Aster HOSPITALS & CLINICS
We'll Treat You Well

HealthNews DIGEST

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CEO & Specialist Anaesthetist
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The 12th edition of "HealthNews Digest" is here!

It's almost a year since we started this, and I am delighted to share the overwhelming success of these newsletters. Without the remarkable dedication and steadfast commitment of our doctors to patient care and medical excellence, this would not have been possible.

Throughout the year, you have diligently worked to present our distinguished readers with various topics, from innovative treatments to perceptive articles. Your commitment, enthusiasm, and expertise have been instrumental in the success of HealthNews.

As we embark on this edition, I wish to express my gratitude to everyone for sharing their profound knowledge. Let us continue to innovate, explore, and collaborate to create content that remains at the forefront of medical knowledge dissemination.

Dr. Ramanathan V

Medical Director
Aster Hospitals & Clinics, UAE

Welcome to our 12th edition of HealthNews Digest.

The collective effort and dedication of our exceptional team of doctors have made these newsletters nothing short of remarkable. I sincerely thank the contributors for making these newsletters a rousing success.

Your dedication to patient care, compassion, and resilience is a testament to the significant difference healthcare professionals can make in their patient's lives. The efforts you put forth to diagnose and treat complex cases and support patients during their most vulnerable times are incredibly admirable.

Together, let's continue our mission of making a positive impact in the medical world with even more remarkable accomplishments.

Thyrotoxic Periodic Paralysis (TPP)

A complex case of Thyrotoxic Periodic Paralysis (TPP) treated successfully at Aster Cedars Hospital and Clinic, Jebel Ali

Dr Ayaz Ahmed
Internal Medicine (Specialist)

PRESENTATION

- 41 year old male
- No medical history of Diabetes Mellitus / Hypertension / Dyslipidemia
- No family history of medical illness
- Admitted with:
  - Sudden onset of weakness in the lower limbs after breakfast

FINDINGS

During Examination:

- GCS 15/15, higher mental function normal
- Normal cranial nerve assessment
- Upper limbs examination came normal
- Lower limbs: Power 3/5 bilaterally, reduced tone and reflexes, normal sensations, and plantar bilateral flexor
- ECG – Presence of U wave, small P wave amplitude, bradycardia
- GRBS – 100 mg/dL
- Potassium – 1.9 mEq/L
- TSH - 0.005 - low
- T4 - 199 - high (<150)
- T3 - 5.41 - high (<3)

DIAGNOSIS

Patient was diagnosed with thyrotoxic periodic paralysis. Initially, potassium correction with betablocker given after which power in both limbs improved 5/5 within 12 hrs.

TREATMENT

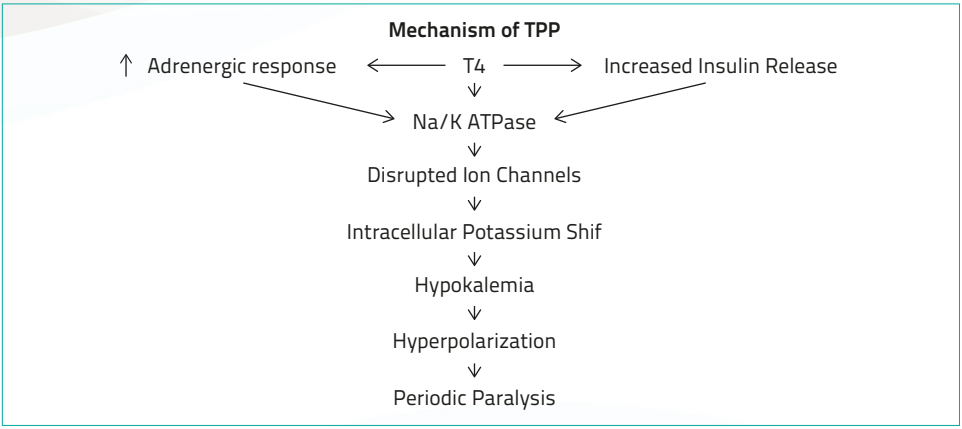
IV potassium correction, beta blocker, and NeoMercazole.

