INTROSPECT

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E-JOURNAL BY
OGSOS

EDITOR'S NOTE

We are pleased to proudly present to you "INTROSPECT" - Official online journal from Obstetric and Gynaecological Society of Salem (OGSOS), commenced in order to acknowledge and recognize the work done by our very own members. The online media has become a vital component for the dissemination of knowledge and an imperative vehicle for wide access.

Our OGSOS has always turned heads, be it academics, conferences, skills, culturals, etc. This is our next step, where our vision has taken shape and this endeavor marks a major milestone in taking our society to greater heights. The objective of this journal is to promote research, share ideas, help in day to day clinical practice and promote a spirit of oneness among us. This would provide an exciting opportunity to showcase our work and share our skills. The journal aspires to be vibrant, engaging and accessible, and at the same time integrative and challenging. It will continue to evolve with fresh ideas and guidance at each step,encouraging debates and discussions.

We hope that this journal will offer ample opportunity to our members to learn about and reflect upon the practices and possibilities and help in their achievements and challenges at work. We are privileged to have the expertise and enthusiasm of our authors and believe that every member will play a pivotal role in leading the journal through the exciting phase of its development. Finally we remain very grateful to our President, Vice President, Secretary and Patrons for constant encouragement and guidance. Let us all join together in solidarity and introspect our ideas, thoughts and practices, with the aim of better practices and better outcomes, following the footsteps of our seniors and setting examples for those next in line.

HAPPY LEARNING!

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PRESIDENT'S NOTE

Dear Esteemed Colleagues and Readers,

It is with great pride and enthusiasm that I address you in this edition of Introspect, our cherished platform for advancing knowledge and fostering collaboration in obstetrics and gynecology. I am happy that "Introspect" the brain child of our past president Dr Jayamala has taken shape and continuing to be as a platform for our interaction as contributors to a journal which we consider as our own. As we navigate the evolving landscape of women's healthcare, our commitment to excellence, innovation, and compassion remains steadfast.

This issue of Introspect showcases research, insightful clinical perspectives, and inspiring stories from our community. From latest advancements to addressing disparities in maternal health, the contributions within these pages reflect the dedication and ingenuity of our members. I encourage you to engage with these works, share your expertise, and contribute to the ongoing dialogue that drives our field forward. This edition is to remind us all, that our tireless editors have been persisting in getting articles, editing and bringing forth E journal on a quarterly basis and an acknowledgement of their efforts which may be missed when we tend to overlook non printed materials.

As a society, we stand at the forefront of transformative change, advocating for evidence-based practices and equitable care. Let us continue to support one another, mentor the next generation, and champion the health and well-being of women worldwide.

Thank you for your unwavering commitment to our shared mission. I look forward to seeing you at our upcoming events and continuing this journey together.

Warm regards,

Dr.N.SARAVANA KUMAR

President, OGSOS

SECRETARY'S NOTE

Respected Seniors and my dear colleagues,

It gives me immense pleasure to present our next edition of "Introspect" and the first release of 2025-26 , the official e-journal of the OGSOS. This edition stands as a testament to our society's commitment to academic excellence, clinical insight, and professional growth.

Introspect is more than just a journal — it is a platform that reflects the collective intellect, experience, and enthusiasm of our members. The articles featured in this issue represent a diverse array of topics in obstetrics and gynecology as well as general interest articles , ranging from evidence-based clinical updates and original research to case reports and thought-provoking opinion pieces. Each contribution has been meticulously curated and peer-reviewed to uphold the highest standards of scientific integrity and relevance.

I extend my heartfelt thanks to all the authors who have taken the time to share their work, as well as to our esteemed editorial board, whose dedication and rigorous standards have shaped this journal into a scholarly endeavor we can all be proud of.

This issue of Introspect also captures the spirit of our society — one that fosters knowledge-sharing, encourages academic inquiry, and nurtures professional camaraderie. As we look to the future, it is our hope that this journal continues to inspire and serve as a source of learning and reflection for practitioners, residents, and students alike.

On behalf of the OGSOS, I thank each and every contributor, reviewer, and supporter who has helped bring this edition of Introspect to life. Let us continue to grow together — in knowledge, in service, and in excellence.

Dr.V.DHAVASHREE

Secretary, OGSOS

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Thrombophilia In Pregnancy: A Comprehensive Overview

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INTRODUCTION

Thrombophilia, a condition that increases the risk of blood clots, poses significant challenges during pregnancy. The physiological changes of pregnancy itself, such as increased clotting factors and decreased fibrinolysis, create a hypercoagulable state. When combined with a pre-existing thrombophilic condition, the risk of serious complications for both the mother and the fetus rises dramatically. Understanding the different types of thrombophilia, their clinical presentation, and appropriate management is crucial for ensuring a healthy pregnancy outcome. Figure 1 showing Virchow's triad of coagulation and figure 2 showing factors making pregnancy hypercoagulable state.

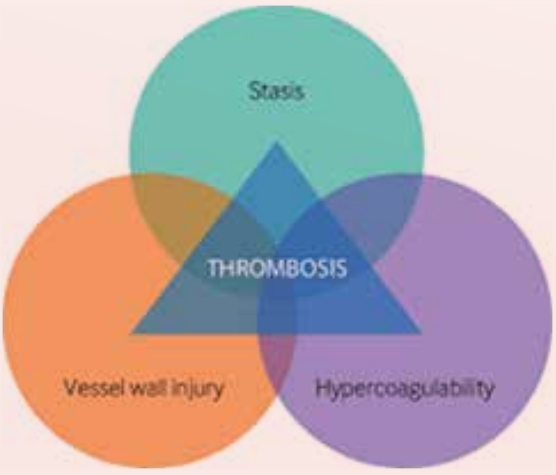


Figure 1

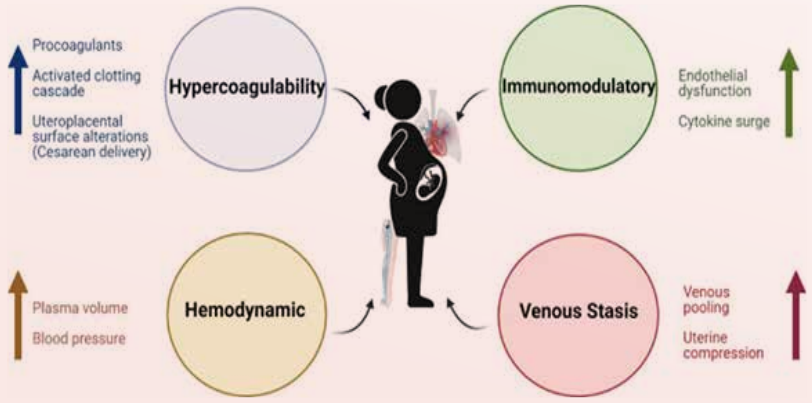


Figure 2

BACKGROUND

Thrombophilia is an umbrella term for a group of disorders, both hereditary and acquired, that predispose an individual to venous thromboembolism (VTE), which includes deep vein thrombosis (DVT) and pulmonary embolism (PE). In the context of pregnancy, thrombophilia is also linked to adverse outcomes such as recurrent pregnancy loss, preeclampsia, placental abruption, and fetal growth restriction. The underlying mechanism is often the formation of microthrombi in the placental circulation, leading to placental insufficiency. The hypercoagulable state of pregnancy means that even a mild thrombophilic defect can become clinically significant.

PREVALENCE

The prevalence of thrombophilia varies depending on the specific type and the population studied. Hereditary thrombophilia is more common in individuals of European descent. The most common hereditary thrombophilia is Factor V Leiden mutation, with a prevalence of about 5% in the general white population. The homozygous mutation is less common, affecting about 1 in 5,000 individuals. Other hereditary forms include prothrombin gene mutation (G20210A), with a prevalence of about 2% in the general population, and deficiencies in natural anticoagulants like protein C, protein S, and antithrombin, which are much rarer, affecting less than 1% of the population.

Acquired thrombophilia, with antiphospholipid syndrome (APS) being the most significant, is also a concern. APS is an autoimmune disorder characterized by the presence of antiphospholipid antibodies, such as lupus anticoagulant, anticardiolipin antibodies, and anti-β2-glycoprotein I antibodies. It's the most common cause of acquired thrombophilia and a leading cause of preventable recurrent pregnancy loss. The prevalence of these antibodies in the general population is low, but they are found in up to 15% of women with a history of recurrent pregnancy loss.

CLINICAL PRESENTATION

The clinical presentation of thrombophilia in pregnancy can be twofold: VTE and adverse pregnancy outcomes.

Venous Thromboembolism (VTE): The most classic presentation is DVT (Deep Vein thrombosis) and . Pulmonary Embolism, a rare but potentially life-threatening complication, presents with sudden shortness of breath, chest pain, and sometimes coughing up blood. While DVT and PE are a risk for all pregnant women, those with thrombophilia have a significantly higher risk, with the risk peaking in the postpartum period. Thrombophilia's are Responsible for >50% of the thrombotic events diagnosed during pregnancy and the postnatal period.

Adverse Pregnancy Outcomes: Thrombophilia is strongly associated with a number of adverse pregnancy outcomes, which can often be the first indication that a thrombophilic condition exists. These include Recurrent Pregnancy Loss (particularly in morphologically &/ or genetically normal fetus), Preeclampsia, Placental Abruption, Fetal Growth Restriction, Stillbirth.

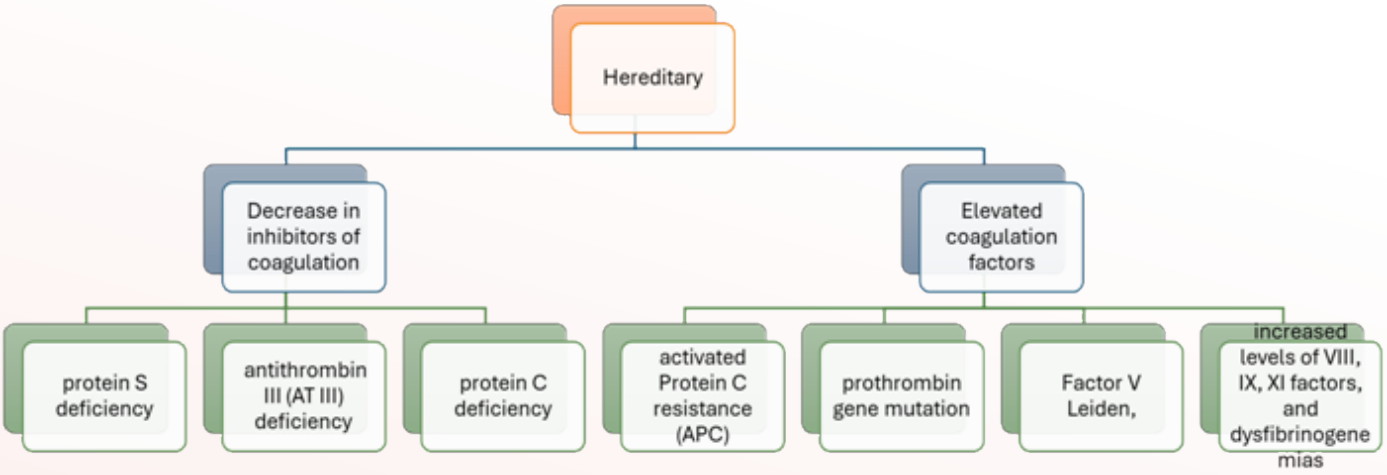


Figure 3 giving classification of thrombophilia's.

