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HealthNews DIGEST

JULY 2024

Dr. Sherbaz Bichu

CEO & Specialist Anaesthetist
Aster Hospitals & Clinics, UAE

On behalf of the leadership, I am delighted to congratulate all on the 23rd edition of the Healthnews Newsletter. As we continue to navigate through the rapidly evolving landscape of healthcare, it is essential that we take a moment to reflect on the incredible work that you do every day.

At Aster, we are fortunate to have a team of dedicated and talented doctors who are committed to providing the highest quality care to our patients. Your expertise, compassion, and tireless dedication are what make Aster a beacon of hope for so many in our community.

I encourage our exceptional team of Aster physicians to enthusiastically continue supporting by contributing their knowledge to ensure clinical excellence and the highest quality of patient care. Your unwavering commitment to excellence is what sets us apart and makes us a leader in the healthcare industry.

Let's maintain the momentum as we explore this fascinating learning via HealthNews Digest together.

Dr. Ramanathan V

Medical Director
Aster Hospitals & Clinics, UAE

As the Medical Director for Aster Hospitals and Clinics, I am pleased to welcome you to the 23rd edition of our newsletter and express my admiration and gratitude for the extraordinary cases our doctors have encountered and navigated with exceptional expertise and dedication. Your unwavering commitment to patient care and relentless pursuit of excellence have been remarkable.

This newsletter provides a forum for sharing your excellence stories, successes, and challenges with your colleagues and the broader healthcare community, and I encourage you to take advantage of this opportunity to showcase your work and share your knowledge and experience with others.

I am pleased that HealthNews Digest continuously receives resounding support and commitment from our medical fraternities and allied health professionals. By fostering a culture of collaboration and continuous learning, we can collectively elevate the standards of patient care.

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An Interesting Rare Case of Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP) treated successfully at Aster Hospital, Sharjah

Dr. Rajesh Chaudhary
Neurology (Specialist)

PRESENTATION

- 46 year old female
- Medical history of Herpes Zoster and Thyroid with controlled type 2 Diabetes Mellitus for 15 years and on medications
- Surgical history of 2 LSCS
- No significant family medical history
- History of insidious onset gradually progressive weakness of all four limbs 6-7 months back that started from the lower limbs and gradually progressed to the upper limbs
- Consulted other specialists with no improvement, referred to Aster for further management
- Admitted with complaints of:
  - Progressive weakness in all four limbs
  - Difficulty in walking and getting up, affecting daily activities
  - Gradually increasing tiredness and weakness
  - Muscle loss for 3-4 months

FINDINGS

Assessment:

- Afebrile
- PR: 84 bpm
- BP: 106/66 mmHg
- SpO2: 99% on RA; RR: 18/min

General Examination:

- No pallor, icterus, cyanosis, lymphadenopathy, or edema
- Warm peripheries, peripheral pulses ++

Systemic Examination:

- Airway: Normal, airway intact
- Breathing: Clear, no added sounds
- Circulation: S1S2+, no murmurs, no rub, no precordial tenderness
- GCS: 15/15
- No rashes
- RBS: 158 mg/dl

Neurological Examination:

- Fully conscious, alert
- Higher Mental Function (HMF): Normal
- Cranial Nerve: Normal
- Fundus: Normal
- Motor System:
  - Bulk - Mild wasting of muscle was present in both lower limbs proximally & distally

- Tone - Reduced in all the four limbs
- Power - Upper Limb: 4/5
  Lower Limb: 3/5

- Reflex:
  - Deep - All reflexes were absent
  - Superficial - Normal
- Sensory System: Normal
- Plantar Reflexes: Flexor Bilateral
- Cerebellar Sign: Negative
- Rhomberg Sign: Negative
- GAIT: Walk with the support of one person
- Single Breath Count: 32
- Chest Expansion: >5 cm

COURSE

- The patient was admitted and evaluated in the ICU under Neurology with the above history and clinical and radiological features.
- Primarily, on history and examination, it seemed to be CIDP, which was further confirmed by Nerve Conduction Studies (NCS) and Cerebrospinal Fluid (CSF) examinations.
- NCS showed predominant Motor Demyelinating Axonal Polyradiculoneuropathy. CSF analysis showed Albuminocytological Dissociation (ACD), which was suggestive of non-infectious root inflammation favouring CIDP.
- After confirmation of CIDP, all treatment options were explained to the patient and family. All indications, contraindications, effects, and side effects were explained to the family in detail.
- The steroids were not given because of long-standing type-2 Diabetes Mellitus, and the patient was planned for Intravenous Immunoglobulin (IVIG). It was given for 5 days regularly under monitoring in the ICU.
- On day 5 of IVIG, the patient felt mild improvement in the power of both upper limbs that continued later on.

Motor Nerve Conduction Study

	Site	Lat (ms)	Amp.	NCV (m/s)
Median Left	Wrist	8.1	4.1 mV	-
	Elbow	18.3	1.1 mV	22.5
Median Right	Wrist	7.3	4.2 mV	-
	Elbow	17.0	1.8 mV	24.2
Ulnar Left	Wrist	7.4	3.1 mV	-
	B Elbow	16.3	2.1 mV	23.6
	A Elbow	19.6	1.8 mV	26.9
Ulnar Right	Wrist	6.5	3.3 mV	-
	B Elbow	14.6	3.0 mV	29.2
	A Elbow	18.4	2.4 mV	23.7
Peroneal Left	Ankle	6.7	2.9 mV	-
	Head of Fibula	16.4	2.5 mV	33.8
	Popliteal	19.2	1.9 mV	32.7
Peroneal Right	Ankle	7.2	3.7 mV	-
	Head of Fibula	16.7	1.8 mV	36.8
	Popliteal	22.2	1.1 mV	14.4
Tibial Left	Ankle	8.0	3.8 mV	-
	Popliteal	22.5	1.0 mV	29.7
Tibial Right	Ankle	6.7	3.7 mV	-
	Popliteal	19.7	0.3 mV	32.4

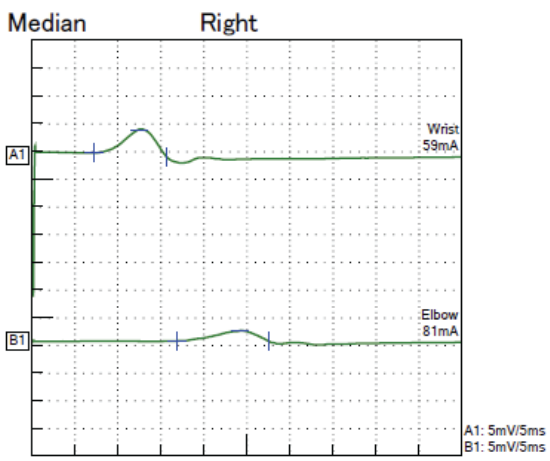
Sensory Nerve Conduction Study

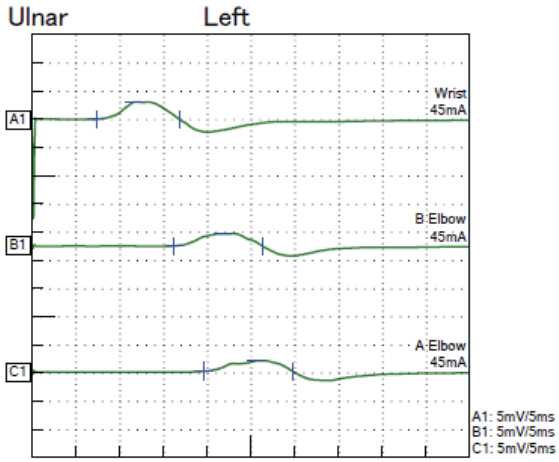
	Site	Lat (ms)	Amp.	NCV (m/s)
Median Left	Wrist	4.2	12.8 uV	33.7
Median Right	Wrist	4.2	4.6 uV	33.7
Ulnar Left	Wrist	3.9	10.1 uV	36.4
Ulnar Right	Wrist	3.7	8.0 uV	38.4
Sural Left	Sural	3.1	9.2 uV	45.9
Sural Right	Sural	3.5	17.7 uV	40.0

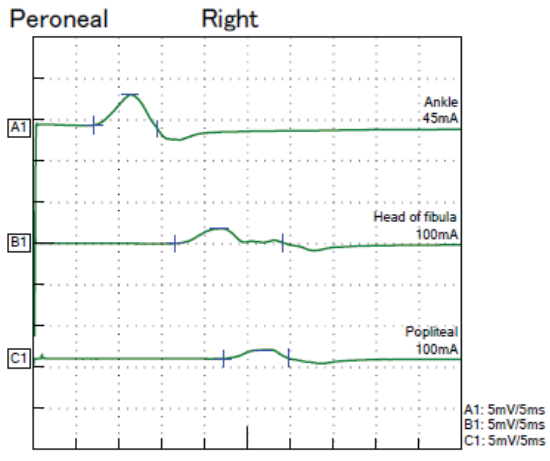
F-wave

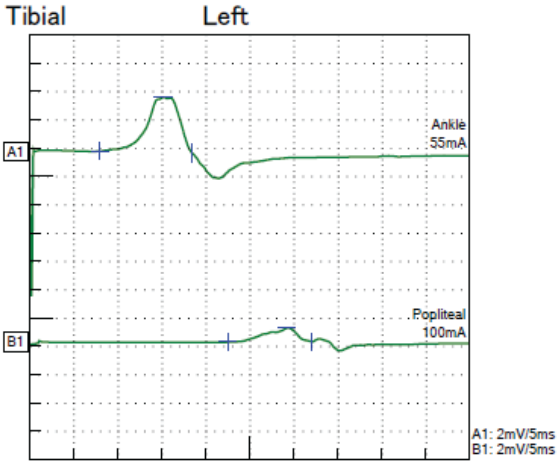
Nerve Side	F-Lat.
Median Left	-
Median Right	-
Ulnar Left	-
Ulnar Right	-
Peroneal Left	67.4 ms
Peroneal Right	87.5 ms
Tibial Left	80.4 ms
Tibial Right	78.4 ms

