 16/mt, Per abdominal examination - fundal height of 34-36 week, relaxed, both fetal heart sounds good. She was admitted and fever evaluation was done. COVID RTPCR,Dengue NS1, H1N1 throat swab were negative. Blood investigation were normal. Physician consultation was done, managed conservatively as viral fever. But after few days patient developed thrombocytopenia upto 90000, but gradually picked up to 2 lakhs. No signs of ecchymosis or haemorrhage. patient got better, fever and cough subsided and platelet count became normal. Patient was taken up for elective LSCS under SA at 36 weeks of gestation. Fetus A-live preterm male baby of B.wt 1.81. kg fetus B-live preterm female baby of B. wt 1.29 kg. Patient had high Blood pressure postoperatively, managed with T Nicardia Retard 10 mg 1-0-1and T labetolol 200 mg 1-1-1 and blood pressure was normalised. Post operatively patient developed dyspnoea, and decreased oxygen saturation, PR-115/mt, BP-140/90 mm Hg, SPO2-96% with 10 L O2. Chest - fine basal crepitation present. CXR-shows pulmonary edema. Cardiology consultation done. Echo was taken - LV mildly dialated. Mid apical LV segment severly hypokinetic. Basal segments contracting well. LV ejection fraction 35-40%. No clot /PAH/Effusion. suggestive of stress cardiomyopathy, mild-moderate LV dysfunction. Mild MR. Patient was managed with IV Diuretics, antihypertensives. Patient was better, symptoms subsided, hence discharged on post op day 9. Patient was reviewed after 1 week. Repeat Echo was taken-LVEF 64%, good Biventricular function. Patient is now on antihypertensives and on follow up.



DISCUSSION:

Peripartum cardiomyopathy (PPCM) is a dilated cardiomyopathy defined as systolic cardiac heart failure in the last month of pregnancy or within five months of delivery. The definition of PPCM includes four criteria: 1) development of cardiac failure in the last month of pregnancy or within five months of delivery, 2) absence of an identifiable cause for the cardiac failure, 3) absence of recognizable heart disease before the last month of pregnancy, and 4) left ventricular (LV) dysfunction (ejection fraction of less than 45% or reduced shortening fraction).

The principal hypothesis with regards to the pathogenesis of this cardiomyopathy include an autoimmune response, an abnormal reaction to physiologic hormones or a viral aetiology as in our case scenario. Risk factors include multiparity, multiple pregnancy, black race, older maternal age, pre-eclampsia, and gestational hypertension.

CLINICAL FEATURES

PPCM usually presents with classical symptoms and sign of systolic heart failure with ventricular enlargement and dysfunction seen on echocardiography. Often there is significant mitral and tricuspid regurgitation. Unusual presentations include thromboembolism or hepatic failure secondary to heart failure. The development of heart failure and the usual time of diagnosis are during the post-partum period in more than 90% of the cases. PCM can occur at any age with a higher incidence in women older than 30 years.

The diagnosis of PCM is challenging because most women in the last months of a normal pregnancy or soon after the delivery experience dyspnoea, fatigue and pedal oedema. Symptoms and signs which should raise the suspicion of heart failure and could help the clinicians in the diagnosis, are the presence of paroxysmal nocturnal dyspnoea, nocturnal cough, new regurgitant murmurs, pulmonary crackles, jugular venous distention and hepatomegaly. The diagnosis is often delayed and the disorder is under recognized, with devastating consequences: Mortality is as high as 20% to 50%.

PATHOGENESIS

Gestational hypertension, tocolytic therapy and twin pregnancy have been proposed as possible risk factors because they were commonly associated with PPCM. The association between PCM and twin pregnancy could support the theory of autoimmunity as a possible mechanism. This could depend on an excessive traffic of haematopoietic lineage cells from the foetus to the mother as manifest in twin pregnancy. Usually the lower concentrations of these foreign proteins could contribute to tolerance of the foetus while increased levels could theoretically lead to the initiation of autoimmune disease. Following postpartum, the recovery of immune competence could trigger a pathologic autoimmune response against cardiac cells where haematopoietic cells have taken up residence during pregnancy and therefore myocardial cells are recognised as nonself.Multiparity could be another risk factor for the development of PCM.