There is another risk-based classification proposed on severity of disease/ risk of thromboembolic episodes as follows:

High risk
- AT III deficiency
- Protein C/ S deficiency
- Acquired (APS)

Moderate risk
- Activated C resistance
- Factor V leiden
- Prothrombin gene mutation

Low risk
- Hyperhomocysteinemia

DIAGNOSIS

Diagnosis of thrombophilia is typically prompted by a history of VTE, unexplained adverse pregnancy outcomes, or a family history of thrombotic events. The diagnostic approach involves a combination of clinical evaluation and specific laboratory tests. It's important to note that testing for thrombophilia should ideally be performed outside of pregnancy, as the hypercoagulable state can alter test results, particularly for protein S and antithrombin levels.

Current recommendations for screening for thrombophilia in pregnancy as per ACOG/ ASH

Testing recommendation	Clinical scenario	Strength of evidence
Do not test	Unprovoked VTE/ VTE provoked by surgery/ unspecified VTE	Conditional
Test for thrombophilia	VTE provoked by pregnancy/ postpartum/ COC/ non surgical major transient risk factor	Conditional
Test for thrombophilia known in the family	Individuals with family history of VTE and known thrombophilia, protein C/ S/ antithrombin deficiency	Conditional

Hereditary Thrombophilia Testing:

* Genetic Testing: Blood tests are used to identify genetic mutations, such as Factor V Leiden and prothrombin gene mutation (G20210A).

* Functional Assays: These tests measure the activity of natural anticoagulants.

* Protein C and Protein S: Low levels may indicate a deficiency.

* Antithrombin: A deficiency can be detected by measuring its activity.

Acquired Thrombophilia Testing:

* Antiphospholipid Antibodies: Testing for APS involves measuring the presence of three key antibodies:

* Lupus Anticoagulant: A clotting time test that is sensitive to the presence of these antibodies.

* Anticardiolipin Antibodies: Detected by an enzyme-linked immunosorbent assay (ELISA).

* Anti-β2-glycoprotein I Antibodies: Also detected by an ELISA.

For a diagnosis of APS, at least one of these antibodies must be present on two separate occasions, at least 12 weeks apart, in addition to a clinical criterion (e.g., VTE or adverse pregnancy outcome).

For diagnosis of APS, Sapporo criteria are to be followed which are as following:

Summary of the Sydney Consensus Statement on Investigational Classification Criteria for the Antiphospholipid Antibody Syndrome

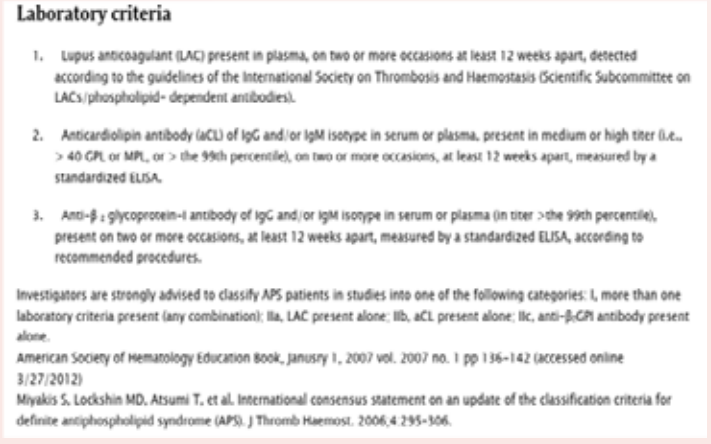
Antiphospholipid antibody syndrome (APS) is present if at least *one of the clinical criteria* and *one of the laboratory criteria* that follow are met.

Clinical criteria

Vascular thromboses

1. One or more documented episodes of arterial, venous, or small vessel thrombosis—other than superficial venous thrombosis—in any tissue or organ. Thrombosis must be confirmed by objective validated criteria. For histopathologic confirmation, thrombosis should be present without significant evidence of inflammation in the vessel wall.
2. Pregnancy morbidity
   a. One or more unexplained deaths of a morphologically normal fetus at or beyond the 10th week of gestation, with normal fetal morphology documented by ultrasound or by direct examination of the fetus, or
   b. One or more premature births of a morphologically normal neonate before the 34th week of gestation because of: (i) eclampsia or severe pre-eclampsia defined according to standard definitions, or (ii) recognized features of placental insufficiency, or
   c. Three or more unexplained consecutive spontaneous abortions before the 10th week of gestation, with maternal anatomic or hormonal abnormalities and paternal and maternal chromosomal causes excluded.

In studies of populations of patients who have more than one type of pregnancy morbidity, investigators are strongly encouraged to stratify groups of subjects according to a, b, or c above.



Laboratory criteria

1. Lupus anticoagulant (LAC) present in plasma, on two or more occasions at least 12 weeks apart, detected according to the guidelines of the International Society on Thrombosis and Haemostasis (Scientific Subcommittee on LACs/phospholipid- dependent antibodies).
2. Anticardiolipin antibody (aCL) of IgG and/or IgM isotype in serum or plasma, present in medium or high titer (i.e., > 40 GPL or MPL, or > the 99th percentile), on two or more occasions, at least 12 weeks apart, measured by a standardized ELISA.
3. Anti-β_2 glycoprotein-I antibody of IgG and/or IgM isotype in serum or plasma (in titer >the 99th percentile), present on two or more occasions, at least 12 weeks apart, measured by a standardized ELISA, according to recommended procedures.

Investigators are strongly advised to classify APS patients in studies into one of the following categories: I, more than one laboratory criteria present (any combination); IIa, LAC present alone; IIb, aCL present alone; IIc, anti-β_2GPI antibody present alone.

American Society of Hematology Education Book, January 1, 2007 vol. 2007 no. 1 pp 136–142 (accessed online 3/27/2012)

Miyakis S, Lockshin MD, Atsumi T, et al. International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). J Thromb Haemost. 2006;4:295–306.

MANAGEMENT

The management of thrombophilia in pregnancy is focused on preventing VTE and improving pregnancy outcomes. The approach is tailored to the specific type of thrombophilia and the patient's individual risk factors. The need for prophylactic/ therapeutic anticoagulation is assessed on basis of risk factors and type of thrombophilia. Various risk factors which are to be taken into account are as follows:

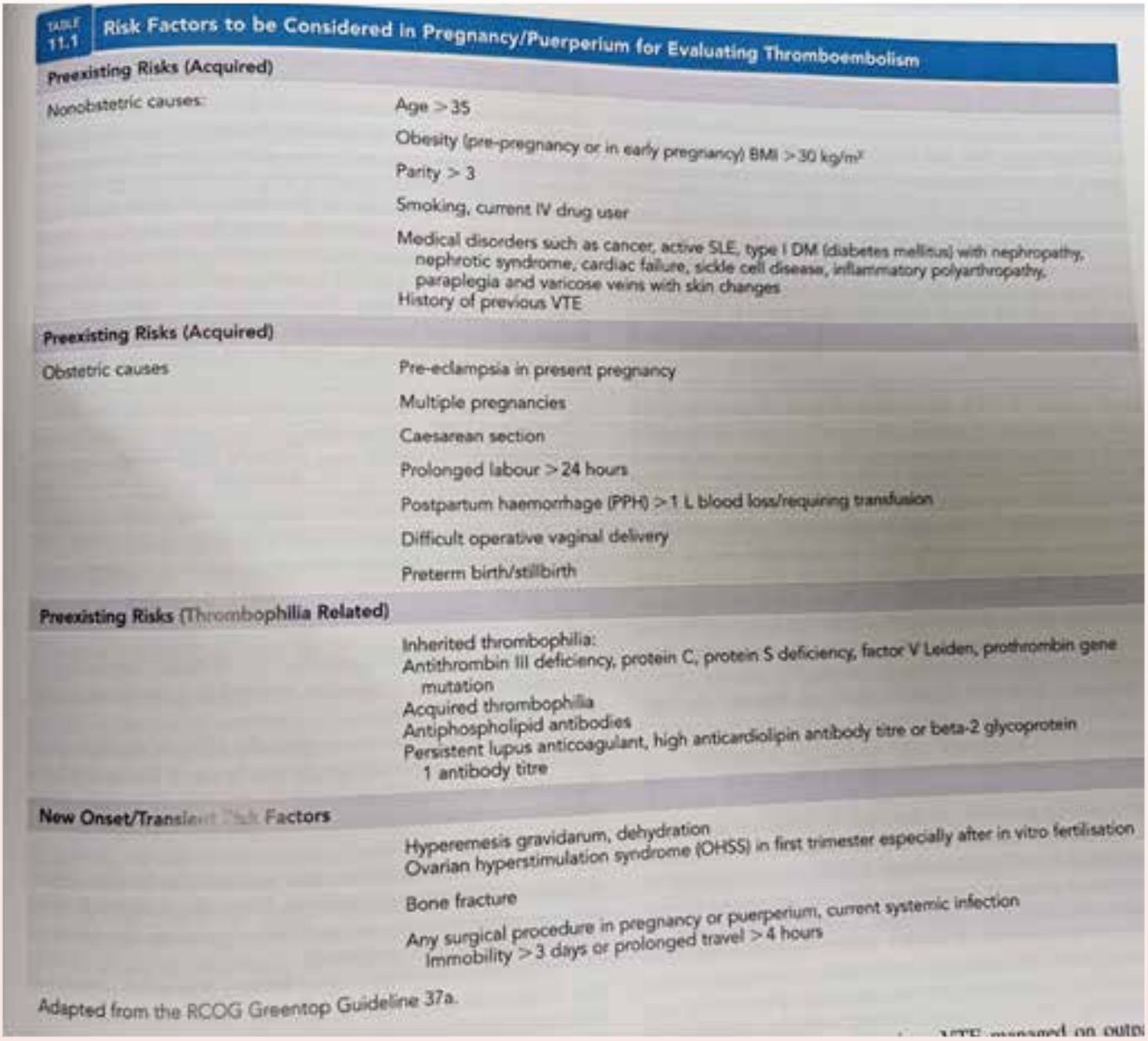


TABLE 11.1 Risk Factors to be Considered in Pregnancy/Puerperium for Evaluating Thromboembolism

Preexisting Risks (Acquired)	
Nonobstetric causes:	Age > 35
	Obesity (pre-pregnancy or in early pregnancy) BMI > 30 kg/m²
	Parity > 3
	Smoking, current IV drug user
	Medical disorders such as cancer, active SLE, type I DM (diabetes mellitus) with nephropathy, nephrotic syndrome, cardiac failure, sickle cell disease, inflammatory polyarthropathy, paraplegia and varicose veins with skin changes
	History of previous VTE
Preexisting Risks (Acquired)	
Obstetric causes	Pre-eclampsia in present pregnancy
	Multiple pregnancies
	Caesarean section
	Prolonged labour > 24 hours
	Postpartum haemorrhage (PPH) > 1 L blood loss/requiring transfusion
	Difficult operative vaginal delivery
	Preterm birth/stillbirth
Preexisting Risks (Thrombophilia Related)	
	Inherited thrombophilia: Antithrombin III deficiency, protein C, protein S deficiency, factor V Leiden, prothrombin gene mutation
	Acquired thrombophilia Antiphospholipid antibodies Persistent lupus anticoagulant, high anticardiolipin antibody titre or beta-2 glycoprotein 1 antibody titre
New Onset/Transient Risk Factors	
	Hyperemesis gravidarum, dehydration Ovarian hyperstimulation syndrome (OHSS) in first trimester especially after in vitro fertilisation
	Bone fracture
	Any surgical procedure in pregnancy or puerperium, current systemic infection Immobility > 3 days or prolonged travel > 4 hours

Adapted from the RCOG Greentop Guideline 37a.