DISCUSSION

The association of periodic paralysis and thyrotoxicosis had been documented by Rosenfeld as early as 1902 (1). Thyrotoxic periodic paralysis (TPP), a disorder most commonly seen in Asian men (2), is characterized by abrupt onset of hypokalemia and paralysis (3).

It has also been reported in Western countries as a result of the migration of different ethnic populations (4). The condition primarily affects the lower extremities and is secondary to thyrotoxicosis. The underlying hyperthyroidism is often subtle, causing difficulty in early diagnosis. Factors like high-carbohydrate meal exercise, steroids, and stress can precipitate an attack of TPP.

Evidence shows the role of genetic mutations in the Kir2.6 channel in the pathogenesis of TPP. Loss of function of Kir2.6, together with increased activity of Na+/K+ ATPase, may trigger a positive feed-forward cycle of hypokalemia (5). In addition, biochemical hyperthyroidism with normal urinary potassium excretion and ECG changes are characteristic of TPP. Therefore, treatment with low-dose potassium supplements and nonselective beta-blockers should be initiated upon diagnosis, and the serum potassium level should be frequently monitored to prevent rebound hyperkalemia (6).



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Erectile Dysfunction

An Overview of Causes, Diagnosis, and Treatment of Erectile Dysfunction

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Erectile Dysfunction (ED) is characterized by the inability to attain/ maintain a consistent penile erection to engage in satisfactory sexual intercourse (1). Men of all ages and races are affected by erectile dysfunction, which has a negative psychological impact on a patient's quality of life (QoL) and that of their spouses or partners (1). Due to methodological variations among studies and patients' reservations about the condition, epidemiological data estimates the prevalence of ED to range between 14% and 48% (1).

In this article, we will be discussing the various anatomical and physiological causes of ED, its diagnosis, and available management approaches.

ANATOMY & PHYSIOLOGY OF ERECTIONS

ED was once thought to be a psychological disorder, but research indicates that more than 80% of cases may have a biological cause related to the neurological, vascular, and endocrine systems (2,3). Understanding the pathophysiology of ED and the justification for treatment alternatives will be made easier with a brief explanation of the anatomy and physiology of erections (3).

The corpora cavernosa, which run the length of the penile shaft, and the corpus spongiosum, which encircles the urethra, make up the structure of the penis (3). The penile tissue is innervated by the peripheral nervous system, which regulates blood flow during erections, controls ejaculation and detumescence, and provides sensation to the entire pelvis as well as motor function to all sphincters, the pelvic floor, and rigidity of muscles (3).

Upon sexual stimulation, parasympathetic nerves release acetylcholine, triggering a series of events that leads to increased blood flow and rapid filling of the sinusoidal system. With the expansion of sinusoids, the efferent veins are compressed causing near-complete obstruction of venous outflow and trapping of blood in corpora (3). Subsequently, activation of the sympathetic nervous system leads to detumescence and flaccidity post-ejaculation (3).