PROGNOSIS

Overall prognosis of PCM is good in majority of the cases, although some patients may progress to irreversible heart failure. Progression of the condition requiring heart transplantation is described in 4% and death in 9% at a two

years follow up. Sudden cardiac death has been reported to account for up to 50% of the mortality.

Normalisation of left ventricular systolic function occurs in 23% and in 54% of the patients respectively at six months and at two years and is more likely if EF at diagnosis is more than 30%. Higher left ventricular end diastolic dimension and lower fractional shortening at diagnosis seems to be associated to a worse prognosis. A fractional shortening of less than 20% and a left ventricular end diastolic dimension of 6 cm or greater was associated with a more than 3-fold higher risk for persistent left ventricular dysfunction . 75% of the patients, who recover, have an EF of more than 45% at two months after diagnosis .Complete recovery of systolic function occurs usually in the first 6 months after delivery.

MANAGEMENT:

Management involves conventional therapy for heart failure with diuretics, ACE-inhibitors, beta-blockers and aldosterone antagonists. Angiotensin-receptor blockers should be added in case of ACE-inhibitors intolerance. Anticoagulant therapy should be considered in view of the low left ventricular EF, which predisposes to thrombus formation, especially in the peripartum period when a hypercoagulable state exists. In patients not improving on conventional therapy or in patients with critical hemodynamic state with cardiogenic shock, hemodynamic support with pressors should be considered.

Non-responsive patients should be considered for heart transplantation even if there are some reports about effective use of extracorporeal membrane oxygenation, intraaortic balloon pump or mechanical assist devices.

NEXT PREGNANCY??

A subsequent pregnancy carries a high risk of relapse, significant decrease of left ventricular function and mortality. Mortality rate is described to be approximately 55% during subsequent pregnancy even though it seems associated more with patients who entered the subsequent pregnancy with abnormal systolic function i.e. without making a complete recovery. Complete recovery from a relapse is very rare. There is no consensus regarding recommendations for future pregnancy after PCM but patients whose left ventricular size or function does not return to normal should be counselled strongly to avoid subsequent pregnancy .

CONCLUSION

PCM is a relatively rare disease, which can have devasting consequences and should be promptly identified and correctly treated. Early diagnosis is important and therefore women who develop symptoms of heart failure during pregnancy or shortly after should be investigated for this condition. Effective treatment reduces mortality rates and increases the chance of complete recovery of ventricular systolic function.

An effective "Diluted Vasopressin" assisted method for excision of ovarian endometrioma by Laparoscopy

Dr KOKILA B.T. MD (OBG)
Fellow in Reproductive Medicine

Endometriosis is a common disease with an incidence rate of 15% in women of childbearing age, and is a chronic disease that significantly affects women's quality of life by causing two problems: pain and infertility. The usual treatment for ovarian endometrioma is surgery, and the most common surgical method is laparoscopy.

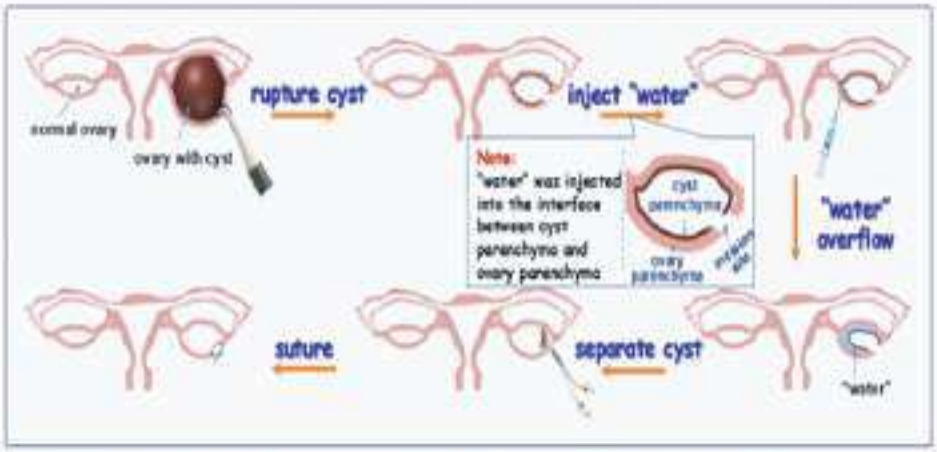
Surgery has the advantage of reducing the pain of ovarian endometrioma and lower recurrence rate compared to other treatment methods, but it also has the disadvantage of deteriorating ovarian function. This is because healthy ovarian tissue adjacent to the ovarian tumor are damaged at the time of surgery, and damage to the ovarian tissue while using an electric cauterizer for hemostasis when removing the ovarian tumor.Therefore, various surgical methods have been studied to minimize damage to the ovarian tissue during the surgical procedure including local vasopressin injection into the surgical site. Previous studies reported that this may minimize damage to the ovarian tissue during the surgical procedure.