Following are the 2015 RCOG guidelines thromboprophylaxis in pregnancy.

Clinical scenario with risk factors	
>4 current risk factors	Prophylactic LMWH from early pregnancy, may need postpartum after risk assessment
>3 current risk factors	Prophylactic LMWH from 28 wk
>2 current risk factors	Prophylactic LMWH 10 days postpartum
If admitted in 1st trimester with hyperemesis/ OHSS	Offered LMWH unless contraindication
Women with previous VTE	LMWH prophylaxis throughout pregnancy
Previous VTE associated with high risk thrombophilia	Higher doses of LMWH with postpartum prophylaxis for 6 wk till oral anticoagulation initiated
Recurrent VTE on oral anticoagulant	Convert to heparin in early pregnancy
Asymptomatic thrombophilia	LMWH throughout/ from 28 wk based on risk factors till 6 wk postpartum

Prophylaxis and treatment include mechanical modalities and anticoagulation.

Non-invasive/ mechanical methods include following-

- **Early mobilisation**
- **Adequate hydration**
- **Calf exercises**
- **Graduated stockings have a role in**
    - **o Hospitalised with LMWH contraindicated.**
    - **o Post-caesarean with high risk for VTE while in hospital**
    - **o Previous VTE which was managed on outpatient basis.**

Anticoagulation is offered using unfractionated heparin, low molecular weight heparin or heparinoids. Few points above each are mentioned below- Unfractionated heparin-

- **Safe, does not cross placenta, no fetotoxicity.**
- **Subcutaneous administration for prophylaxis**
- **Standard dose 5000 IU 12 hourly for 50-90kg wt.**
- **High risk group, therapeutic dose aims to maintain INR 2-2.5**
- **IV administration in acute episode of DVT**
- **Loading dose 80U/kg bolus followed by 18U/kg IV to achieve aPTT 1.5-2.5 of control**

Low molecular weight heparin:

- Drug of choice for thromboprophylaxis
- Baseline platelet and after initiation should be done.
- High risk group, therapeutic dose is 1ml/ kg twice daily.
- Prophylaxis 40 mg daily
- Cost, longer half-life, partial reversal with protamine sulfate are limiting factors.
- Reversal
    - o < 8 hr- 1 mg protamine sulphate per mg enoxaparin/ 100 U deltaparin
    - o >8 hr- 0.5 mg/ mg enoxaparin or 100U deltaparin
    - o >12-24 hr, due to metabolism, reversal may not be needed.

Heparin induced thrombocytopenia (HIT)

- Early onset or benign or reversible
    - o Generally, in first 48 hrs
    - o Self-limiting
    - o Resolves in 5 days.
- Late onset
    - o Immune mediated
    - o Platelet count <50% of baseline
    - o Formation of antibodies against heparin platelet factor 4 complex
    - o Can cause bleeding/ paradoxical thrombosis.
    - o 5-14 days of therapy
    - o Platelets not to be given.

Contraindications to heparin therapy

- Active antepartum/ postpartum hemorrhage
- History of stroke in last 4 weeks
- Thrombocytopenia <75,000/ ml, hemophilia, von Willebrand disease
- Uncontrolled hypertension (>200/120 mm hg)
- Severe renal and hepatic disease

Alternatives

- Heparinoid- Danaparoid with anti II and anti Xa activity, subcutaneous or IV
- Direct thrombin inhibitors like lepirudin, hirudin, bivalirudin, argatroban. Category B,
- Anti Xa inhibitor- Fondaparinux. Safest newer anticoagulant

Treatment of acute VTE and superficial vein thrombosis is as follows-

Clinical scenario	Recommendation
Pregnant women with acute VTE	Anti thrombotic therapy with LMWH OD/ BD
Pregnant woman with proven acute superficial vein thrombosis,	LMWH
pregnant women receiving therapeutic dose LMWH for the treatment of VTE	No need for routine monitoring of anti factor Xa
Pregnant with acute lower limb DVT	Anticoagulation preferred over catheter directed thrombolysis
Acute pulmonary embolism and right ventricular dysfunction in the absence of hemodynamic instability	Anticoagulation alone Systemic thrombolysis only if associated hemodynamic instability
low-risk acute VTE	Consider outpatient anticoagulation

In cases of antiphospholipid syndrome, following is the recommended thromboprophylaxis

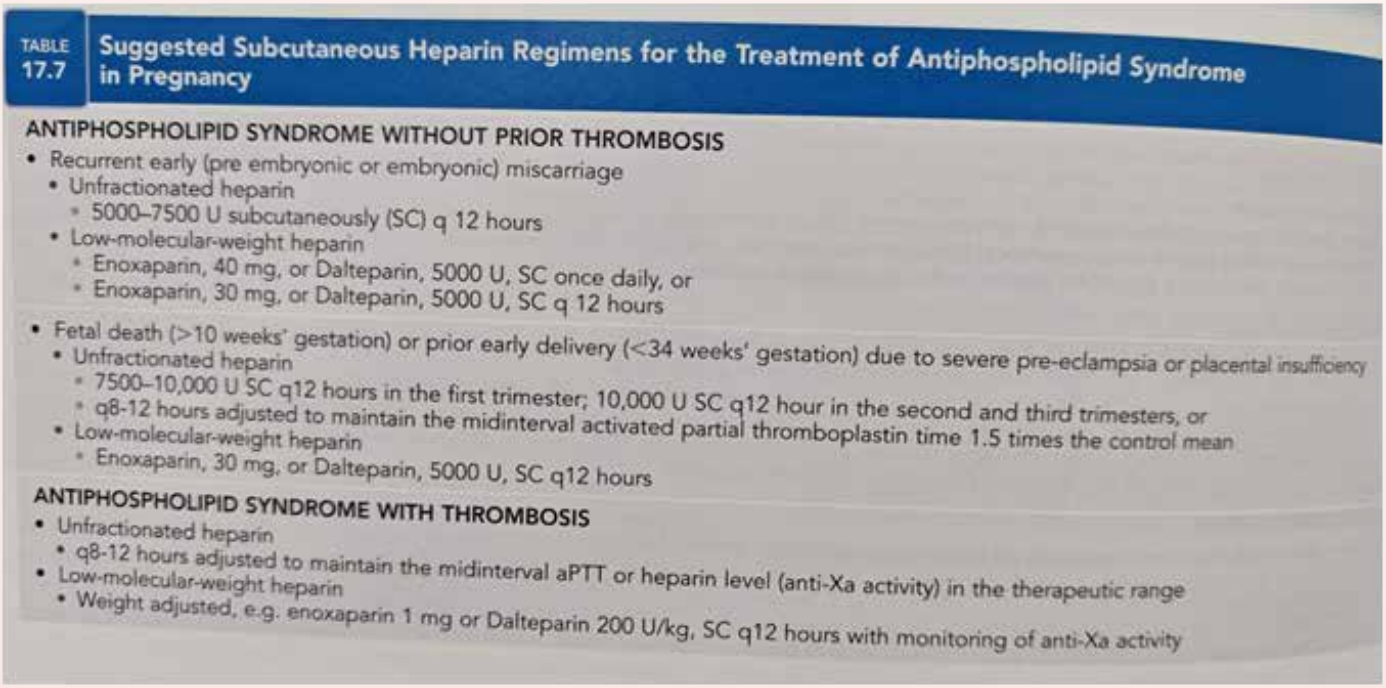


TABLE 17.7 Suggested Subcutaneous Heparin Regimens for the Treatment of Antiphospholipid Syndrome in Pregnancy

ANTIPHOSPHOLIPID SYNDROME WITHOUT PRIOR THROMBOSIS

- Recurrent early (pre embryonic or embryonic) miscarriage
  - Unfractionated heparin
    - 5000–7500 U subcutaneously (SC) q 12 hours
  - Low-molecular-weight heparin
    - Enoxaparin, 40 mg, or Dalteparin, 5000 U, SC once daily, or
    - Enoxaparin, 30 mg, or Dalteparin, 5000 U, SC q 12 hours
- Fetal death (>10 weeks' gestation) or prior early delivery (<34 weeks' gestation) due to severe pre-eclampsia or placental insufficiency
  - Unfractionated heparin
    - 7500–10,000 U SC q12 hours in the first trimester; 10,000 U SC q12 hour in the second and third trimesters, or
    - q8-12 hours adjusted to maintain the midinterval activated partial thromboplastin time 1.5 times the control mean
  - Low-molecular-weight heparin
    - Enoxaparin, 30 mg, or Dalteparin, 5000 U, SC q12 hours

ANTIPHOSPHOLIPID SYNDROME WITH THROMBOSIS

- Unfractionated heparin
  - q8-12 hours adjusted to maintain the midinterval aPTT or heparin level (anti-Xa activity) in the therapeutic range
- Low-molecular-weight heparin
  - Weight adjusted, e.g. enoxaparin 1 mg or Dalteparin 200 U/kg, SC q12 hours with monitoring of anti-Xa activity

Monitoring and Delivery:

* Monitoring: Regular monitoring is essential to assess for side effects of anticoagulation, such as bleeding or heparin-induced thrombocytopenia.