Motor Nerve Conduction Study

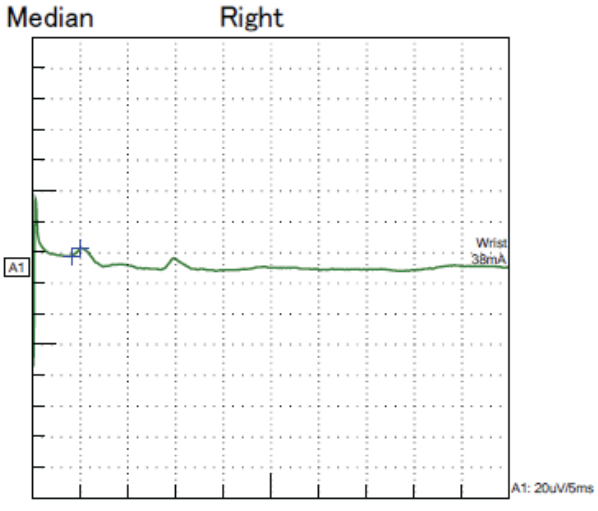


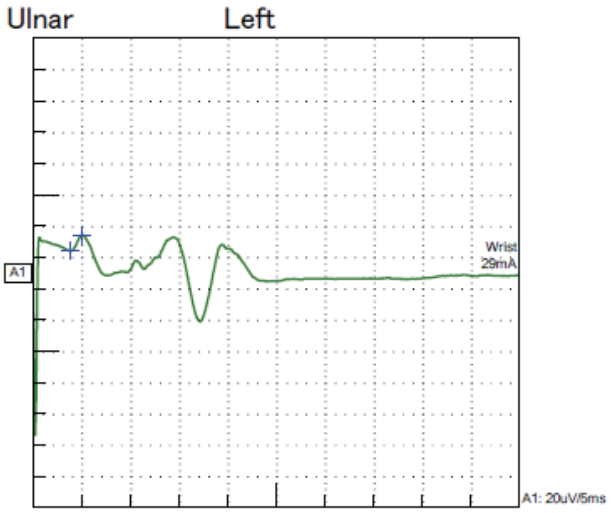






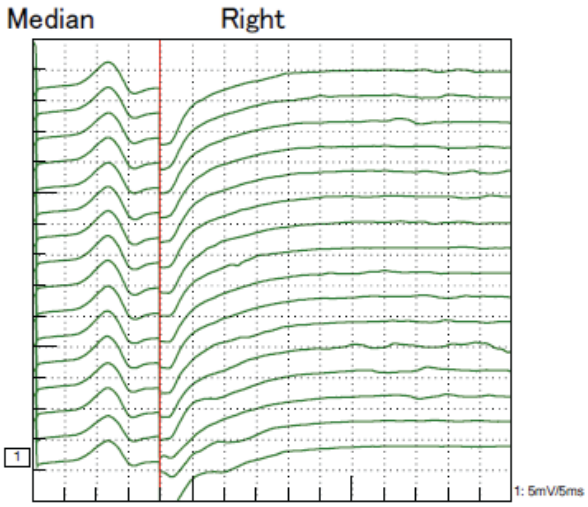
Sensory Nerve Conduction Study

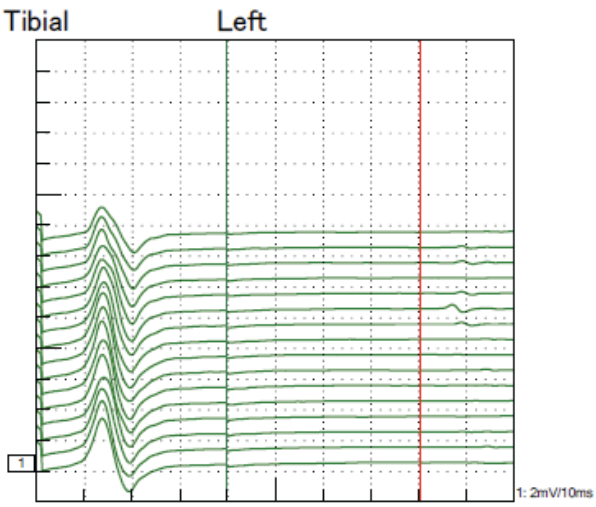




All the above graphs show prolonged Distal Latency, reduced Amplitude, and decreased Conduction Velocity

F-wave





F-wave Latency was non-recordable in the Upper Limb and prolonged in the Lower Limb

POST PROCEDURE

The patient was discharged the next day after finishing the IVIG doses. On follow-up after 10 days, the patient showed significant improvement and left with minimal weakness in both lower limbs. Upper limbs were perfectly normal. Given minor weakness in lower limbs, she was advised to rest for 7 more days and asked to return for a follow-up. On the next visit, she was found to be normal in power for all four limbs and allowed to join the job.

CURRENT STATUS

At present, the patient is hemodynamically stable. She is ambulant without support and can follow all her daily activities without any weakness or problems. She is also regular at her job.

DISCUSSION

CIDP is a very rare auto-immune disorder of the peripheral nervous system. The prevalence of CIDP may range from about 1 to 2 cases per 100,000 people to as high as 9 cases per 100,000 in some areas (1). The patient develops gradual, progressive weakness of all four limbs in this disease. Usually, weakness starts in the lower limb, such as difficulty getting up from a squatting position and walking upstairs, along with slippage of slippers with or without the patient's knowledge. Primary symptoms can include problems in walking due to lack of strength, loss of sensation, trouble using arms and legs, and a sense of tingling or pain, among others (2).

This weakness remains progressive and involves the upper limbs, with difficulty combing hair and lifting the head over-head. Due to the gradual worsening of symptoms, 1 in 3 people with CIDP will need a wheelchair without treatment (3). This disease has multiple variants, including sensory-motor, predominantly Demyelinating Polyradiculoneuropathy, which is the most common. A disabling disease that leads to loss of strength and sensation, CIDP affects males twice as often as females, and the average age of onset is 50 (4).

After the initial history, examination, and nerve conduction study, the diagnosis is usually almost confirmed, but for documentation, sometimes we need to go for CSF analysis to see A-C dissociation and nerve biopsy for final confirmation. Multiple treatment options, like Steroids, IVIG, Plasmapheresis, and other immunosuppression agents, are available. For this patient, IVIG was the first choice as she had been diabetic since 2009, but her status was under control with HbA1c at 6.9.

Most CIDP patients are well-responsive to steroids, IVIG, and other immunosuppression agents. IVIG shows rapid recovery among all. But sometimes, this weakness might recur, and then the IVIG must be administered again.

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Platelet Rich Plasma (PRP) Therapy for Hair Loss

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Aster Clinic, Al Muteena, Deira, Dubai

INTRODUCTION:

Androgenetic alopecia (AGA) is a polygenetic condition characterized by a specific hair loss pattern that affects 80% of men and 50% of women (1). AGA is governed by the male hormone testosterone, therefore hair loss is more severe in men than in women (2). Current approved medication include minoxidil and finasteride, which act on the testosterone cycle and telogen phase, however, their efficacy is limited (3). Therefore, there is a need for a more effective and faster-acting therapy (2). Platelet-rich plasma (PRP) has emerged as a novel therapy for hair loss conditions due to its regenerative properties (1,2).

PRP therapy was initially used as a hemostatic for thrombocytopenia, since then it has been used for its wound healing and tissue repair properties (1). It has also gained popularity in dermatology for scar healing, skin rejuvenation, and hypertrophic keloids (4). Recently it has expanded to hair loss treatment, in promoting hair regrowth, and graft survival (2). PRP is rich in insulin-like growth factors, epidermal growth factor, interleukin (IL)-1, cytokines and several other growth factors that stimulate the hair follicles (4).

This article discusses how PRP concentrate is obtained, its mechanisms of action, and its application in male and female hair loss.

WHAT IS PLATELET RICH PLASMA THERAPY?

PRP consists of platelet concentrate derived from whole blood, the blood sample is centrifuged to remove red blood cells leaving behind a platelet-rich plasma protein (5). This concentrate is rich in platelets, leukocytes and platelet-activating factors (6). These platelets when activated release various growth factors that stimulate cell proliferation, differentiation and regeneration (7). The efficacy of PRP therapy in tissue regeneration depends on the platelet concentration, a concentration 2-6 times higher than normal platelet count shows optimal response (6).