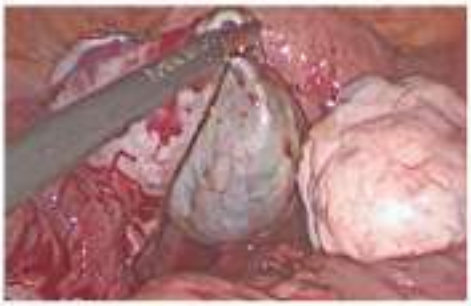
CASE REPORT

A 29 yr old female, married for 2 yrs, a known case of fibroid uterus and right ovarian endometriotic cyst with dysmenorrhoea and heavy menstrual bleeding, was referred to our hospital for laparoscopic myomectomy and right ovarian Cystectomy. Patient was not planning for pregnancy now. Her routine blood investigation were sent. Her USG revealed multiple small intramural fibroids (FIGO - 3), close to the endometrial cavity. Both ovaries were adherent to the uterus. Left ovary showed 2 endometrioticcyst of size 5.9 x 5.5 cm and 1.6 x 1.9 cm. Adequate AFC.Her preprocedure AMH was 2.4 ng/ml .Proceeded with the Operative laparoscopy.

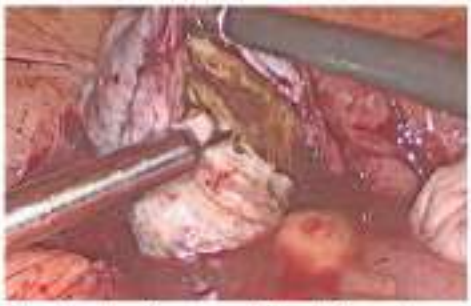
Intraoperatively, her Uterus was bulky with multiple subserous and intramural fibroids, with largest intramural fibroid measuring 3 x 2 cm. Left ovary had a large endometriotic cyst of size 6x 6 cm and was adherent behind the uterosacrals and uterus, flight ovary polycystic. POD completely obliterated. POD had bowel adhesions seen up to the fundus. Endometriotic deposits present on the posterior surface of uterus, uterosacrals and surface of right ovary. It was Severe Endometriosis with AFS 44 and EFI 7. Proceeded with Left ovariolysis, Left endometriotic cyst drained of its contents, chocolate material draining. Inj Vasopressin injected into the cyst wall and separated from ovarian bed, cyst wall enucleated and proceeded with ovarian cystectomy and sent for HPE. Left ovary reformed using 3-0 vicryl as continuous purse string sutures. Then proceeded with Laparoscopic Myomectomy ,adhesiolysis and Fulguration of Endometriotic deposits.

Her post operative period was uneventful. Her Histopathological report was consistent with endometriotic cyst and AMH level at 6 weeks was 2.1 ng/ml

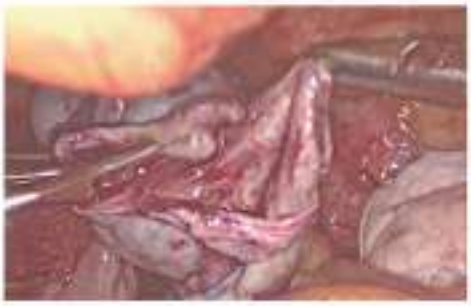




Rupture of cyst



Injecting diluted vasopressin into the interphase



Separate the cyst wall

Ovary reformed

DISCUSSION

Vasopressin is a peptide hormone secreted by the posterior lobe of the pituitary gland, which promotes reabsorption of water in the kidney when administered systemically, but when administered locally, it constricts blood vessels and prevents bleeding such as in esophageal variceal therapy or myomectomy. There are previous studies that demonstrated, vasopressin injection prior to endometrioma resection, the interface between the endometrioma and normal ovarian tissue is dissected, and the amount of deterioration in ovarian function after surgery is reduced compared to the group without vasopressin by reducing the amount of bleeding through vasoconstriction.