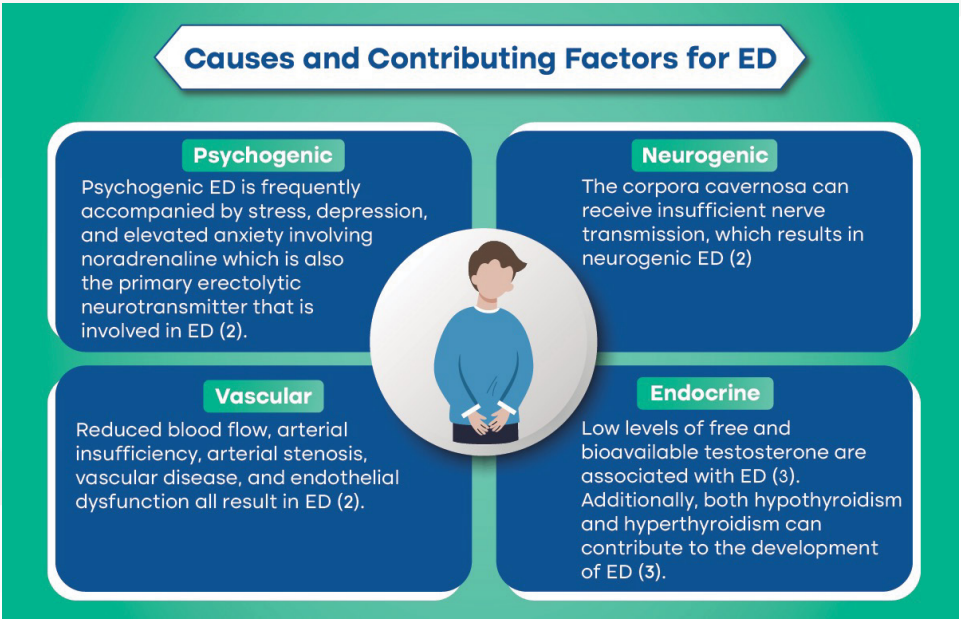

Figure 1: Causes and contributing factors of ED (2, 3)

DIAGNOSIS OF ED

The diagnosis of ED does not require extensive diagnostic procedures. It is based on the patient reporting inability to attain or maintain an erection sufficient for sexual satisfaction. It should be carefully studied and severity assesses as it may represent underlying organic dysfunction. Assessment should include a proper patient history considering the onset of ED, progression, association with a specific partner, couple problems, and nocturnal and spontaneous erections (1). It is also critical to evaluate using questionnaires such as the International Index of Erectile Function (1). The severity of impairment is determined by the IIEF score (mild, moderate, or severe), which determines the course of treatment (1). Laboratory testing is not needed for diagnosis but may help in identifying the underlying pathology and includes serum chemistries, fasting glucose, or hemoglobin A1c, complete blood count (CBC), lipid profile, and morning serum total testosterone.

Further testing like penile color Doppler, nocturnal penile erection monitoring, and neurogenic reflex testing can also be considered based on clinical scenarios (1).

MANAGING AND TREATING ED

There are several Therapeutic options available to alleviate the symptoms of ED (3). These include lifestyle modifications, oral drugs, injectable/ intraurethral vasodilator agents, and vacuum erection devices (3). These can be used alone or in combination depending on severity and patient

preference. Surgical interventions are typically reserved for patients who have contraindications to non-surgical treatments, those who do not respond to medical therapy, and those who have additional conditions such as penile fibrosis or penile vascular insufficiency

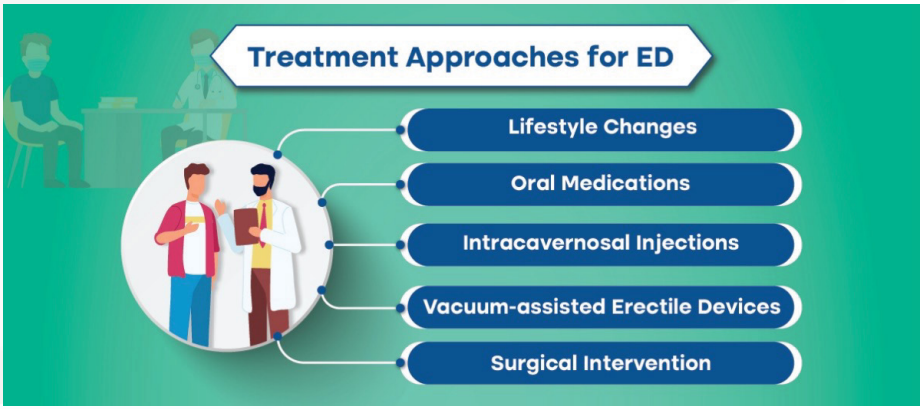

Figure 2: Treatment Approaches of ED

Lifestyle Changes: Lifestyle changes and adequate management of relevant medical comorbidities are unarguably the most beneficial and safest therapy options (4). Lack of physical activity, obesity, poor diet, and cigarette smoking have all been linked to ED (4). In the early stages of ED, lifestyle adjustments might potentially prevent progression or enhance regression (4). It should be combined with other modes of treatment.

Oral Medications: Cyclic guanosine monophosphate (cGMP) plays a crucial role in regulating erections by inducing the relaxation of smooth muscles, enabling blood to flow into the penis (4). PDE5 (phosphodiesterase type 5) gradually breaks down cGMP, allowing the penis to revert to a flaccid condition (4). PDE 5 inhibitors are the first line of treatment for ED (4). Several medicines, including sildenafil, tadalafil, vardenafil, and avanafil (4).