Process to obtain Platelet-Rich Plasma Concentrate:

There are no standardized methods to obtain PRP, general process includes collection of 10-60 ml of the patient's blood into an anticoagulant containing tube for two rounds of centrifugation (5). After centrifugation, the blood gets separated into red blood cells in the blood, leukocyte and PRP in the middle layer and platelet poor top layer (5). The top layer is discarded and the rest of the content is redissolved.

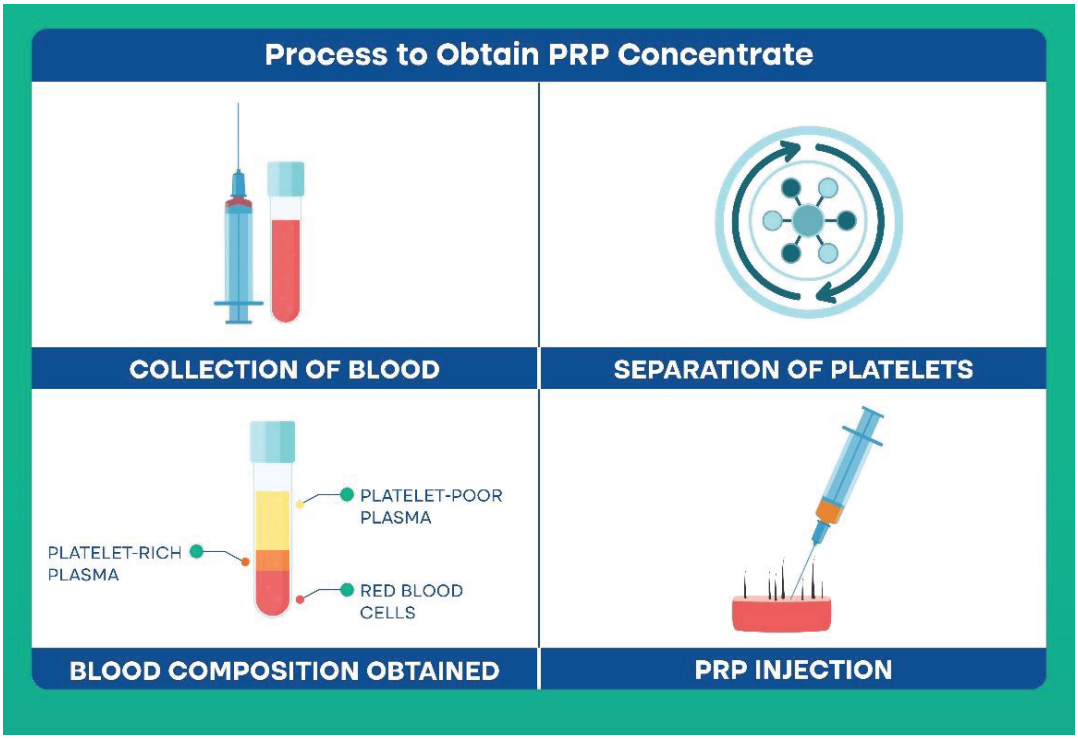

Figure 1: Steps In PRP Therapy For Hair Loss

There are commercial kits available to obtain PRP, these differ in the platelet yield and volume of PRP produced by the device (8).

PRP can be roughly classified as:

First-generation PRP which have a short half-life of few minutes to an hour (8). The second-generation concentrate has platelet-rich-fibrin that can provide a sustained release of growth factors over 7-10 days (8). Other newer forms of PRP are (8):

- PRF (advanced) - Monocyte-rich PRF
- I-PRF (injectable)
- CGF (concentrated Growth factors)
- HAS - Hyperacute serum
- T-PRF (titanium-activated)

MECHANISM OF PLATELET RICH PLASMA ON PROMOTING HAIR GROWTH:

Platelet function is not limited to only hemostasis, they also play a role in inflammation, stem cell induction, angiogenesis, and cell proliferation. Degranulated or activated platelet release platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), transforming growth factor β (TGF-β), epidermal growth factor (EGF), glial cell line-derived neurotrophic factor (GDNF), and matrix metalloproteinases 2 and 9.

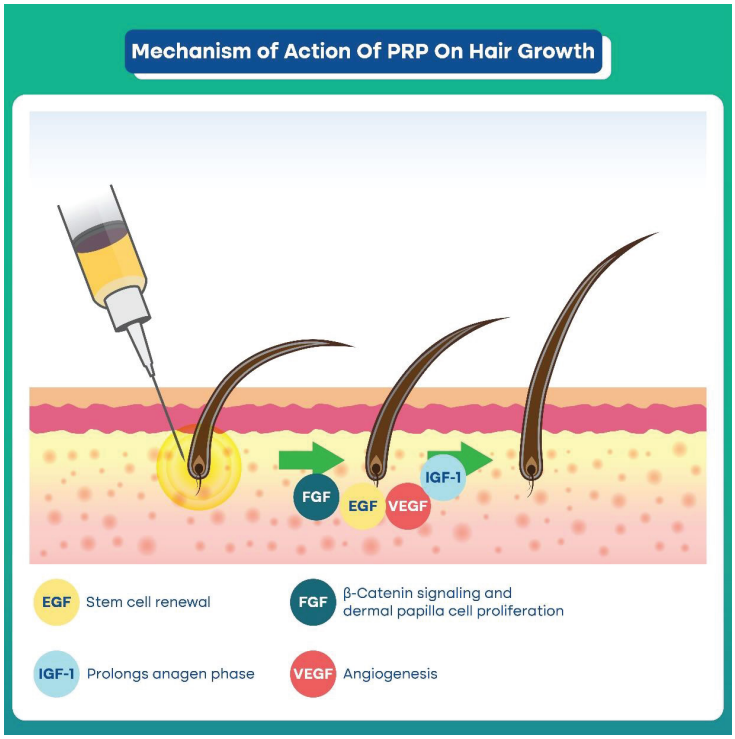

Figure 2: Stimulation of Growth Factors By PRP

The bulge of the hair follicle has receptors for various growth factors (2). These GFs bring about air growth by cell proliferation, differentiation, and angiogenesis (5).

GDNF promotes cell proliferation and prevents catagen stage, while IGF-1 prolongs the anagen phase. VEGF promotes hair growth due to angiogenetic action. PRP also activates the extracellular signal-related kinase, fibroblast growth factors, and beta-catenin which contribute to proliferation of the dermal papilla cells and antiapoptosis by the Akt signaling (5).

Thus PRP therapy stimulates hair growth via stimulation of different growth factors and prolongs hair survival by delaying the anagen phase and stopping apoptosis (5).

UTILITY OF PRP THERAPY IN HAIR LOSS CONDITIONS:

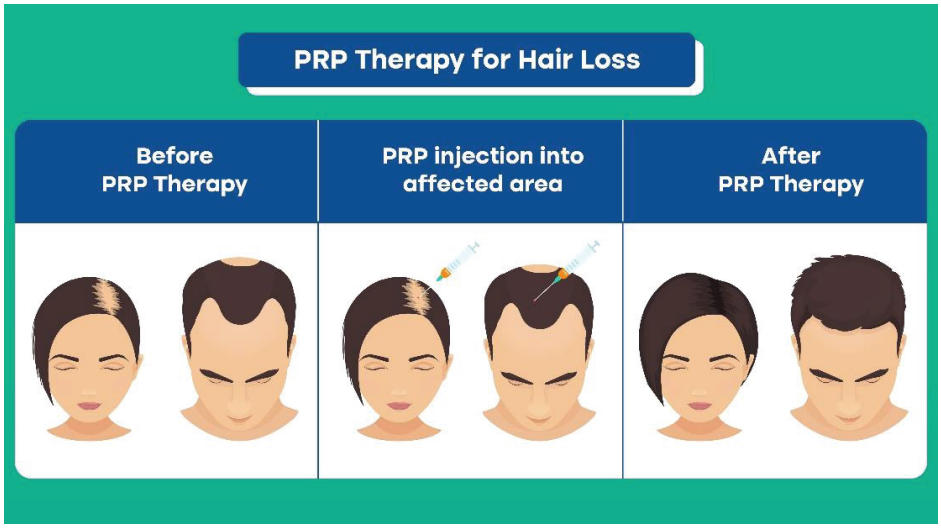

Figure 3: Effect of PRP Therapy on Hair Loss

I. ANDROGENETIC ALOPECIA

Androgenetic alopecia (AGA) is a common type of non-scarring hair loss observed in men and women (1). AGA has short anagen phase and gradual hair follicle miniaturization of the terminal hair (5). Hair loss in this condition has a specific clinical pattern in both genders, in males there is frontal recession and hair thinning on the vertex area (MPHL). While in women, there is diffuse hair thinning over the crown area (1).

The effect of PRP on 30 men with stage II-V AGA (Norwood-Hamilton Scale) and 10 women with stage I-III AGA (Ludwig Scale) was studied in a randomized, blinded clinical trial (9). The results highlighted the efficacy of PRP therapy is influenced by the frequency of its treatment (9). The patients treated with 3 monthly sessions and a booster session 3 months later had quicker results compared to patients who received only 2 sessions every 3 months (9).

A randomized placebo-controlled trial studied the effect of PRP on hair growth. The study was conducted in 32 men with stage II-V AGA (Norwood-Hamilton scale) and 20 women with stage I-III AGA (Ludwig scale) (2,10). They received subdermal injection of PRP on half of the head and the other half received normal saline (2,10). The patients underwent 3 sessions with an interval of one month (2,10). By the end of 3rd month there was improvement in hair density and by 6th month there was an increase in hair diameter, hair count, and anagen hair ratio (2,10).