The intervention consists of five steps: rupture the ovarian endometrial cyst and remove the "chocolate fluid;" inject diluted vasopressin solution into the interface between endometrioma and ovarian parenchyma; stop injecting until the solution overflow; separate the endometrioma away from the ovarian parenchyma; and coagulate bleeding spots and suture the ovary.

ESHRE guidelines Endometriosis 2022, states that the clinicians are not recommended to routinely perform surgery for ovarian endometrioma prior to ART to improve live birth rates, as the current evidence shows no benefit and surgery is likely to have a negative impact on ovarian reserve. But it also mentions" Surgery for endometrioma prior to ART can be considered to improve endometriosis associated pain or accessibility of follicles."

The GDG recommends that the decision to perform surgery should be guided by the presence or absence of pain symptoms, patient age and preferences, history of previous surgery, presence of other infertility factors, ovarian reserve, and estimated EFI.

RESULTS

The "water injection" diluted vasopressin-assisted laparoscopic excision of ovarian endometrioma was feasible and effective. In the follow-up period, the patient did not report any symptom of dysmenorrhea; and the antimüllerian hormone tests remained within normal levels.

CONCULSION

Our surgical approach demonstrated several noteworthy advantages. After "water injection", the endometrioma and ovarian parenchyma was easily distinguished and separated. The approach avoided normal ovarian tissue destruction during endometrioma separation. The utilization of diluted vasopressin solution might decrease bleeding of ovarian wound, thus reducing the need for cautery and hence minimize damage to the ovarian tissue.

LAPAROSCOPIC MANAGEMENT OF LARGE UTERUS
(32 WEEKS SIZE)

Dr SRILATHA DHULIPUDI MBBS, DGO
Jr. Consultant

Case Report

44Y/O Nulliparous woman came with complaint of Progressive abdominal distension since 1yr, Was evaluated and was diagnosed as a case of large fibroid and planned for surgery.

MRI Pelvis - Subserosal SOL of size 15x18x11 cm noted arising from anterior uterine wall and occupying the pelvis and lower abdomen upto level of kidneys, centered more to the left of midline. There is compression with inferior displacement of uterine corpus seen more to right side. Left ovary is displaced to left iliac fossa. Right ovary noted in the right adnexa. Indirect mass effect on the Urinary bladder seen, Compression of left lower abdominal ureter with mild left hydronephroureterosis. No evidence of any ovarian or adnexal mass or any other obvious pathology observed.

Proceeded with Total Laparoscopic Hysterectomy+ Bilateral Salpingectomy after getting cardiology clearance.

Hysterectomy was done and specimen retrieved partially by In bag morcellation and partially vaginally.

Figure 1 - Uterus Enlarged to 32 weeks size

Figure 2 - Bilateral Round & Ovarian Ligaments coagulated and cut using Ligasure

Figure 3 - Vault is closed with v loc barbed sutures

Figure 4 - Bilateral VUR confirmed by Cystoscopy

Discussion

Uterine fibroids (also known as leiomyomas or myomas) are the most common form of benign uterine tumors. Clinical presentations include abnormal Uterine bleeding, pelvic masses, pelvic pain, infertility, bulk symptoms and obstetric complications. Almost a third of women with leiomyomas will request treatment due to symptoms. Current management strategies mainly involve surgical interventions, but the choice of treatment is guided by patient's age and desire to preserve fertility. The management of uterine fibroids also depends on the number, size and location of the fibroids. Hysterectomy has many positive attributes as a treatment for fibroids. Because the entire uterus is removed, the possibility of new fibroids growing back is eliminated. Menstrual bleeding is stopped permanently and recurrent menorrhagia cannot occur. The risks of cervical, uterine, endometrial, and if combined with bilateral salpingo- oophorectomy, ovarian cancer are eliminated. The likelihood of repeated gynecological surgery is greatly reduced. Laparoscopic hysterectomy is the preferred surgery for patients who do not desire further childbearing to obviate the need for repeat surgeries and because in most cases large uteri are secondary to multiple fibroids. Devascularization of the uterus at the start of hysterectomy and obtaining a good blanche markedly reduces blood loss. The blood supply to the uterus should preferably be controlled before morcellation of an enlarged uterus. Sometimes, the injection of dilute vasopressin solution around the largest myoma before morcellation is begun may help control capillary bleeding, making it easier to identify larger vessels for coagulation with bipolar cautery and keeping the field dry to maintain good exposure.