* Delivery: The management of anticoagulation around the time of delivery is critical to minimize the risk of bleeding. The timing of LMWH discontinuation before delivery, and its reintroduction postpartum, must be carefully planned. Salient features for labor/ postpartum management include following-

- **Omit heparin on day of onset of labor.**
- **Induction/ planned CS- omit heparin 24 hr prior for LMWH, 12 hr for UFH.**
- **For regional anesthesia, last LMWH >12 hr earlier**
- **Resume anticoagulation 6 hr after vaginal and 12 hr after caesarean birth**
- **Dose adjustment to achieve INR 2-3**
- **Estrogen OCP contraindicated.**

Management of anticoagulants around the time of delivery (ASH Guideline)

- **Pregnant women receiving therapeutic dose LMWH for the management of VTE.**
  - **o scheduled delivery with prior discontinuation of anticoagulant therapy (conditional recommendation, very low certainty in evidence)**
- **Pregnant women receiving prophylactic dose LMWH- allowing spontaneous labor (conditional recommendation)**
- **Breastfeeding women who have an indication for anticoagulation- using UFH, LMWH, warfarin, acenocoumarol, fondaparinux, or danaparoid as safe options (strong recommendation)**

In conclusion, thrombophilia in pregnancy is a complex condition that requires careful diagnosis and management. With a thorough understanding of the different types of thrombophilia and the appropriate use of prophylactic measures, healthcare providers can significantly reduce the risk of serious complications and improve the chances of a successful pregnancy outcome.

VULVAL LICHEN PLANUS

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INTRODUCTION

Vulval mucosal lichen planus (VMLP) is a T-Cell mediated chronic inflammatory condition that remains underrecognized and underdiscussed. Often presenting with erosive, painful lesions on the vulva and surrounding mucosa, it is not just a dermatological condition—it is a condition that erodes quality of life, intimacy, dignity and exist too quietly on the margins of clinical awareness and social conversations and hence the need for this discussion.

Lichen planus is a relatively uncommon inflammatory dermatologic condition with sub type that can affect the skin, mucosa, nails and scalp. Vulvar lichen planus is a subtypes of lichen planus that is characterized by erosive, papular, or hypertrophic lesions on the vulva, with or without concomitant vaginal involvement. Erosive lichen planus can also affect other mucosal sites, such as the oral cavity, nasal mucosa, esophagus, larynx, conjunctiva, and urethra.

Despite affecting a deeply personal part of the body, VMLP is often misdiagnosed or missed entirely. Many women suffer in silence, dismissed or treated for recurrent infections or vague 'stress-related' issues. What emerges is not just a physical burden-but an emotional, relational, and systemic one.

EPIDEMIOLOGY

Lichen planus is estimated to affect 0.5 to 2 percent of the population; estimates have varied based on geographic location and diagnostic criteria. The incidence and prevalence of vulvar lichen planus has not been clearly established, but vulvar disease may be a common manifestation of lichen planus in females. In one series of 37 females diagnosed with lichen planus, vulvar lesions were present in51 percent.

ETIOLOGY

1.Usually Undetermined

2.Many have attributed to auto immune mechanism, T-cell mediated autoimmune reaction against basal keratinocytes.

3. Hepatitis C

CLINICAL MANIFESTATIONS

Vulval lichen planus often occurs in females 50 to 60 years of age, though younger and older individuals can be affected,

1.Papulosquamous- popular, possibly asymptomatic

2.Hypertrophic- thickened, warty, may mimic malignancy

3.Erosive- the most symptomatic and easily scarred form

4.lichenplanopilaris

SYMPTOMS

1. Itching-vulva is most common, soreness of the vulva.
2. Dyspareunia/ post coital bleeding.
3. Burning vulva especially while passing urine.
4. Irritating vaginal discharge that does not respond to standard therapies for vaginitis.
5. Painful and scarring vulvo vaginal erosions leading to absorption of the labia and stenosis or total obliteration of the vagina in extreme conditions and urethral obstructions.
6. The coexistence of vulvar lichen planus with cutaneous lichen planus and oral lichen planus is common.

Often these symptoms are associated with symptoms of oral LP such as bleeding gums and difficulty in tolerating even mild spicy foods.

DIAGNOSIS

Vulval LP isn't just hard to diagnose-it's hard to talk about. It sits at the intersection of dermatology, gynecology, and psychology-and too often, it falls through the cracks.

It is the moment, a clinician names the condition, often brings relief, not because the disease is benign, but because the patient's pain is affirmed and validated. That shift-from being dismissed to being heard-is a powerful acknowledgment of suffering.

This diagnosis brings not only validation but also fear-especially with the risk of malignancy.

On examination, white striae on the vulva may clinch the diagnosis which is often associated with white striae on the buccal mucosa- Wickham striae and occasionally, a violaceous border is noted.

The lesions may occur on the labia minora and vestibule as isolated lesions on an otherwise normal vulva, or they may be associated with marked architectural destruction, including loss of the labia minora and narrowing of the introitus. Small, localized lesions on the labia majora are less common. Anal involvement is rare.

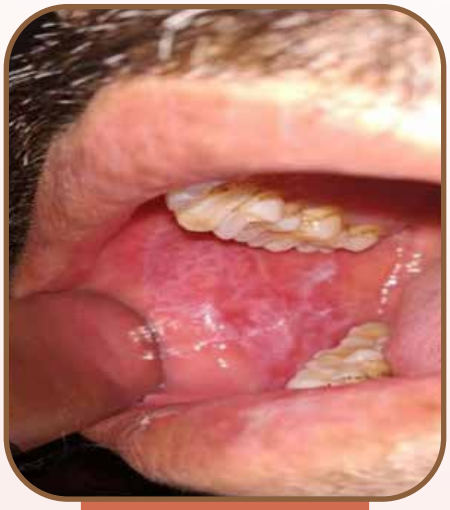
Vaginal involvement has been reported in up to 70 percent of patients with erosive lichen planus.

Extreme conditions- labial fold fusion, stenosis of the vaginal opening (partial/complete).

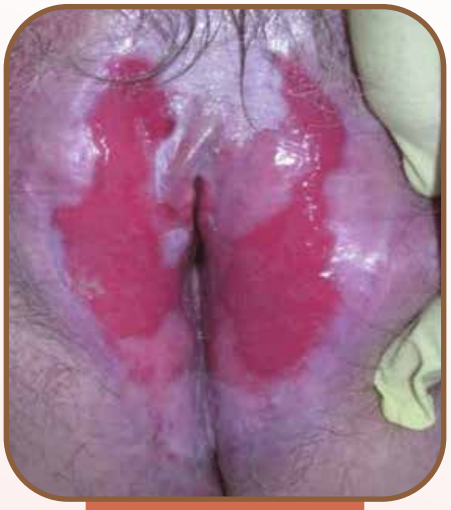
Confirmation is often by biopsy. Biopsy is essential if diagnosis is uncertain, suspicion of Vulvar Intraepithelial Neoplasia (VIN) / Squamous Cell Carcinoma (SCC),(if recalcitrant lesions).

Screening for autoimmune comorbidities such as diabetes mellitus (DM), thyroid disorders, and hepatitis C virus (HCV) should be considered.

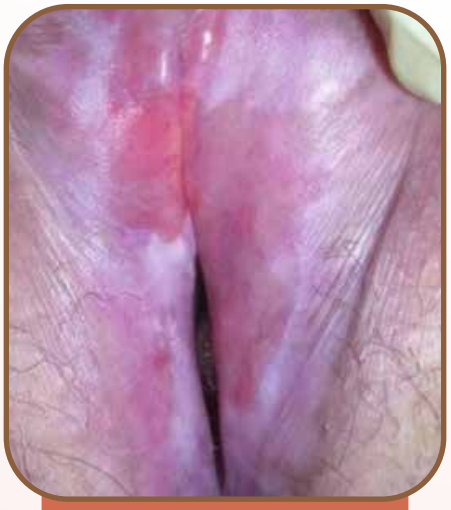
Though genetic predisposition is not fully established, obtaining a good family history may be helpful.



LICHEN PLANUS OF ORAL MUCOSA WICKHAMSTRIAE



VULVAR LICHEN PLANUS



VULVAR LICHEN PLANUS WITH OBLITERATION OF THE VULVAL FOLD

DIFFERENTIAL DIAGNOSIS

1. Lichen sclerosus
2. Autoimmune bullous diseases
3. Plasma cell vulvitis
4. Desquamative inflammatory vaginitis(DIV
5. Crohn disease
6. Behcet Syndrome
7. Erythema multiforme and stevens Johnson syndrome
8. Lichenoid drug eruption

MANAGEMENT (MEDICAL MANAGEMENT)

TOPICAL MANAGEMENT

Management requires not just pharmalogical intervention but multi-disciplinary approach including psychosexual and social rehabilitation.

1.Ultra-potent topical corticosteroids (eg., clobetasol propionate ointment) are gold standard-initiate nightly or twice daily, taper based on response.

2. Maintenance therapy is necessary after remission is achieved because symptoms often recur once treatment is discontinued. The goal of the maintenance phase is to maintain improvement while reducing the frequency of topical corticosteroid treatment to minimize risk for local corticosteroid side effects. This is performed as a step-wise process that eventually reveals the lowest dose and frequency that sustains remission.

3.For vaginal disease, steroid suppositories (hydrocortisone or prednisolone) are effective to reduce mucosal inflammation. (subject to availability)

4.Steroid foams can be applied locally to reduce inflammation.

5.Local application of -Triamcinolone acetonide a corticosteroid, Topical Tacrolimus, a calcineurin inhibitor, are effective either alone or in combination with systemic therapy.

Adjunctive measures - Avoid irritants, fragrance products, and tight clothing. Encourage cotton underwear. Use emollients, sitz baths, lidocaine gel for symptom relief and to minimize scratching-triggered cycles.

Consider systemic treatment if symptoms persist despite potent topical therapy and low compliance.

SYSTEMIC MANAGEMENT

1. Oral prednisone four-to-six-week course of 40 to 60mg per day, tapered over four to six weeks and commence topical corticosteroid therapy after one week. Twice daily sitz baths in water followed by the application of a greasy emollient (such as petroleum jelly) may also help restore the epithelial barrier.

2. An alternative approach is the use of intramuscular Triamcinolone. Intramuscular Triamcinolone(1mg/kg) may be given as a single dose or as a series of injections separated by one month.

3. Either oral prednisone 40 mg daily tapered over four to six weeks, or intramuscular triamcinolone, 60 to 80 mg once per month three to four month, may be tried before local maintenance therapy is initiated.

4. Immnuotherapy with Methotrexate- an anti-metabolite, Cyclosporine-an immuno suppressive agent, Azathioprine-an immuno suppressive agent, Thalidomide-an immuno modulatory agent, Mycophenolate mofetil an immuno modulatory agent may be started in patients resistant to initial management strategies.

5. Apremilast, a phosphodiesterase inhibitor widely used for the treatment of psoriasis, has promising results with less side effects.

6. Retinoids such as Acitretin can be added as adjunctive treatment along with other systemic therapies or used alone as a second-line management.
7. Hydroxychloroquine an antimalarial can also be tried as adjuvant.

SURGICAL MANAGEMENT

In patients with adhesions and scarring, vulvar or vaginal involvement by lichen planus can lead to anatomic distortion and functional limitations secondary to the formation of adhesions and scarring. Dilators and surgery can be useful for improving these complication after mucosal inflammation is controlled with medical therapy. Follow-up care is necessary to maintain benefit after these procedures:

Dialator therapy (with rigid dilators) can be effective for distending mild to moderate vaginal synechiae that interfere with intromission or speculum insertion.