Subdermal PRP therapy is efficacious in men and women with AGA and significant results are observed with frequent sessions (9).

II. FEMALE PATTERN HAIR LOSS

Female pattern hair loss or female androgenic alopecia is a common cause of hair loss in women (5). The miniaturization of hair leads to hair thinning, reduced hair density and diffuse alopecia in the frontal, central and parietal regions (5,11). This change causes a considerable psychological impact on the patient (11).

A randomized controlled trial that evaluated the efficacy of PRP versus placebo in 30 female patients showed significant increase in hair density from (+71.1 vs –26.7 hairs/cm2; $P < .01$) at week 8 to (+105.9 vs –52.4 hairs/cm2; $P < .01$) at week 24 (12). Likewise, the hair caliber also increased from (+0.0043 vs –0.0034 mm; $P < .01$) at week 8 to (+0.0053 vs –0.0060 mm; $P < .01$) at week 24 in the PRP and placebo groups respectively (12).

In another small retrospective observational study consisting of 20 female patients who showed no improvement after minoxidil therapy demonstrated good efficacy and safety of PRP in women diagnosed with AGA (13). The patients received 6 sessions of treatment with one month of interval and had good improvement of their SALT score from 27.5 ± 6.35 before treatment to 9.41 ± 3.71 after treatment (13).

Although AGA is prevalent in males, a considerable percentage of females also experience hair loss. In such cases, PRP proves to be a valuable treatment (11).

CONTRAINDICATIONS

A key attractive feature of PRP is the autologous nature of platelets, avoiding the risk of transmittable diseases and adverse reactions (5). The safety of PRP therapy is limited in patients suffering from platelet dysfunction, thrombocytopenia, hemodynamic instability, anemia, and local infection (5). Certain cancers affecting the bone and the hematolymphoid system are contraindicated to PRP therapy (5).

Use of NSAIDs in the past 48 hours, intravenous glucocorticoid in the past one month, and oral glucocorticoid in the past two weeks is also contraindicated (5).

Key Highlights

- Androgenetic alopecia affects nearly 80% of men and 50% of women and presents as a normal aging process and of pathological origin respectively (1,11).
- PRP injections can be effective in treating male pattern baldness, both in preventing hair loss and promoting new hair growth (2). PRP can also help in the stimulation of hair growth and graft survival after hair transplantation (2).
- PRP therapy can stimulate different growth factors and cell signaling cycles involved in the hair cycle causing new hair growth (8).
- This fast-acting PRP therapy is autologous in nature which omits the risk of any disease transmission and adverse reactions (2).

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Successful Treatment of Industrial Fingertip Injury with Cross Finger Flap at Aster Cedars Hospital and Clinic, Jebel Ali

Dr. Hardikkumar S Pawar
Orthopaedics (Specialist)

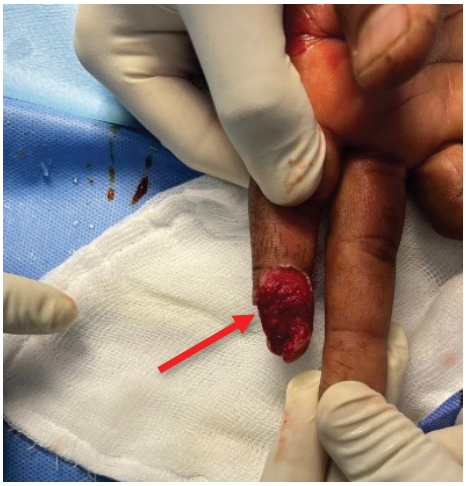
PRESENTATION

- 37 year old male
- Presented in the Emergency department with:
  - Crushing injury to the tip of the right index finger
  - Finger came in contact with a sharp machine at the workplace

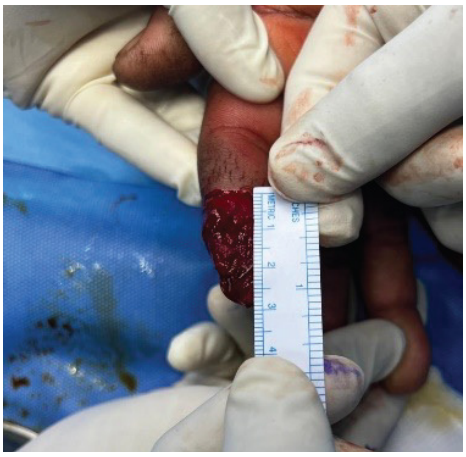
FINDINGS

During Right Hand Index Finger Examination:

- L/E: Severe crush injury at the distal end of the right index finger with soft tissue loss of around 3x2 cm
- Skin loss
- Subcutaneous tissue loss
- Pulp partially loss
- Bone not exposed
- Painful restricted ROM+
- No neurological deficit
- Active bleeding present from the wound



Pre-op Tissue Loss in Index Finger



Pre-op Evaluation of Deficient Area showing 2.5/3 cm Tissue Loss

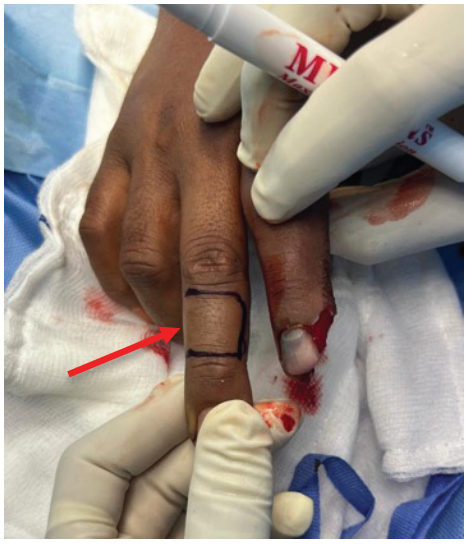
X-ray showed:

- No bony injury to the distal phalanx

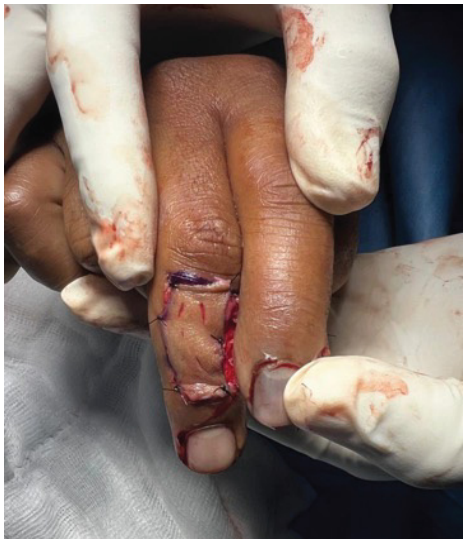
DURING PROCEDURE

- The procedure was done under a digital block and short GA.
- After debridement of the injured fingertip, the defect size was measured.
- For a pulp defect, the flap was designed on the dorsum of the middle phalanx of an adjacent digit.
  Middle finger in this case.

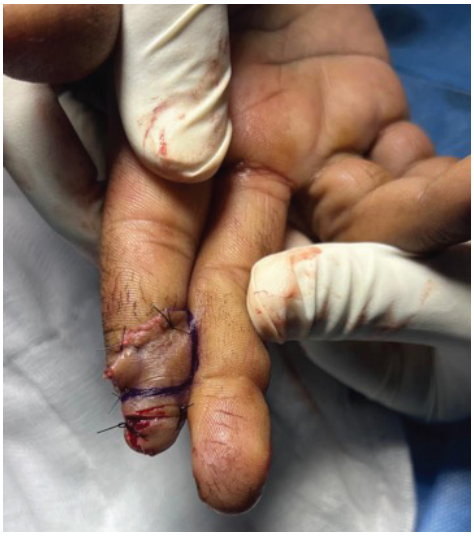
- The proximal and distal extent of the flap was incised first, and dissection was carried down to the paratenon of the extensor tendon.
- The flap was then separated from the paratenon with blunt dissection.
- A rectangular flap was harvested, leaving the edge of the flap closest to the recipient's finger intact. To ensure the paratenon of the extensor tendon is intact, a good take of the skin graft is essential.
- Flap inset was then performed. One end was anchored at the distal defect's lateral aspect to get a good fingertip contour, and about 5 mm of the excess flap was left hanging out distally.
- After the proximal part of the flap inset was done, the tip was turned down and sutured to the sterile matrix.
- A full-thickness skin graft was harvested and applied to cover the donor site defect and the exposed skin bridge.
- A tie-over dressing was placed over the skin graft recipient site.
- Dressings were applied, and two fingers were immobilized with a splint to prevent undue tension on the flap.



Flap marked from another Finger Dorsum



Donor Site covered with Full Thickness Skin Graft



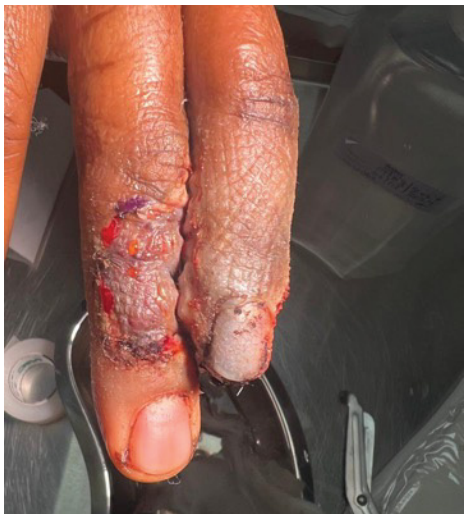
Flap Crossed and attached to the Injured Finger

The patient was discharged and reviewed for dressing after 4 days, which showed good uptake. On the 10th day, the next dressing was done, which showed excellent graft uptake.