Occasionally, starting morcellation of a large myoma before its vasculature is totally controlled is the only way to gain enough exposure to complete the hysterectomy.

With large sized uteri, upper quadrant punctures are needed for visualization and proper traction angles with instruments. Exposure of the vascular pedicles can be greatly enhanced by using gravity as a retractor, leaning the patient to the right when working on the left infundibulopelvic ligament and uterine vessels and then to the left when working on the right side.

Conclusion

Total laparoscopic hysterectomy can replace abdominal hysterectomy for large uteri if the surgeon has additional skills and uses specialized techniques beyond those necessary for the majority of total laparoscopic hysterectomies. It should be noted, however, that suspicious fibroids growing in menopausal women, growing on leuprolide or other GNRH agonists, rapidly growing over a short time frame in premenopausal women, exceeding 10 cm in diameter, or that appear visibly suspicious must be treated with prudence because of the rare possibility of a leiomyosarcoma.

STATISTICS

Jan. - Dec. 2022

TOTAL CASE	**1101**
LAPAROSCOPY	312
HYSTEROSCOPY	286
MINOR CASE	135
MALE CASES	44
OBSTETRIC CASE	318
OPEN CASES /OTHERS	6
LAPAROSCOPY	**312**
TLH+BSO	39
TLH	38
LAVH	6
MYOMECTOMY	59
OVARIAN CYSTECTOMY	38
ADNEXECTOMY	16
PARA OVARIAN CYSTECTOMY	3
SALPINGOSTOMY	8
SALPINGECTOMY	9
LAP STERILISATION	11
PCO PUNCTURING	23
ENDOMETRIAL DEPOSIT FULGURATION	38
TUBAL RE CANALISATION	6
LAP CERVICAL ENCIRCLAGE	6
LAP OVARIAN PRP INSTILLATION	7
LAP SACROCOLPOPEXY	3
ISTHMOCELE REPAIR	1
CS SCAR ECOPIC	1

HYSTEROSCOPY	**286**
PRE IVF	123
ENDOMETRIAL POLYPECTOMY	25
SMF RESECTION	10
ENDOMETRIAL SAMPLING	22
SEPTAL RESECTION	09
TUBAL CANNULATION	09
DIAGNOSTIC HYSTEROSCOPY	88
MINOR CASE	**135**
FRACTIONAL CURETTAGE	17
SUCTION EVACUATION	42
CERVICAL ENCIRCLAGE	21
ENDOMETRIAL SAMPLING	09
MIRENA INSERTION	06
MARSUPIALIZATION	03
ERA	07
SSG	05
PPS	07
FETAL REDUCTION	02
THERAPEUTIC CURETTAGE	01
AMNIOCENTESIS	06
CVS SAMPLING	02
EXAMINATION UNDER ANAESTHESIA	02
VAGINAL BOTOX INJECTION	05
MALE SURGERY	**44**
TESA	27
PESA	12

TESE	03
NEEDLE ASPIRATION BIOPSY	02
OPEN CASE	
MYOMECTOMY	03
TAH	02
VH	01
OBSTETRIC CASE	**318**
LSCS	209
FTND	109
ELECTIVE LSCS	112
EMERGENCY LSCS	97
VACUUM DELIVERY	77
Conception + IUI statistics	
Total conceptions	402
Total IUI conception	68
IUI conception rate	10.6 %
Spontaneous	166
COH +Natural	44
ART	124
Annual IVF/ICSI Statistics of 2022	
Total No of cases	476
Total Conception	131
Total Conception Rate	41.06 %
Total FET cycles	248
Conception rate after Frozen ET	35.4 %
Total Fresh cycle	71
Conception rate after fresh cycle	50.7 %
OOP	11
OOP	63.6 %

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