Severe vaginal synechiae require surgical release. All surgical approaches to vaginal occulusion must be followed by diligent therapy with dilators and topical therapy to maintain control of the inflammation. Isolated vaginal procedures without this follow-up therapy invariably result in recurrent vaginal occlusion.

Although topical estrogen has no therapeutic role in treatment of the vulvar lichen planus, its contribution to epithelial integrity of the vagina cannot be underestimated. Many individuals with vulval lichen planus are post-menopausal, making the estrogen an important adjunct to restore suppleness.

Treatment of concurrent infection- An associated bacterial or fungal infection should be treated with antimicro-bials concurrently with corticosteroid therapy.

SPECIAL MENTION

1. Vulvo – vaginal-gingival syndrome- The vulvo-vaginal-gingival syndrome is a variant of erosive lichen planus that involves the epithelium of the vulva, vestibule, vagina, and mouth. Additional sites (eg: skin, esopha-gus, ear canal) may also be involved. Although all three areas can be affected, the lesions may not be concurrent. White plaques,or a whitish and lace-like reticular pattern may occur on the buccal mucosa, tongue, and palate. Scarring and stricture formation are common and a major cause of long-term morbidity. Vulvo-vaginal-gingival syndrome is particularly resistant to treatment.
2. Genitourinary syndrome of menopause Vulvovaginal atrophy or Atrophic vaginitis is common in peri- or postmenopausal females, in females with hypoestrogenism, and may coexist with vulvar lichen planus. The clinical features can be similar to erosive lichen planus; however, hyperkeratosis and wickhamstriae are not evident. Histology should not show any of the features of lichen planus.

Associated malignancy A slightly increased risk of vulvar malignancy in patients with vulval lichen planus has been postulated but not confirmed. Vulval squamous cell carcinoma that develops in patients with vulval lichen planus may demonstrate aggressive features.

PSYCHO SEXUAL REHABILITATIONS

Psycho sexual rehabilitation plays a vital component of managing vulval lichen planus. Management includes counseling, pelvic floor physiotherapy, and the use of lubricants or topical therapy to reduce discomfort. Addressing anxiety, relationship dynamics, and sexual confidence through cognitive behavioral therapy (CBT) or psychosexual therapy can significantly improve quality of life. A multidisciplinary approach involving dermatologist, gynecologists, and mental health professionals is essential for holistic care.

PSYCHOSOCIAL REHABILITATION

Psychosocial rehabilitation in vulval lichen planus focuses on improving the emotional, psychological, and social well-being of affected patients. Chronic pain, itching, and sexual dysfunction can lead to anxiety, depression, low self-esteem, and relationship difficulties. Supportive interventions may include psychological counseling, support groups, stress management techniques, and education about the condition. Encouraging open communication with partners and providing reassurance can also help patients cope better. A holistic, multidisciplinary approach is essential to enhance coping skills, restore confidence, and improve overall quality of life.

CONCLUSION

VMLP is more than a dermatological condition-it is a test of how we respond to pain hidden in silence. To care for these patients, we must expand beyond evidence-based practice into empathy-based approach. We must learn not only to treat-but to listen, to validate, and to accompany.

Obesity And Its Impact On Female Reproductive Health

Dr Esai Amuthu

INTRODUCTION

Defined primarily by an excessive accumulation of adipose tissue, obesity is no longer merely a reflection of lifestyle choices metamorphosed into a multifaceted disorder with profound implications on physiological and metabolic functions

Alongside the rising obesity issue, female infertility, defined as the inability to attain clinical pregnancy following 12 months of consistent unprotected intercourse, is increasingly becoming a major global health concern. structures that underpin female fertility is susceptible to perturbations, and obesity, with its widespread systemic effects, has been identified as a significant disruptor.

Infertility can be a source of profound psychological distress, impacting relationships, mental health, and overall quality of life. Moreover, in many cultures, childbearing is intricately linked with societal roles and expectations, and infertility can lead to stigmatization and marginalization.

The economic burden associated with treating infertility, particularly in the context of obesity, is substantial diagnostic procedures to therapeutic interventions, the costs can be prohibitive, placing additional strain on already stretched healthcare systems

Impact of obesity on polycystic ovary syndrome

PCOS, a hormonal disorder with irregular menstrual cycles and hyperandrogenism, is often linked with metabolic issues like insulin resistance. The complex interaction between obesity and PCOS influences both its etiology and

management. Visceral adiposity, more than subcutaneous fat, is notably associated with the hormonal imbalances in PCOS, impacting female fertility . Obesity, particularly central obesity, exacerbates the metabolic and reproductive abnormalities associated with PCOS . Women with PCOS and obesity are at a heightened risk for insulin resistance, hyperinsulinemia, and type 2 diabetes . The hyperinsulinemia, in turn, can lead to increased ovarian androgen production, further aggravating the symptoms of PCOS . Additionally, obesity influences the secretion

of various adipokines, such as leptin and adiponectin, which are known to play significant roles in reproductive health. Elevated leptin levels, often found in obese individuals, can disrupt normal ovarian function and are associated with the pathophysiology of PCOS .Conversely, adiponectin, known for its anti-inflammatory and insulin-sensitizing properties, is typically reduced in obesity and may contribute to the reproductive dysfunctions seen in PCOS.This vicious cycle underscores the importance of weight management in women with PCOS. However, studies clearly indicate a higher prevalence of PCOS among overweight and obese individuals. In women with PCOS, cardiovascular risk factors such as hypertension and dyslipidemia are intensified by obesity.

Impact of obesity on menstrual disturbances

Obesity's influence onmenstrual disturbances is multifaceted . The adipose tissue, abundant in obese individuals, plays a significant role in steroid metabolism, leading to increased estrogen production. This increase is largely due to the activity of aromatase in adipose tissues, which converts androgens to estrogens. The elevated estrogen levels associated with obesity can disrupt the hormonal balance, potentially contributing to menstrual disturbances .Obesity also alters the levels of adipokines like leptin and adiponectin, which are crucial in regulating reproductive hormones and menstrual cycles. High leptin levels in obese women can lead to menstrual irregularities and anovulation . Elevated estrogen levels can disrupt the regular menstrual cycle, leading to early menarche in adolescents and potentially early menopause in older women . metabolic syndrome, which is closely associated with obesity, has been linked to menstrual disturbances. The syndrome's components, including insulin resistance, dyslipidemia, and hypertension, can influence reproductive health and menstrual regularity

Impact of obesity on female infertility

Obesity's influence on female infertility is a topic of significant concern in reproductive medicine . The altered levels of adipokines in obesity, particularly the decrease in adiponectin and increase in leptin, are implicated in the pathogenesis of infertility. These changes can affect ovarian function, disrupt the hormonal balance necessary for ovulation, and impair endometrial receptivity treatment of obesity should be the initial aim in obese infertile women before embarking on fertility treatments

Impact of age and obesity on female reproductive health

The interplay between age and obesity significantly influences female reproductive health. In women of reproductive age, obesity is associated with various reproductive challenges, including impaired ovulatory function, reduced implantation and pregnancy rates, and increased miscarriage rates. These issues become more pronounced with advancing age, particularly in women approaching the upper limits of their reproductive years. women aged 38 years and older with obesity experience sub-optimal reproductive performance, impacting

fertilization rates, embryo development, and pregnancy outcomes overweight and obesity in early adulthood are linked to an increased risk of menstrual irregularities and hypertension in pregnancy.

Molecular and physiological mechanisms

The complex relationship between obesity and female infertility is driven by numerous molecular and physiological mechanisms.

Disruption of the hypothalamic-pituitary-ovary axis

The hypothalamic-pituitary-ovary (HPO) axis, which orchestrates a delicate balance of hormonal interactions, is fundamental to female reproductive physiology. It regulates the cyclical patterns of menstruation and the intricate process of ovulation . However, obesity disrupts this harmonious system, primarily through elevated leptin levels . Leptin, reactive oxygen species (ROS), and other adipokines are significantly altered in obesity, contributing to the dysregulation of the HPO axis.This elevation in leptin can interfere with the rhythmic secretion of Gonadotropin-releasing hormone (GnRH) from the hypothalamus. Specifically, high levels of leptin are thought to disrupt the pulsatile nature of GnRH release. This disruption can lead to altered secretion patterns of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which are crucial for the normal menstrual cycle and ovulation . In obesity, the increased leptin levels may desensitize the GnRH neurons to leptin's regulatory effects, leading to a dysregulation in the release of GnRH. This dysregulation can result in either an increase or decrease in the frequency and amplitude of LH and FSH pulses. The altered LH and FSH pulses can then impact follicular development, leading to menstrual irregularities and ovulatory dysfunction . Additionally, chemerin, another adipokine, is often elevated in obesity and metabolic syndromes, contributing to the disruption of normal reproductive functions. Chemerin has been implicated in the regulation of adipogenesis and inflammation, and its elevated levels in obesity are associated with insulin resistance and dysregulated lipid metabolism, further impacting the HPO axis . Adiponectin, typically decreased in obesity, plays a role in insulin sensitization and has anti-inflammatory properties. Its reduction in obesity can exacerbate the hormonal imbalances associated with reproductive dysfunctions. The decrease in adiponectin in obese individuals may contribute to insulin resistance, which can further impact the HPO axis by affecting the secretion and action of GnRH, LH, and FSH. The consequences are wide ranging: from anovulatory cycles and irregular menstrual periods to a challenging fertility landscape. This hormonal imbalance not only diminishes natural conception chances but also poses challenges for assisted reproductive treatments.

The endocrinological role of adipose tissue

Once regarded merely as a passive fat store, adipose tissue is now understood to be an active endocrine organ.Beyond their central role in metabolic homeostasis, these adipokines also intersect with reproductive functions. Imbalances in adipokine levels can affect ovarian steroidogenesis, resulting in a disrupted hormonal environment. This altered balance, particularly between estrogen and progesterone, can adversely affect the endometrial setting, rendering it less receptive to embryo implantation and early gestation.

The dual threat of insulin resistance and hyperinsulinemia

Obesity's distinct metabolic profile, marked by insulin resistance, has significant repercussions for reproductive health. Elevated insulin levels, which arise as a countermeasure to resistance, trigger a cascade of effects in the ovary. Specifically, they prompt the ovarian theca cells to overproduce androgens, leading to a hyperandrogenic state .This scenario, reminiscent of PCOS, is further exacerbated when insulin inhibits the liver's synthesis of sex hormone-binding globulin(SHBG). The result is an increase in freely circulating androgens,which can interfere with ovulation, causing menstrual irregularities and reducing fertility potential .