Post-op 10th day follow-up showing Good Graft Uptake

The second stage was the Flap Division, which was done 3 weeks later. Before the flap division, it is imperative to ensure good dermal healing at the recipient site.

- The procedure was done under a digital ring block.
- After cleaning the operative site, the base of the flap was divided, ensuring adequate skin for coverage of the defect.
- The cut edge of the flap was shaped, defatted to ensure good contour, and closed primarily.
- The cut edge of the flap at the donor site was also trimmed and closed primarily.
- Dressings were placed, and early range-of-motion exercises were started after the 5th day of division.



Donor Site also showing Good Uptake

POST PROCEDURE

The patient tolerated the procedure well. Post-division, the patient was advised to wear regular dressing and follow up in the outpatient department, and range-of-motion exercises were advised with the dressing in place. At the end of 6 weeks, the finger's full function was seen with a well-healed scar, and full ROM was achieved.

Final outcome showing full Extension, full Flexion and well-healed Scar

DISCUSSION

Fingertip amputations are prevalent hand injuries in industrial areas and are also common in households/kitchens. Finger injuries are frequently encountered as they are the most exposed parts of the body, and they are in contact with devices and tools, so they are exposed to a multitude of risks. Fingertip amputation is a potentially serious injury to the hand that can lead to significant functional loss.

Different surgical options are available, such as skin grafting, stump closure, and microvascular reconstruction (1).

Treatment goals include reducing pain, optimizing the healing time, preserving local tissue, pulp sensitivity and finger length, preventing neuromas and restoring an appearance that is acceptable to the patient (2).

The cross-finger flap is a 2-staged procedure first published by Gurdin and Pangman (3). The cross-finger flap was described initially in 1950 and is one of the workhorse flaps for finger reconstruction. It can be done as described originally or as a modification in multiple scenarios of finger trauma (2,3).

The flap is taken from the dorsum of an adjacent digit, usually at the level of the middle phalanx, and is used to resurface a volar unfavourable pulp amputation. This flap does not require the patient to place the arm in an awkward position, is easier to perform, and is less time-consuming than raising an island flap (4,5).

CONCLUSION

A cross-finger flap is a simple and reliable flap among the various reconstructive options available for fingertip injuries. The main criticism of the cross-finger flap is that it is a two-stage procedure, uses an uninjured digit, and may result in stiffness of the donor finger.

In addition, it does not provide glabrous skin for coverage. Although the flap is not innervated, it has been shown to achieve good sensory recovery and results in younger patients.

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Pelvic Inflammatory Disease

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INTRODUCTION:

Pelvic inflammatory disease (PID) is a syndrome that predominantly affects sexually active young women, characterized by inflammation of the upper genital tract in response to pathogenic microorganisms (1). Common symptoms include fever, pain, vaginal discharge, and abnormal bleeding (1).

However, due to its varied symptom presentation, PID is frequently underdiagnosed (2). If left untreated, PID can lead to chronic pelvic pain, ectopic pregnancy, infertility, and infection-related abscesses (3). Treatment for PID varies based on its severity and can range from oral or parenteral medications to hospitalization in more severe cases (3).

This article elaborates on PID, its causes, risk factors, complications that may arise from PID, and its treatment regimens.

PELVIC INFLAMMATORY DISEASE:

Pelvic inflammatory disease (PID) can be defined as the inflammation of the uterine adnexa, which includes the uterus, fallopian tubes, ovaries, and the pelvis (4). The causative organisms of PID include 3 general groups of pathogens: 1) sexually transmitted infections from *Neisseria gonorrhoeae, Chlamydia trachomatis, Trichomonas vaginalis and Mycoplasma genitalium.* 2) Bacterial vaginosis-associated organisms like BVAB3, *Prevotella bivia, Atopobium vaginae, Leptotrichia/Sneathia* spp, and 3) gastrointestinal or respiratory infections like *Haemophilus influenzae, Escherichia coli, and Bacteroides* (5). While STIs are the most prevalent cause of PID, the use of intrauterine contraceptive devices, and certain abortion procedures can contribute to PID (6).

PID can manifest as tubo-ovarian abscess, oophoritis, pelvic peritonitis, ovarian carcinogenesis, endometritis, and Fitz–Hugh–Curtis syndrome (4). Patients present with sudden onset of lower abdominal pain and sometimes upper abdominal pain (1).

CAUSES AND RISK FACTORS OF PID:

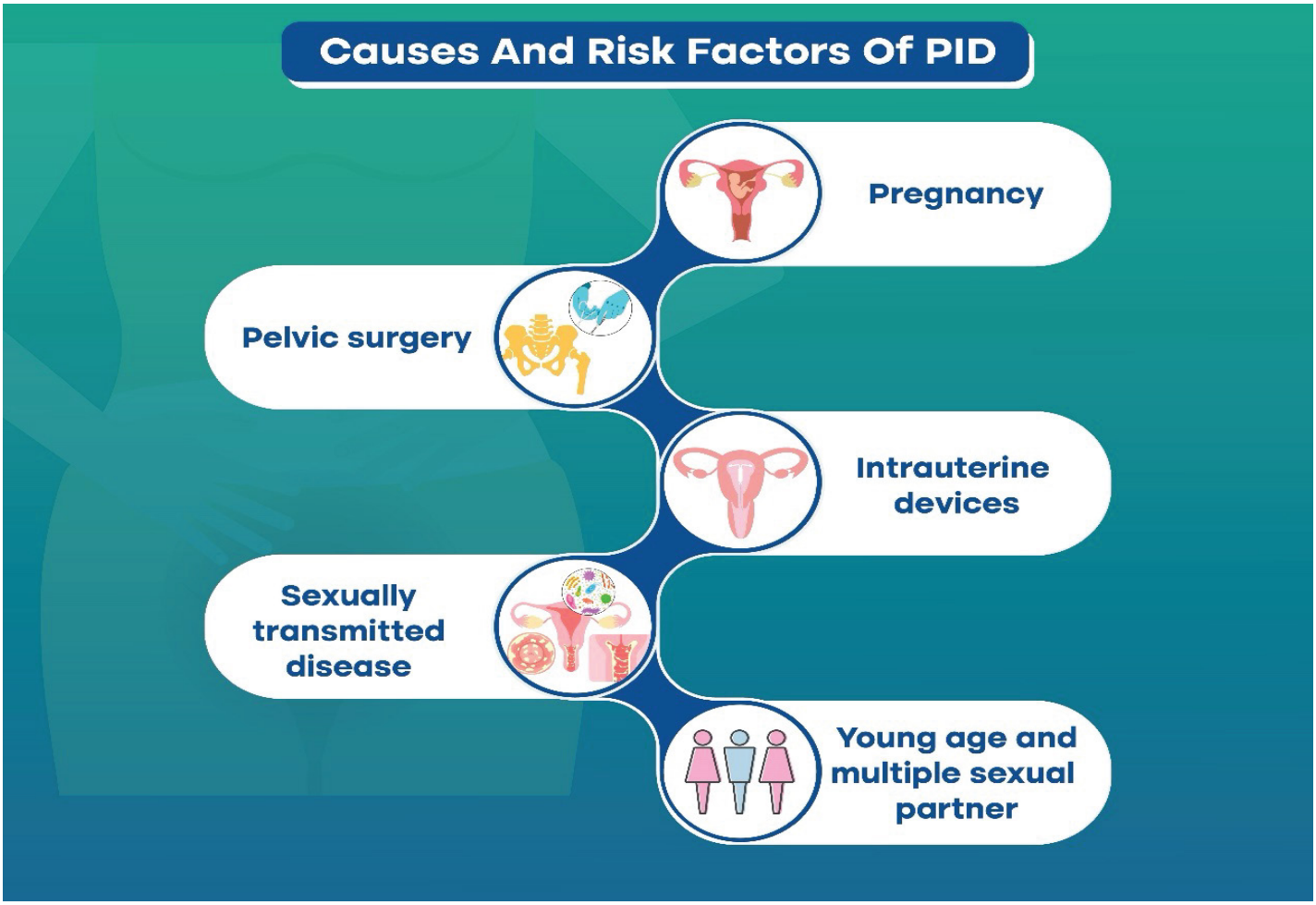

Figure 1: Causes and Risk Factors in PID

1. Pregnancy:

Pregnancy results in immunological alteration subjecting mothers to be immunocompromised (7). A disturbance in the natural microflora can lead to pathogenesis and increase the susceptibility rate of pregnant mothers to intrauterine infections (7). The microbes can cross the placental barrier leading to pelvic infections (7). It makes them vulnerable to *Toxoplasma gondii,* syphilis, varicella-zoster, parvovirus B19, Rubella, and Cytomegalovirus (7). This type of bacterial infection along with microbial dysbiosis can lead to an inflammatory cascade affecting the mother and the fetus (8).

2. Pelvic surgeries:

Gynaecological or related pelvic surgeries pose a risk in which pathogenic microorganisms from the skin or vagina can migrate from the site of operation to the cervix (9). Women may undergo hysterectomies, laparoscopic procedures, hernia repair, and other curative surgeries that expose them to bacteria (9). Long preoperative and postoperative hospital stays also increase the risk of infections (9).

Other risk factors for surgical-related infections include diabetes, obesity, comorbidities, prior radiation therapies, and advanced age (9). Intraoperative risk factors such as anemia, prolonged operation times, staple closures, and laparoscopic procedures also predispose patients to inflammatory infections (9).

3. Intrauterine devices:

Intrauterine infections are found to be highest in the month following IUD implantation (10). The cervical mucus gets disrupted during the insertion of the intrauterine device, which can introduce the bacteria into the uterine cavity (10). A missed clinical diagnosis of an STI before inserting an IUD can further aggravate PID (10). If there are any symptoms

of cervical infections, IUD insertion should be delayed, and asymptomatic patients should be screened before IUD implant to reduce the risk of PID (11). In a case-controlled study of 155 women with PID, the risk of PID was higher in women who used the Dalkon shield (82%) compared to copper IUD (30%) and the Lippes loop (28%) (12).

4. Sexually transmitted diseases:

A majority of PID cases are due to sexually transmitted infections, the spread of the bacteria to the upper genital tract causes inflammation (3). *Neisseria gonorrhoeae* or *Chlamydia trachomatis* are the most common cause of PID infection (3). In some cases, there are multiple causative bacteria, which brings about a synergistic response that worsens the severity of the disease (5).

The STI-causing bacteria induce an inflammatory response in the host (13). Although this response is intended to protect the host it can have the opposite effect by favoring the pathogen (13). The inflammatory response triggered by the bacteria leads to the activation of neutrophils and cytokines that are associated with discharge and further damage to the epithelial tract, allowing more bacterial invasion (13).

5. Young age and multiple sexual partners:

PID can occur at any age amongst sexually active women, but it is more common in those aged 16 to 19 years and in males who are 20 to 25 years of age (6). Young adults have behavioral risk factors such as sexual concurrency and biological risk factors such as cervical ectopy that can further increase the risk for PID (1).

Another risk factor is the incomplete maturity of the cervix in young females (6). The presence of the transitional epithelium increases the risk of STI, compared to adult females who have a squamous epithelial lining (6). Moreover, the ability of the immune system of young adults to handle STIs also play a role in the development of PIDs (6). The immune system may be overwhelmed in patients who suffer simultaneously from diabetes, endocrine disorders, etc (6).

Young age and having multiple sexual partners and other risky behaviors like unprotected sex with a symptomatic partner, along with a previous history of PID and STIs in either individuals can be considered as causative factors for PID (2).

COMPLICATIONS OF PID:

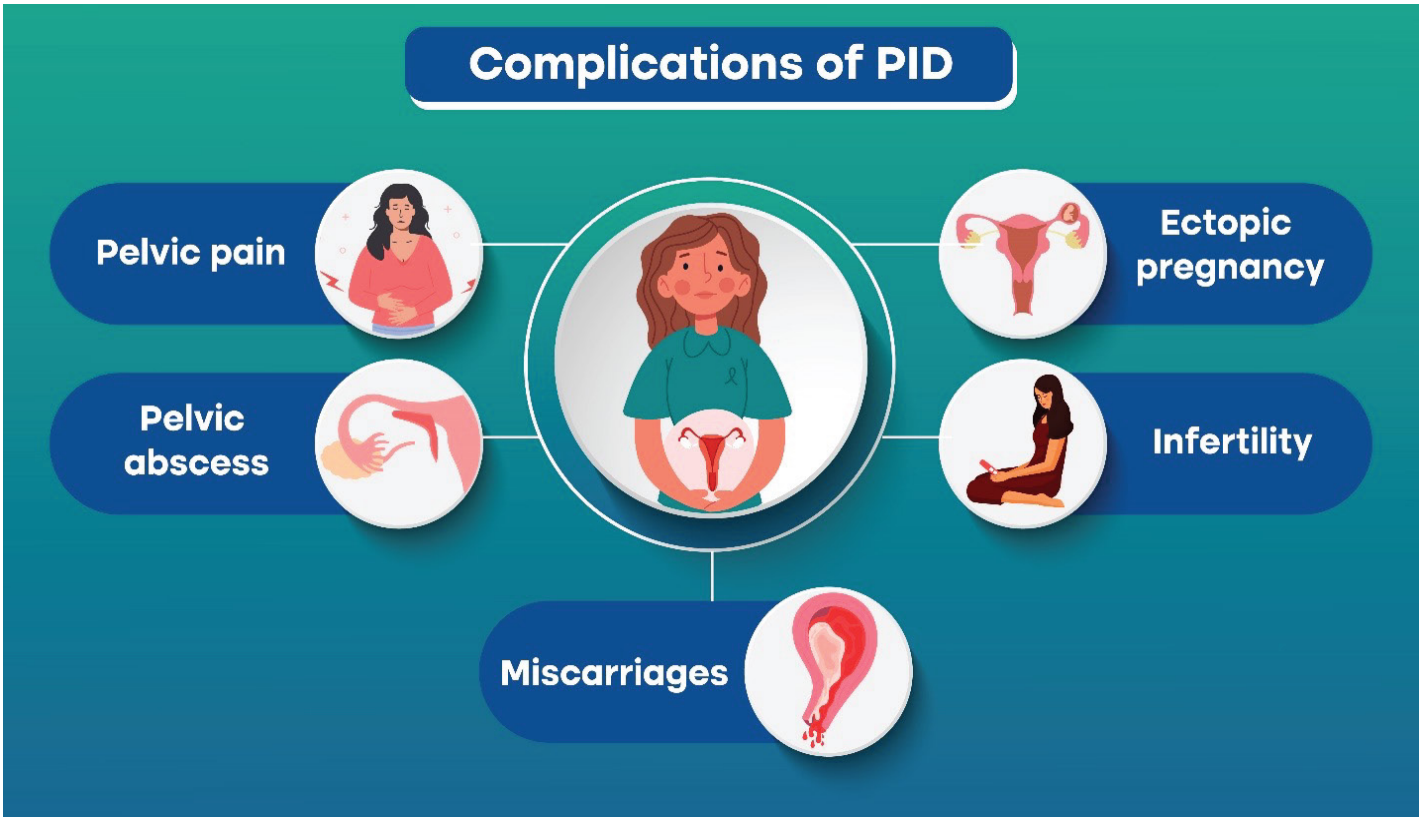

Figure 2: Complications observed in PID

1. Pelvic pain:

At least one-third of women who suffer from PID experience chronic pelvic pain (3). The pain is thought to arise from the inflammation, scarring and adhesions occurring during the infectious process (3). The onset of pain can also be considered as a symptom of PID, it may be mild and bilaterally occurring in the lower abdomen (2). Pain due to PID can cause abdominal during intercourse, uterine bleeding, frequent urination, and dysuria (2).

Upper quadrant pain occurs due to inflammation and liver adhesins, it can worsen during normal breathing and movements (2).

2. Pelvic abscess:

Pelvic abscess is one of the main complications of PID (14). The abscess can form in the fallopian tube, ovary, parametric tissues, or the pouch of Douglas (14). Pelvic and tubo-ovarian abscesses may present concurrently with low-grade fever and weight loss (15). Almost 30-35% of patients with PID observe abscesses (15).

Laparoscopy and direct visualization of the pelvic or tubo-ovarian abscess is the gold standard of diagnosis (15). However, this diagnostic procedure has certain risks and should be reserved for severely ill patients (15).

3. Ectopic pregnancy:

PID-related inflammation can cause scarring and tubal adhesions, which can prevent the implantation of the ovum in the uterus, resulting in ectopic pregnancies (14). The risk of ectopic pregnancies increases with recurrent PID, as women who have suffered from more than three instances of PID have a 50% higher risk of tubal dysfunction (1). A retrospective study demonstrated patients with PID had a 2.121 ($P = 0.003$) times higher risk of ectopic pregnancies compared to patients without PID (16).

4. Infertility:

Infertility is a long-term complication of PID (1). Recurrent inflammation and abscess result in adhesions and scarring of the fallopian tube ciliary epithelium, as well as the ovary, resulting in infertility (1). The risk of infertility rises with the severity of ovarian and tubal damage and can reach up to 30% (1).

5. Miscarriages:

PID during pregnancy can have adverse fetal consequences (17). There is a risk of maternal morbidity and mortality as well as vertical transmission of infection (17). The risk of ectopic pregnancies and infertility often culminate in miscarriages, infections from Listeria spp, Brucella spp, Herpes simplex virus, and rubella virus can increase the incidence of miscarriages (17).

TREATMENT APPROACH FOR PID:

PID should be suspected in any sexually active young woman who presents with pelvic pain, abscess, etc (3). Even if the test results and imaging studies, such as ultrasound or CT scan, show no abnormalities, empiric treatment should be started based on clinical suspicion (3).

The treatment for PID should be broad-spectrum to cover all potential causative pathogens (18). All treatment regimens should cover *Neisseria gonorrhoeae* and *Chlamydia trachomatis,* as they can cause upper genital tract infections (18).