The inflammatory onslaught and oxidative stress

Obesity is frequently associated with chronic inflammation. adipose tissue becomes a major producer of proinflammatory molecules, such as TNF-a and IL-6. Accompanying this rise in inflammation, a hallmark of obesity, is an increase in oxidative stress. This oxidative stress in obesity is characterized by an imbalance between the production of ROS and the body's antioxidant defenses. The excess adipose tissue in obesity contributes to this imbalance, exacerbating inflammation and leading to a cycle of oxidative stress and further inflammatory response, which can disrupt metabolic homeostasis. These combined factors create a hostile environment for oocytes,

affecting their quality and viability. Furthermore, the inflammatory and oxidative conditions can negatively influence the endometrial lining, reducing its receptivity to embryo implantation and thereby presenting substantial challenges to successful conception.

The reproductive repercussions of dysregulated lipid metabolism

The impact of obesity on lipid metabolism is significant. Elevated triglycerides, reduced high-density lipoprotein-cholesterol (HDL-C) levels, and a general dyslipidemic state have implications not only for cardiovascular health but also for reproductive outcomes . Lipotoxicity in ovarian granulose cells, stemming from excessive lipid accumulation, can induce cellular stress through multiple interconnected pathways.

The accumulation of lipids leads to increased ROS production, causing oxidative stress . Concurrently, this lipid overload disrupts endoplasmic reticulum (ER) function, triggering ER stress and the unfolded protein response, potentially leading to apoptosis if unresolved . Additionally, lipid accumulation can provoke an inflammatory response by stimulating proinflammatory cytokines, exacerbating cellular stress . This
scenario is further complicated by impaired mitochondrial function, leading to decreased adenosine triphosphate (ATP) production and further ROS generation, contributing to the overall cellular stress and dysfunction in granulosa cells .
Such stress can hinder their function, affecting oocyte maturation, follicular development, and overall reproductive capability .

The mitochondrial malaise

Mitochondria, cellular powerhouses, play pivotal roles in a plethora of physiological processes, including oocyte maturation and embryonic development. However, obesity can lead to mitochondrial dysfunction. This dysfunction, marked by reduced
ATP synthesis and increased ROS production, can adversely affect oocyte health, impacting both its cytoplasmic and nuclear maturity. Such challenges to mitochondrial function can limit the developmental potential of embryos, creating significant obstacles to successful conception and subsequent embryonic development . In essence, the link between obesity and female infertility is supported by numerous molecular mechanisms, each adding to the diverse reproductive challenges encountered by obese women.

The role of biogenic amines in metabolic disturbances and reproductive health

Biogenic amines, such as serotonin, dopamine, histamine, and norepinephrine, are derived from amino acids and play critical roles in various physiological processes, including mood regulation, appetite control, and cardiovascular function. serotonin and dopamine have been implicated in the regulation of energy balance and appetite, processes often altered in obesity. Dysregulation in these neurotransmitters can contribute to the hormonal imbalances seen in obesity, indirectly affecting reproductive health .histamine and norepinephrine are involved in inflammatory responses and stress regulation. Alongside these biogenic amines,polyamines, including spermine,spermidine, and putrescine, are also integral to the discussion of metabolic and reproductive health. These polyamines are involved in cellular and genetic metabolism, aiding in transcription, translation, signaling, and post translational modifications, which are crucial for maintaining cellular homeostasis and responding to metabolic challenges. Such dysregulation can have implications for reproductive health, as polyamines are known to be involved in cell growth and differentiation, processes that are essential for reproductive function .

Weight loss as a therapeutic strategy for infertility in obese women

Weight loss emerges as a promising avenue for restoring reproductive health in obese women. A myriad of weight loss strategies, ranging from lifestyle modifications to medical interventions, have been explored for their efficacy in ameliorating obesity-induced infertility.

Pharmacological interventions

Pharmacological interventions are key in addressing the reproductive challenges associated with obesity. Metformin, primarily an anti-diabetic drug, is widely recognized for its insulin-sensitizing properties, which have been beneficial in regulating menstrual cycles and improving ovulation rates in obese women, glucagon-like peptide-1 (GLP-1) agonists, originally used for type 2 diabetes, have emerged as a promising option. These agonists have shown potential in improving insulin resistance linked with PCOS, commonly observed in obese women. By enhancing insulin sensitivity, GLP-1 agonists could potentially correct hormonal imbalances and improve fertility outcomes that antioxidants such as a-lipoic acid and myoinositol may ameliorate oxidative stress in the oocyte environment, potentially contributing to improved fertility . Beyond these, the focus is also shifting towards drugs that target adipokines or their receptors. This includes exploring the therapeutic roles of agents that either enhance adiponectin activity or increase its levels,considering adiponectin's crucial role in ovarian function and hormone production.Restoring leptin sensitivity through such interventions could play a significant role in addressing various reproductive dysfunctions linked to obesity.

Surgical interventions

Surgical interventions, particularly bariatric surgery, have been increasingly recognized as a viable approach to address infertility challenges in obese women.

Lifestyle modifications

Lifestyle modifications have emerged as a pivotal therapeutic strategy for addressing infertility in obese women. Comprehensive programs focusing on weight loss have demonstrated significant improvements in reproductive outcomes across various fertility treatments . Particularly in women with PCOS, a condition often associated with obesity and infertility, lifestyle changes have been shown to restore reproductive potential by enhancing insulin sensitivity and regulating luteinizing hormone levels structured exercise training programs, when compared to specific dietary interventions, have also shown promise in treating obese PCOS patients with anovulatory infertility .

Obesity and its implications on assisted reproductive technologies

Assisted reproductive technologies (ART), such as in vitro fertilization (IVF) and intracytoplasmic sperm injection (ICSI),have emerged as revolutionary tools in the realm of reproductive medicine, offering hope to countless couples facing infertility

challenges. Obesity has been identified as a significant factor that negatively impacts various ART outcomes while obesity might diminish clinical pregnancy rates after IVF, its impact on ICSI cycles appears to be less pronounced, suggesting potential intrinsic sperm dysfunctions secondary to obesity might be circumvented in ICSI procedures .

Conclusion

The rising global prevalence of obesity and its profound impact on female reproductive health has become a pressing concern. Obesity's systemic effects, from hormonal imbalances to inflammation, disrupt the intricate processes of female fertility.

Weight loss, through pharmacological interventions, surgery, or lifestyle changes, offers a promising solution. Additionally, the success of assisted reproductive technologies like IVF is influenced by obesity, highlighting the need for optimal body mass index.

CA 125- Let's re-collect our understanding

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INTRODUCTION

Ovarian cancer is the leading cause of death among all the gynaecological malignancies [2] 70% of them present at stage 3 or stage 4. There are no documented effective screening methods that can reduce the mortality of ovarian cancers [1]. Studies using CA125, ultrasonography of the pelvis, and pelvic examination do not have an acceptable level of sensitivity and specificity. The US Preventive Services Task Force recommends against screening asymptomatic women for ovarian cancer with these modalities like CA125.Yet , CA125 has its utility in establishing the risk malignancy index of suspected ovarian masses.Carbohydrate antigen 125 (CA125) is an antigen used in the diagnosis of epithelial nonmucinous ovarian cancers.CA125 may be elevated in many benign and malignant conditions, so elevated levels can cause confusion over patient management. In this writeup, let us recollect our understanding of CA125.

HISTORY

CA125 was first identified by Bast et al. in 1981 [4]. Bast and his team isolated the murine monoclonal antibody OC125, which recognises an epitope on a molecule called CA125 so-named because it is the 125th antibody produced against the ovarian cancer cell line. It is also known as mucin 16 (MUC16) because it is encoded by the MUC16 gene, located on chromosome 19 [5] .In most assays, a normal level of CA125 is considered to be <35 IU/ml [6].

ORIGIN

CA125 is expressed in tissues derived from embryonic coelomic epithelium such as peritoneum, pleura and pericardium[3] .CA125 is synthesised by the mesothelial cells in response to certain stimuli, such as mechanical stress and inflammation.Studies have demonstrated a link between elevated CA125 and the presence of fluid in the serosal spaces(pleural effusion,ascites) regardless of aetiology of fluid accumulation.

CA125 and ovarian tumours

Elevated values of CA125 are seen in patients with ascites associated with ovarian cancer, with a positive correlation between ascites volume and CA125 level. This suggests that the CA125 antigen is not produced directly by the tumour and is therefore not a tumour marker per se. Instead, it is released by the mesothelial cells in response to the mechanical stretch produced by the fluid. Levels of CA125 in the ascitic fluid do correlate with the serum levels, but are much higher than those seen in the blood. This indicates that the antigen originates in the ascitic fluid, rather than in the tumour itself [12]

In vitro studies have demonstrated that mechanical stretch of mesothelial cells causes upregulation of MUC16, lending further support to this theory.CA125 may also be released in response to inflammatory stress, and in response to stimulation with tumour necrosis factors and interleukins.

Serum CA125 levels are often elevated in ovarian nonmucinous epithelial cancers.There is little evidence that this approach can reduce mortality from ovarian cancer, as 50% of women with stage I disease, and those with occult cancers identified at prophylactic surgery may have normal levels of CA125.

Current national guidance recommends CA125 testing as an initial investigation for patients with symptoms of ovarian cancer.NICE guidance for CA125 testing in primary care recommends CA125 testing all women presenting with symptoms of ovarian cancer such as persistent abdominal distension, early satiety/loss of appetite, pelvic or abdominal pain, increased urinary urgency/ frequency, unexplained weight loss, fatigue or changes in bowel habit. If the patient's CA125 level is greater than 35 IU/ml, then an abdominal and pelvic ultrasound is indicated [2]

CA125 level is required to calculate a risk of malignancy index (RMI) for patients presenting with ovarian cysts. A level of over 200 IU/ml should trigger referral to a cancer centre for subsequent management. CA125 testing is useful in patients for follow-up of ovarian cancer. CA125 level may be used to assess patient response to chemotherapy and surgical treatment.Serum levels are expected to fall by half within 10 days of surgical resection. Postoperative levels correlate with residual tumour mass and have a considerable value, which is predictive for survival. For patients who enter complete remission with chemotherapy treatment, the median time for CA125 normalisation is 1.5 months, while for patients achieving partial remission it is 4 months. Despite this, for 40% of patients achieving normal CA125 concentrations, microscopic or macroscopic disease will be found at second-look surgery. The British Gynaecological Cancer Society (BGCS) advises that CA125 measurement during follow-up is not mandatory and has not been proven to be of survival benefit.So decision for CA125 follow-up must be individualised.CA125 levels are elevated in only 75–90% of patients with advanced disease,so it is not an effective screening tool or stand-alone measurement.Higher levels are seen in smokers, women with breast cancer and women using hormone replacement therapy (HRT), although in most women these levels were still within the normal range.

CA125 levels and menstruation

CA125 levels fluctuate across the menstrual cycle, with a peak during menstruation followed by a steady decline until the end of the cycle.