Both parenteral and oral therapy have shown similar efficacy in mild to moderate infection (18). Hospitalization may be required for women with:

- Pregnancy

- Severe illness, nausea, or oral temperature >38.5°C (101°F)
- Incapability to follow or tolerate an outpatient oral regimen
- No improvement with oral antimicrobial therapy
- Tubo-ovarian abscess

1. Parenteral Treatment:

Parenteral doxycycline can cause pain on infusion and may be given orally instead (18). Upon clinical improvement, doxycycline 100 mg BID and metronidazole 500 mg BID orally is recommended for 14 days (18).

2. Alternative Parenteral Regimens:

There is limited data to support the use of parenteral second- or third-generation cephalosporins against anaerobic bacteria, hence metronidazole should be added (18). Women with ovarian abscess must include clindamycin (450 mg QID) or metronidazole (500 mg orally BID for 14 days along with doxycycline to provide more effective anaerobic coverage (18).

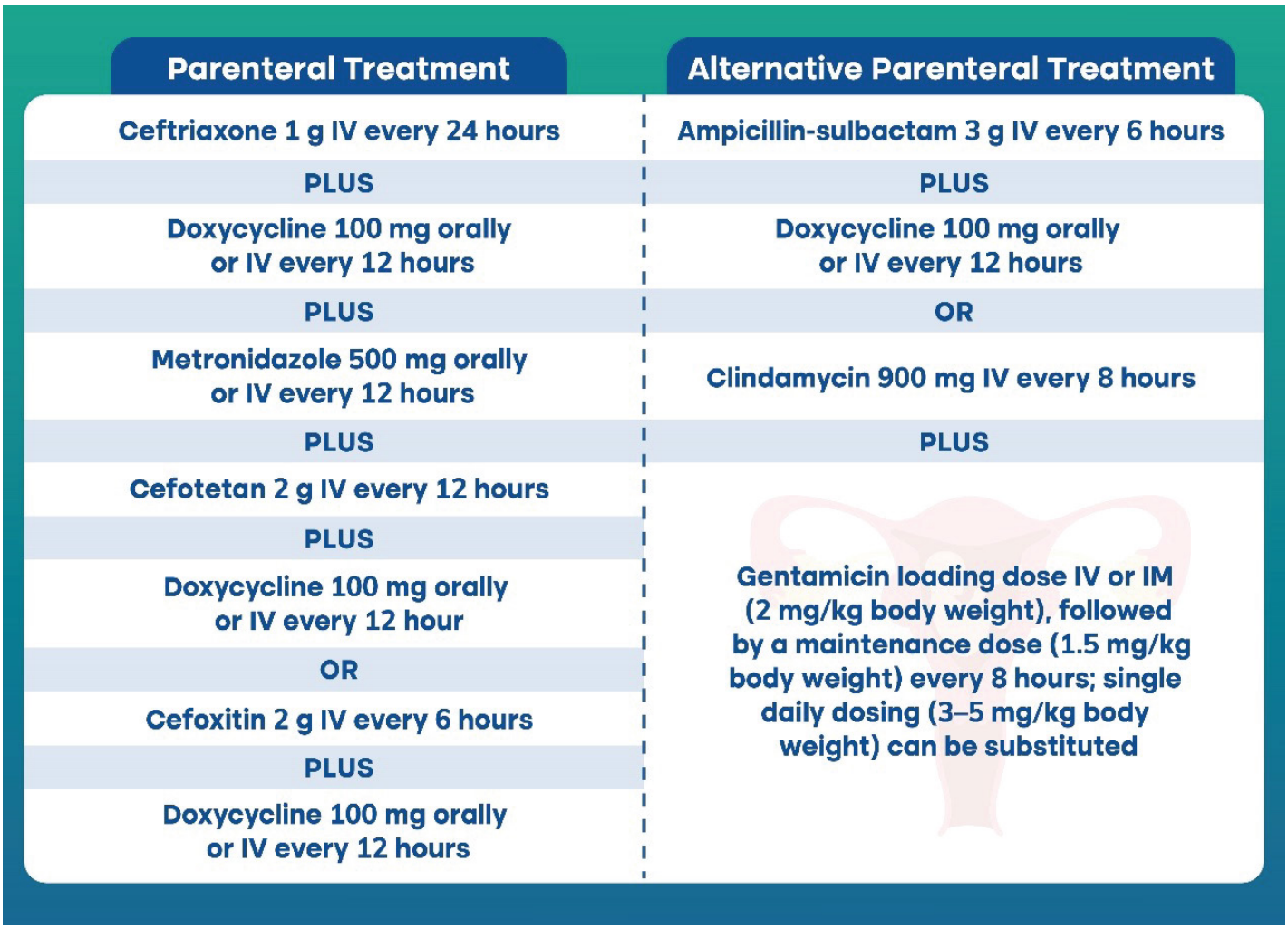

Figure 3: Parenteral treatment for PID

Intramuscular (IM) or Oral Treatment:

IM or oral therapy for mild to moderate cases shows equal efficacy as parenteral treatment (18). Metronidazole should be added to extend the coverage against anaerobic bacteria (18).

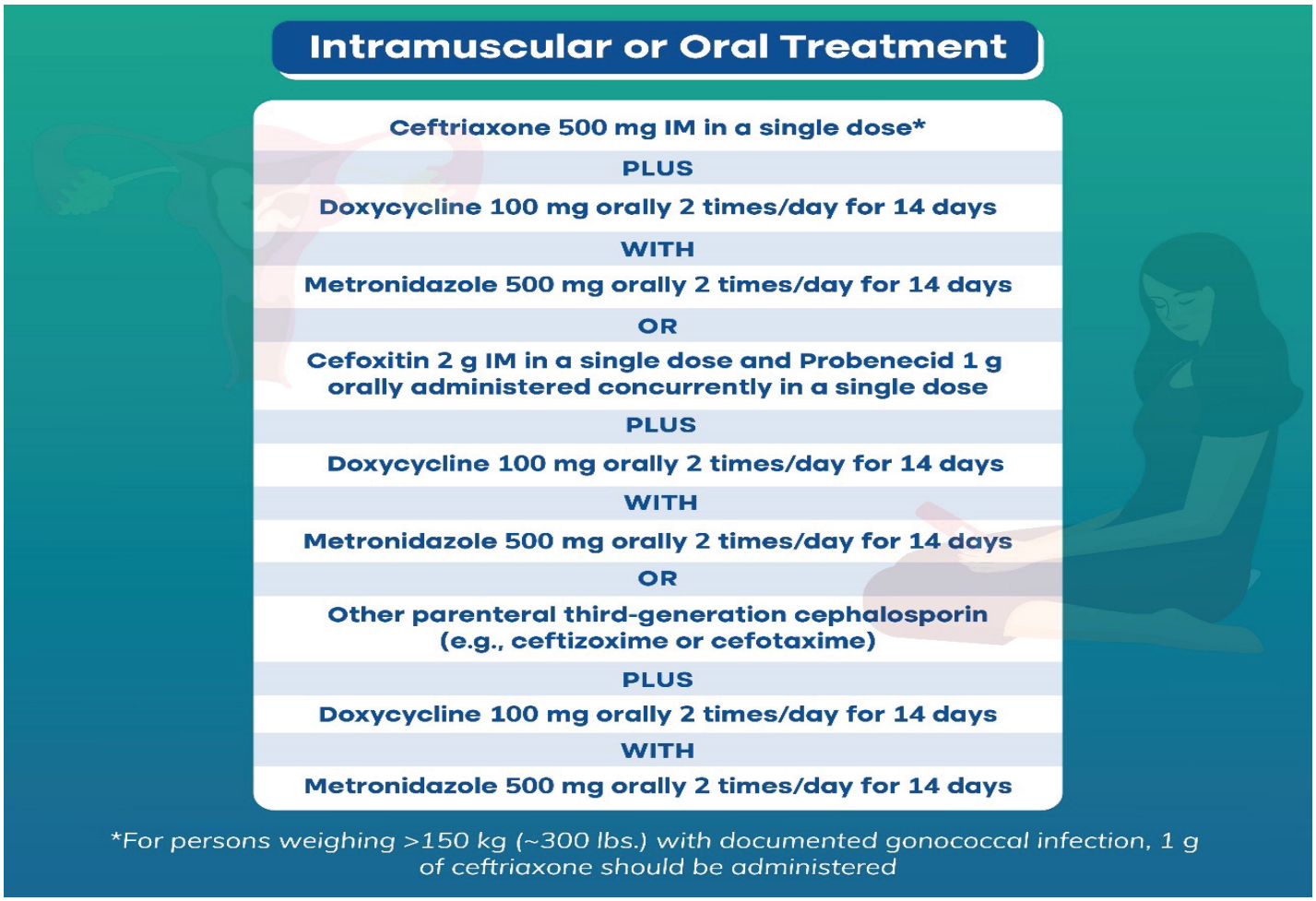

Figure 4: Oral and Intramuscular Treatment for PID

Key Highlights

- Pelvic inflammatory disease is a spectrum of disorders that ranges from pain, ovarian abscess, inflammation of the pelvis, and complications in pregnancy (18).
- Sexually transmitted infections such as Neisseria gonorrhoeae and Chlamydia trachomatis, etc. are attributed to be the cause of PID (3).
- Key risk factors for developing PID include unprotected sex, multiple sexual partners, use of intrauterine devices, and undergoing pelvic surgeries (3).
- If left untreated, PID can lead to serious complications such as chronic pelvic pain, abscess formation, increased risk of miscarriages, ectopic pregnancies, and potential infertility (3).
- Administering broad-spectrum antibiotics by parenteral or oral route is recommended depending on the severity of the infection (18).