CA125 and Pregnancy

Serum CA125 levels are altered in pregnancy, with a rise in the first trimester being attributed to increased production by the decidua.From the start of the second trimester, a reduction in values can be observed.Levels may be further increased by pregnancy complications, such as pre-eclampsia.For these reasons, a higher cut-off value for serum CA125 levels in pregnancy may be applicable but no consensus has been reached.

CA125 and endometriosis

A clear link has been identified between raised CA125 levels and endometriosis. For patients with stage II and above endometriosis, CA125 levels may reach into the hundreds of units per millilitre, compared with healthy controls. Levels have also been shown to be predictive of considerable pelvic adhesions in such patients.CA125 levels have been shown to be elevated in various other benign conditions, such as benign ovarian cysts, tubo-ovarian abscess, pelvic inflammatory disease, fibroids and ovarian hyperstimulation syndrome. Many nongynaecological conditions are also associated with markedly elevated CA125 levels, including liver cirrhosis,lung diseases like interstitial lung disease,tuberculosis and heart diseases such as heart failure, atrial fibrillation and pericardial disease [10].

CA125 and Premenopausal Women.

CA125 level is not routinely needed for the diagnosis of a simple cyst in premenopausal women. It also advocates use of the International Ovarian Tumor Analysis (IOTA) Group M (malignant) and B (benign) rules for ovarian cyst classification.Within this risk stratification process, a serum CA125 level is not required. Any patient with a single malignant feature ('M-rule') identified on ultrasound requires referral to the gynaecological oncology team.

CA125 and Postmenopausal Women.

Current guidance for CA125 testing in postmenopausal women with ovarian cysts is more straightforward. CA125 level is crucial if any cystic lesion of more than 1 cm in diameter is identified on the ovary. Inappropriate use of CA125 testing may result in unnecessary investigations and invasive treatments, which in turn can lead to considerable anxiety for the patient.

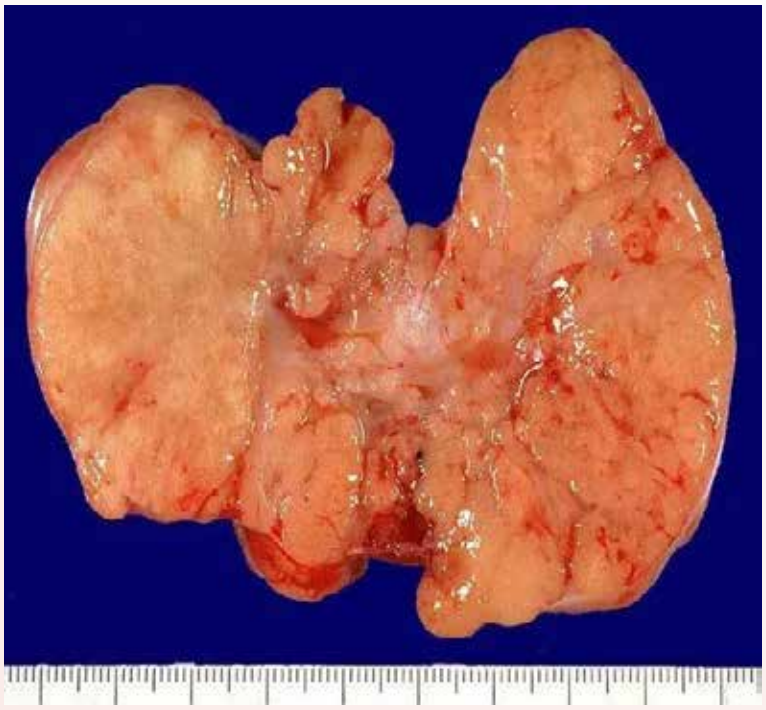
We must understand the psychological impact of cancer screening, even in the context of a non-cancer diagnosis. There are two overriding themes in the literature to consider. The first is 'over-reassurance' for patients, which may subsequently delay seeking help in the future. This is understood to be influenced by patients attributing subsequent symptoms to the benign diagnosis, fear of the distress caused by the previous 'false alarm', and concerns about wasting doctors' time. The second theme relates to 'under-support' following a non-cancer diagnosis. Patients are concerned their symptoms will not be taken seriously or will be dismissed as unimportant.

Conclusion

CA125 testing has a definitive role in everyday clinical practice because it supports clinicians in risk stratification for both gynaecological and nongynaecological conditions. It is the doctor's responsibility to use the test appropriately, while explaining its limitations to the patient. In today's healthcare arena, we should be mindful of the implications of such a test and interpret its results with caution.

Rare Krukenberg Tumor - A Case Study

Dr.P.SELVI



ABSTRACT

Krukenberg tumor is usually but not always a bilateral involvement of ovaries from metastic deposit from adenocarcinoma of stomach and rarely from other gastrointestinal (GI) and non GI organs. The route of metastasis of this rare condition is still not proven.

The Krukenberg tumor is a good example of metastatic ovarian organotropism ("seed and soil" hypothesis).The symptoms are nonspecific and usually don't develop until the tumors reach a certain size.

Krukenberg tumor is attributed to the fact that they represent an advance stage metastatic disease.

Keywords:Krukenberg tumor, metastatic gastric adenocarcinoma signet ring cell gastric carcinoma, palliative chemotherapy, surgery.

INTRODUCTION :

In the year 1896 Krukrnberg presented five cases of peculiar ovarian tumor having appearance of malignant cells as new type primary ovarian sarcomas which he named "fibrosarcoma ovarii mucocellulare (carcinomatodes)". In his thesis he proposed it as a primary tumor of ovary , but latter it was proved to be nearly always secondary to gastrointestinal (GI) tract malignancy particularly stomach.

However there have been rare and isolated cases which have been interpreted as primary tumors. Other primary GI organs responsible are colon, biliary system, jejunum and pancreas. Non GI organs like breast, uterine endometrium, thyroid, kidney and lungs also found to be primary malignancy rarely. Nearly 80% cases are bilateral

Histologically these are usually poorly differentiated intestinal type adenocarcinoma with or without signet ring cells, sometimes producing mucins. It is considered as a metastatic disease with very poor prognosis . Till date optimal treatment has not been estabilished and it is still uncertain whether surgical resection of ovarian metastases and / or the primary could help.

I am reporting a rare presentation of gastric carcinoma with ovarian metastasis with particular importance to its management decision.

The present case highlights the possibility of missing the diagnosis of metastatic disease and subjecting the patient to a surgery and also the dilemma of a surgeon in deciding the treatment for such a disease.

The current standard treatment for patients with metastatic gastric cancer is systemic chemotherapy. Recently, many studies have shown that primary debulking sugery improves overall survival.

CASE STUDY :

A 40yrs old P1L1 with regular cycle had complaints of lower abdominal pain with abdominal distension for 3 months.

She was evaluated outside USG abdomen shows enlarged right ovary of 15.2 x 12.8 cm size with increased echogenic echoes with vascularity and peripherally arranged follicles . Left adnexa shows another sized lesion of 10.1 x 7.6cm in size with internal vascularity with minimal ascites.

MRI pelvis done outside showed.

1. well defined T2 hyperintense T1 hypointense high signal on DW1 of size 10 x 11.5 x 9.5 cm noted in left adnexa , pushing the uterine anatomy.

2. A large T1 hypointense diffusion restricting lesion with few T2 hyperintense peripherally arranged cystic areas noted in right adnexa extends into abdominal cavity. Approximately measures 18 x 16 x 12.1 cm .

3.Minimal ascites seen, Reported as solid cystic lesion noted in bilateral adenexa propably ovarian origion.

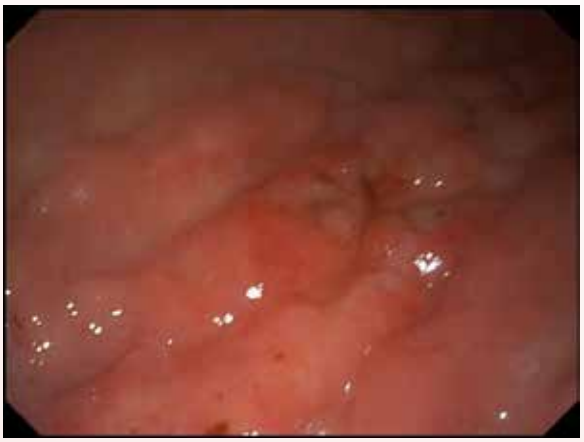
Patient came to our center with complaining of lower abdominal pain and distension for past three months.

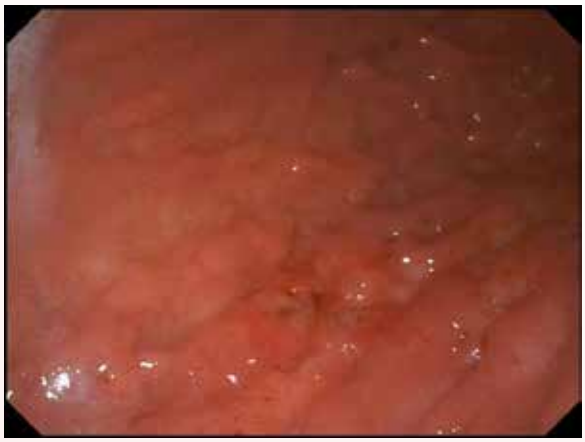
On examination patient is severely pale (Hb – 5.8gms%) not icteric. Per abdomen examination shows cystic mass felt upto the level of umbilicus with restricted mobility.

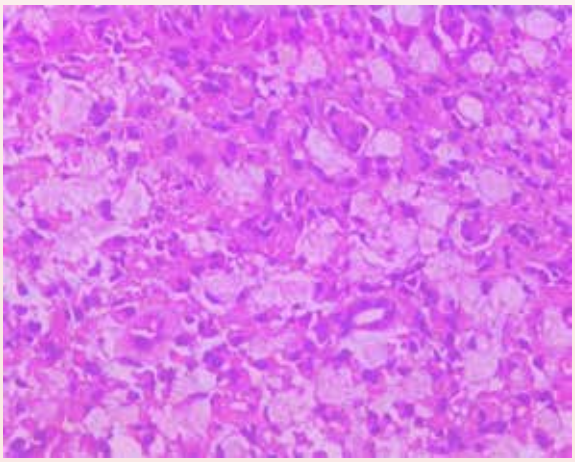
S/E – Cervix healthy, P/V – Cervix felt, exact size of uterus not made out. Cystic mass felt in all fornices.

CA 125 was 55.87 U/ml .Anemia was corrected with 3 units of blood transfusion.

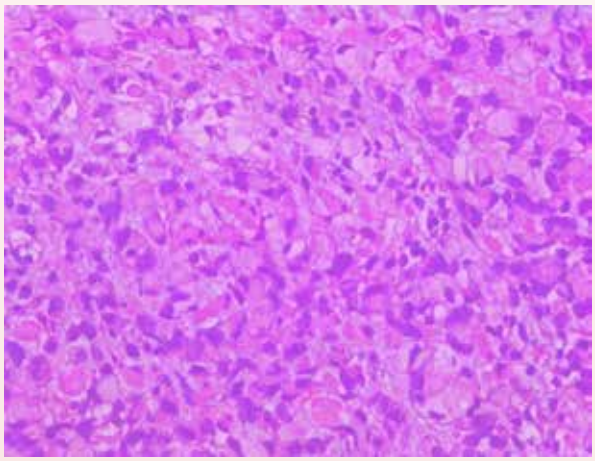
In view of bilateral ovarian mass suspecting Krukenberg's tumor, posted for upper GI endoscopy. Upper GI endoscopy showing multiple ulcerative lesion in body area suspected ulcerative growth, biopsy report came as poorly differentiated adenocarcinoma, diagnosed as Krukenberg's tumor with primary site of origin as carcinoma stomach with bilateral ovarian metastasis.





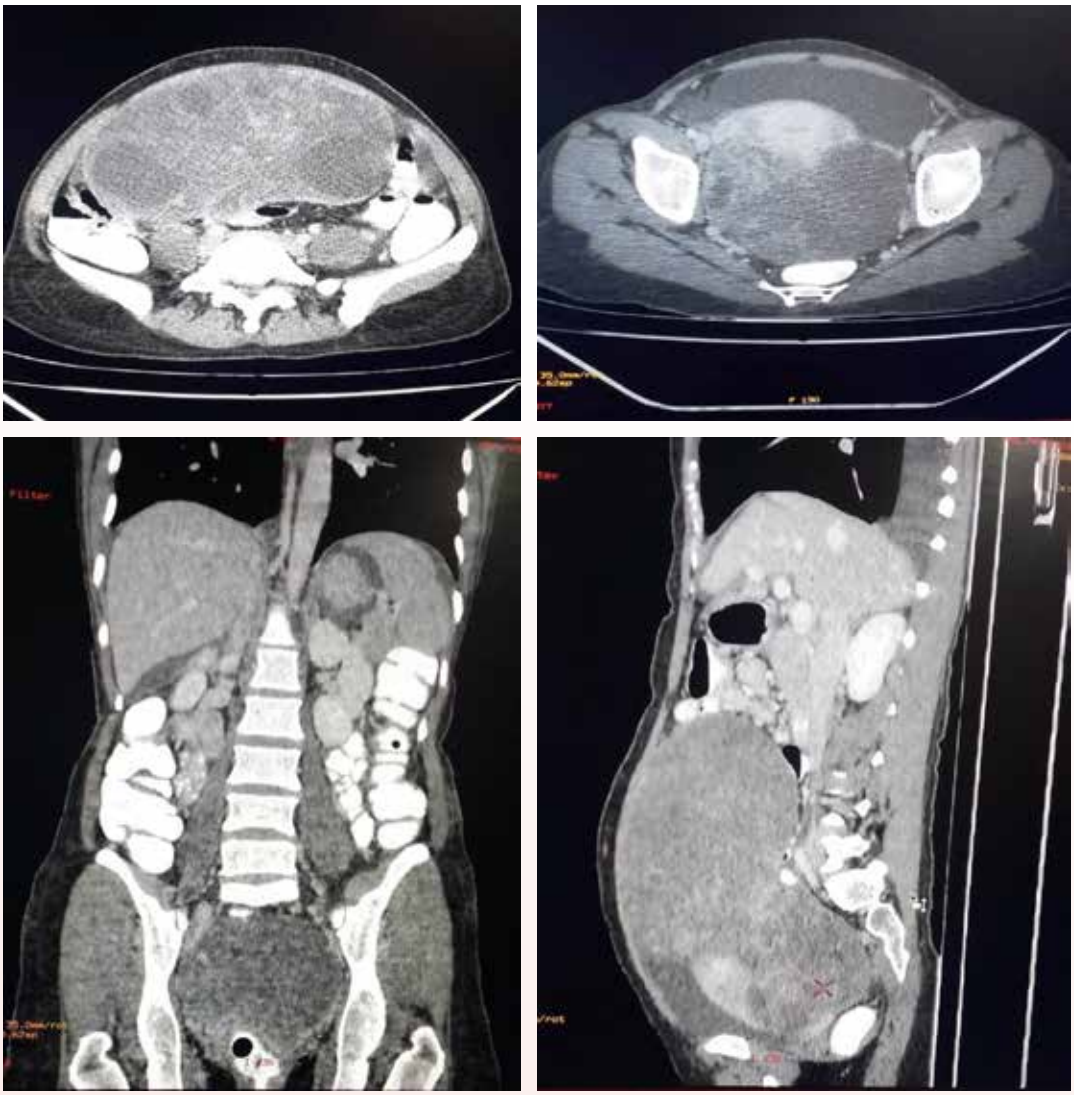


SIGNET RING CELLS WITH MUCINS



SHEETS OF SIGNET RING CELLS

CECT abdomen shows diffuse wall thickening noted involving body of stomach with maximal wall thickness of 13mm. Bilateral adenexa mass seen. Mild ascities noted, diffuse omentum / mesentric fat stranding noted, mild right pleural effusion noted.



She is diagnosed as Krukenberg's Tumor stage 4a

As per current literature treatment for stage 4a disease would be metastasectomy of bilateral ovarian mass with Total Abdominal Hysterectomy and surgical removal of primary tumor ,sub total gastrectomy followed by chemotherapy. Prognosis of the disease explained to the patient about chance of 5yrs survival is 30% with surgery and 5% with chemotherapy alone. Patient opted to go for chemotherapy.

She had completed 6 cycles of chemotherapy with Oxaliplatin , capecitabine and leucovorin 3 months before. Now she is under follow up.

DISCUSSION :

Krukenberg tumor constitute 1% to 2% of all ovarian neoplasms, usually presented in younger female with average age of 45 yrs. The route of spread of this tumor is still not well estabilished. As the tumor is usually well encapsulated and rarely shows any ovarian surface involvement, theory of peritoneal seedling from primary lesion is questioned.

Rich lymphatics draining gastric mucosa and submucosa initiating retrograde lymphatic spread to ovary is mostly accepted theory. Few authors favour theory of hematogenous spread through thoracic duct. The prognosis of a patient with Krukenberg tumor is extremely poor with average survival time between 3 and 10 months. Only 10 % of patient survive more than two years after diagnosis. Treatment of patients with Krukenberg tumor is controversial.

Recent management of Krukenberg tumors, which are metastatic tumors of the ovary, primarily involves surgery and chemotherapy with a focus on cytoreductive surgery and targeted therapies. While the prognosis remains poor due to the advanced stage at diagnosis, advancements in surgical techniques and chemotherapy regimens offer improved survival rates for some patients.

1. Krukenberg tumor is a stage 4 disease. It is further divided into 4a,4b,4c.
2. 4a – also called oligo disease, disease confined to ovary not involving other Sites.
3. 4b&4c–also called pleural disease metastasis involving other organs like peritoneal / omental mets, bone, lungs.

MANAGEMENT - SURGICAL

4a –1. Metastastectomy

1. Surgical removal of the metastatic tumor,
2. sometimes including the primary tumor if it's confined to the stomach or colon

4a –2.Systemic Chemotherapy

Administered to target cancer cells throughout the body often after surg

4b & 4c

1.Neoadjuvant chemotherapy followed by debulking and cytoreductive surger

Removing as much of the tumor as possible, even if complete removal isn't achievable. Surgical removal of the tumor, potentially combined with HIPEC ,has shown improved lifespan in some cases

2.HIPEC – Hyperthermic Intraperitoneal Chemotherapy

Heated chemotherapy drugs are delivered directly to the tumor site after surgical removal

3.SystemicChemotherapy

Administered to target cancer cells throughout the body often after surgery

CONCLUSION :

In summary, while Krukenberg tumors remain a challenging diagnosis, recent advances in surgery, chemotherapy, and targeted therapies offer some hope for improved outcomes in selected patients. Careful consideration of the primary tumor's origin, extent of metastasis, and the patient's overall health are crucial in determining the optimal management strategy, according to the City of hope.

An uncommon illness that progress rapidly is Krukenberg tumor. Prognosis of KT that can be improved with right diagnosis and treatment.

Krukenberg's tumor in young patients are extremely uncommon. They should always be considered when diagnosing bilateral solid ovarian mass in patients of any age.

In the diagnosis of bilateral ovarian tumour routine use of GI endoscopy should be considered

Pursuing your passion after retirement

Dr K.U. Bhavani

When we entere this medical profession,we never had an idea what it holds for us. We were lured by the glamour of the white coat & the stethoscope! It was very late when we came to understand that it is not a profession, but a vocation. Unless you dedicate your body, heart, mind 7 soul you cannot do justice to it. It is a relentless task master.

If you get married,it is still worse. However much you try, you can never achieve a balance between your professional life & personal life. It is just like balancing on a tight rope. Am I successful as a Doctor, Wife, Mother??? A million Dollar question. Nobody can give a honest answer!!

Another sad fact is It has no retirement?! So Many of our colleagues want to work till they can….

With All this turmoil when to think about your passions?? God himself decided to give us a break in the name of CORONA!!

Luckily I had my daughter Sarayu to take over. Considering my age, I decided to go for a complete retirement after a brief period of semiretirement. I want to give her a (or Myself) a break.

After retirement first 3 months were heavenely.Eating,chatting & sleeping. No tension about delivery LSCS or termination. Oh God!Life is so beautiful! Fourth month, I started worrying whether I am going in for depression. Time is hanging heavily in my hands.

Then my D-In-Law wanted me to paint my Grand daughters" room in an innovative way. She said " Amma I Think you can do it in a better & innovatrive way than a professional painter" At the same time My Eldest grand daughter Nethra came out with another blessing in disguise. She said " Patti I am going to learn Tanjore art. As you were very much interested in it , do you want to accompany Me," she asked.

So I decided to learn wall painting & tanjore art from the same teacher. Really it's a back breaking work. But very absorbing 7 very fulfilling. But this teacher did not know the board preparation in tanjore painting.So I went to another expert as I Wnted to learn the art from A to Z. Slowly I switched over to 3 –D Tanjore,Mural Paiiiiintiing,Indian ink painting etc., etc. one fine morning my teacher said," Madam I almost taught all the tricks I know, hereafter you can develop on your own. Spread your wings & fly!! Ofcourse I had my own students also. Sold my creations to close circle.

My other interests are cooking, Gardening, Singing & stitching.As All my family members have a sweet tooth,especially mu hubby 7 my son, I wanted to become an expert in making sweets. Now I am very adept in making mysorepak, Adhirasams (My daughter"s favourite), Kaju Pista roll, Laddu etc .,etc. Even I was successful in making Rasa malaise!!

I am trying to hone my other skills also.I am devoting more time in performing Poojas,which gives me enormous amount of pleasure 7 peace of mind. I am feeling very young because I try to learn a new thing every-day.

So my dear friends, never worry about your retirement. Therte are Umpteen No of Opportunities 7 Venues waiting for you.

Wishing you all a Happy retired life!!