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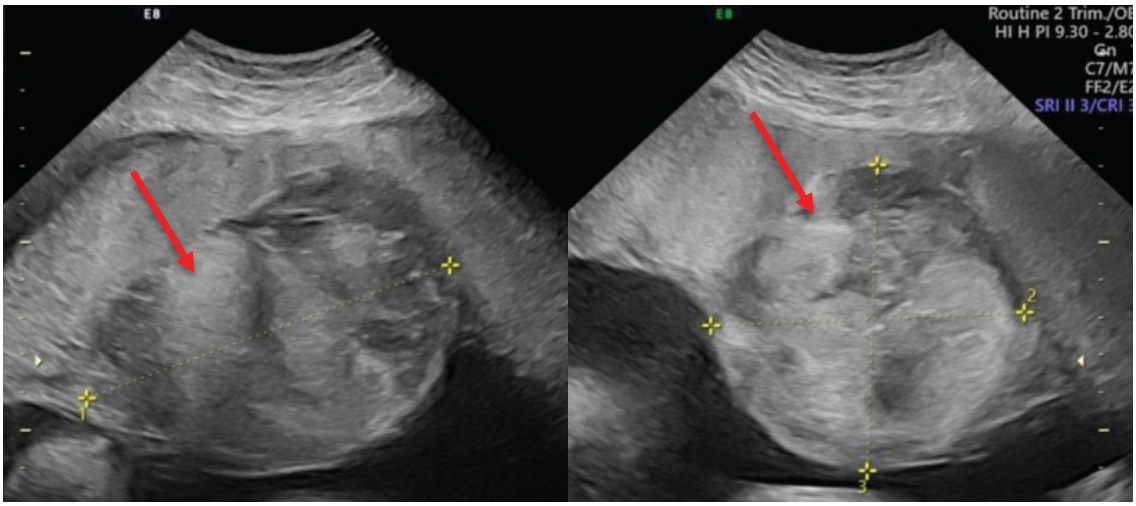
Large Placental Chorioangioma with Severe Polyhydramnios managed successfully at Aster Hospital, Al Qusais

Dr. Fathima Safa
Obstetrics & Gynaecology (Specialist)

PRESENTATION

- 35 year old female, second gravida
- Regular Antenatal check-ups (ANC)
- Surgical history of LSCS 7 years back
- Medical history of Gestational Hypertension in previous pregnancy
- Family history of Hypertension
- NT (Nuchal Translucency) and Anomaly scan were within normal limits
- Uneventful ANC till 28 weeks
- At 28 weeks, during routine ANC, a vascular lesion of 5 cm was noted in the placenta. AFI was found to be at the upper limit of normal.
- Radiologist reviewed the patient after 10 days and diagnosed the lesion as Placental Chorioangioma. By then, the lesion had grown to 8 cm with moderate polyhydramnios (AFI 33).
- The patient was sent for a Foetal Medicine opinion in a week. By then, the scan showed severe Polyhydramnios with AFI 35 cm, and the tumour had grown to10x8x8 cm.
- With high MCA-PSV (not because of anaemia, but high cardiac output at the 99th percentile), signs of high risk for cardiac failure/hydrops fetalis were observed.
- Because of these observations, the patient was admitted.



Placental Chorioangioma

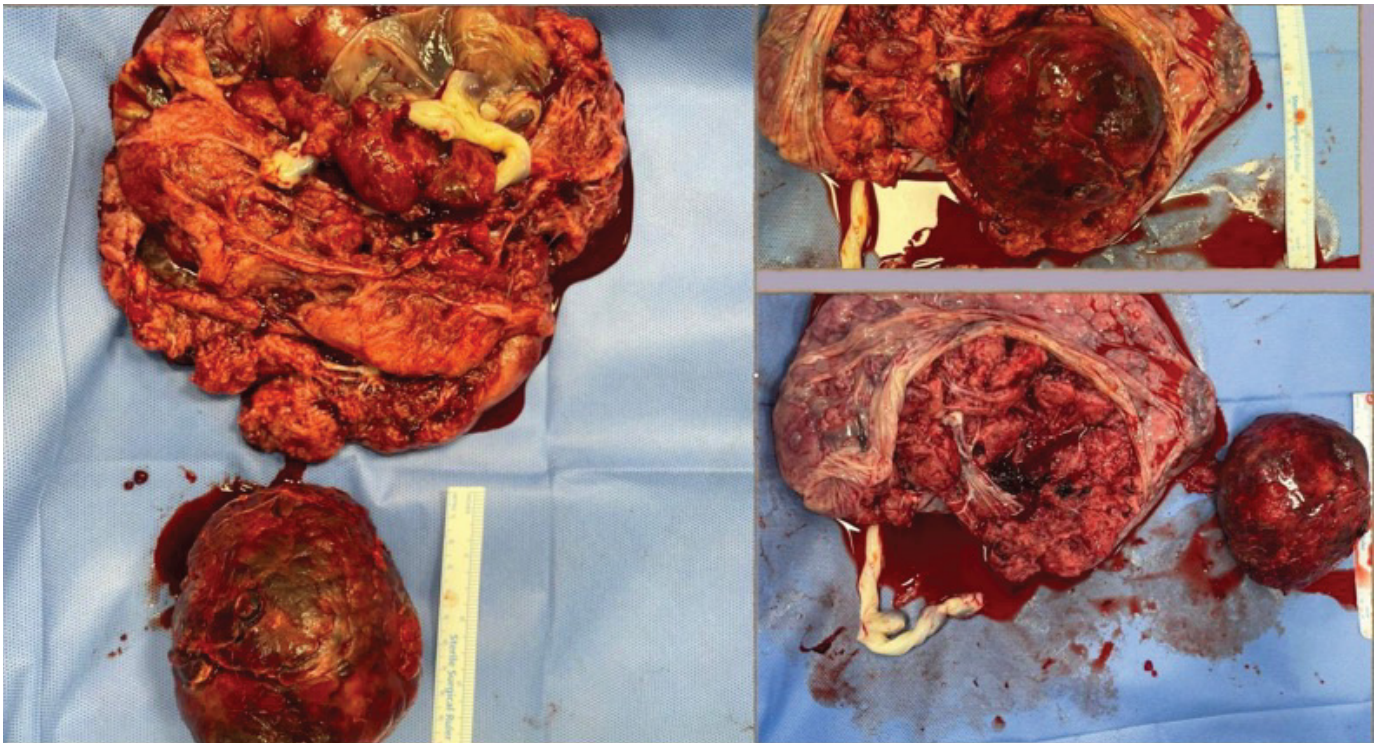
FINDINGS

During Examination:

- Afebrile - 37°C, Pulse rate - 88, BP - 120/70.
- No features of breathlessness, abdominal pain, or leaking or bleeding PV
- P/a over distended, with increased liquor
- Cephalic
- 2 doses of steroids were given 24 hours apart for lung maturity of the foetus.
- MgSO4 infusion was given for the baby's neuroprotection.
- Emergency LSCS was done under spinal anaesthesia in view of the high risk for Cardiac Failure and Hydrops Fetalis for the foetus.

DURING LSCS

- Preterm healthy female baby of 1.9 kgs; APGAR score - 1'8 5'10.
- Placenta weighed 1.2 kg with the tumour.
- Postpartum Haemorrhage (PPH) was seen and managed with Syntocinon, Methergine and intramyometrial injection of Carboprost.
- Both tumour and placenta were sent for histopathology.



Post-op image showing a large placental chorioangioma of 103x83x81 mm size

POST OPERATIVE PERIOD

- Uneventful
- Patient was discharged on post-op day 3
- Histopathology report was consistent with Chorioangioma

DISCUSSION

- Placental Chorioangioma are the most common non-trophoblastic vascular tumour of the placenta with 0.01-1.3% incidence rate.
- Etiology is not precisely known but postulated to be due to the abnormal proliferation of vessels in various stages of differentiation in fibrous stroma arising from chorionic tissue.
- Prenatal diagnosis relies on the visualization of a hypoechogenic, well-rounded, circumscribed placental mass located on the foetal surface of the placenta.
- Doppler shows peritumoral diffuse vascularization.
- Mostly small and asymptomatic. Only large tumours (>4 cm) are consistently detected prenatally, although very rare. And they result in severe foetal complications.

Complications include:

Foetal Complications	Maternal Complications
Intrauterine Growth Restriction (IUGR)	Polyhydramnios
Foetal Anaemia	Antepartum Haemorrhage (APH)
Heart Failure	Postpartum Haemorrhage (PPH)
Foetal Hydrops	Preterm Premature Rupture of Membranes (PPROM)
Intrauterine Foetal Demise (IUFD)	Preterm Delivery
	Mirror Syndrome

- The size of mass and presence of foetal hydrops/ cardiac failure are the main determinants of perinatal outcomes.
- Delivery is suggested once the complications set in.

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