Intracavernosal Injections: Compared to oral ED medications, intracavernosal vasoactive drug injection (ICI) therapy is a beneficial alternative with reduced systemic side effects (5). Vasoactive drugs commonly used in ICI therapy include Prostaglandin E1 (PGE1), namely Alprostadil, which stimulates cyclic adenosine monophosphate (cAMP), papaverine as a non-selective PDE5 inhibitor, and phentolamine as an alpha-adrenergic antagonist for inhibiting smooth muscle contraction (5). Aviptadil, a synthetic vasoactive intestinal polypeptide (VIP) that enhances adenosine cyclase activity, is now available in combination with phentolamine as aviptadil/phentolamine (5).

Vacuum-assisted Erectile Devices: A vacuum-assisted erectile device (Vep-D) is a device that is positioned over the penis and pumped to create a vacuum to draw blood into the penis, resulting in engorgement and erection (4). Despite the fact that VEDs are efficient in achieving an erection in 90% of patients, long-term use diminishes because of its cumbersomeness and long-term effects on sensation (4).

Surgical Intervention: Surgical intervention is typically considered the final resort for treating ED, offering various options (5). The most common ones are inflatable or malleable penile implants/prostheses (5). Penile revascularization surgery may be performed on younger non-diabetic males (below 55 years) with isolated artery stenosis, commonly in the setting of pelvic trauma and without generalized vascular disease. However, it is not as effective or recommended as penile implants (5).

Other treatment options: Intraurethral alprostadil, also known as the Medicated Urethral System for Erection (MUSE), is a single-use pellet that contains alprostadil suspended in polyethylene glycol and is delivered via applicator (5). It is also available in the form of a topical cream (5).

LATEST ADVANCES IN THE TREATMENT OF ED

Shockwave Lithotripsy:

Extracorporeal shockwave therapy (ESWT) involves applying highly focused shock waves of very low intensity to the penis (6). New blood vessels are produced because of the healing process, increasing the blood flow to the penis and enhancing erection (6).

Platelet-rich Plasma (PRP) Injection:

Platelet-rich plasma (PRP) injection, an intriguing biotechnology technique, has found applications in various medical fields (7). In the context of erectile dysfunction (ED), its use as an intracavernosal injection (P-shot) is based on platelets' ability to stimulate angiogenesis and improve vascular health (7). Existing research has shown promising results with PRP demonstrating the potential to enhance erectile function and outcomes in men with ED, accompanied by minimal side effects (7).

Both these advancements are still considered investigational as most trials are small and cannot be considered as strong evidence.

Key Highlights

- Erectile Dysfunction (ED) is a well-documented disorder that affects the lives of many men and their partner of choice worldwide (1).
- It can be caused by psychogenic, neurogenic, vascular, or endocrine factors (2,3). Lifestyle changes are usually advised before beginning treatment (4).
- PDE5 inhibitors such as sildenafil, vardenafil, and avanafil are the major and most used ED treatments (4). Although, depending on the aetiology, various alternatives such as intracavernosal injections, VEDs, and so on are available to patients (5).
- Prostheses and penile implants are advised if other therapies fail and have demonstrated positive results, particularly in elderly individuals (5). Surgical interventions, on the other hand, are only used as a last resort (5).

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Malignant Neoplasm of Head of Pancreas

Role of EUS in Diseases of Pancreas: Successful Management of Incidentally Detected Malignant Neoplasm of Head of Pancreas at Aster Hospital, Mankhool

Dr. Amal Premchandra Upadhyay
Gastroenterology & Hepatology (Consultant)

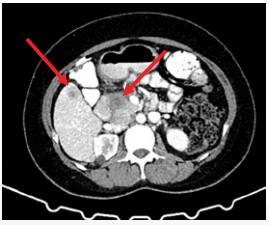
PRESENTATION

- 36 year old female
- Admitted with severe, symptomatic anaemia:
  - Complaints of dizziness and giddiness
  - Patient was brought by DCAS; initial BP in ambulance was 113/66>90/60 and fluids on flow
  - Severe iron deficiency anaemia with positive Fecal Occult Blood Test (FOBT)

RADIOLOGICAL FINDINGS

Computerized Tomography scan showed a mass in the head of the pancreas with multiple space-occupying lesions in the liver.

Two lesions in Liver suggestive of Secondaries



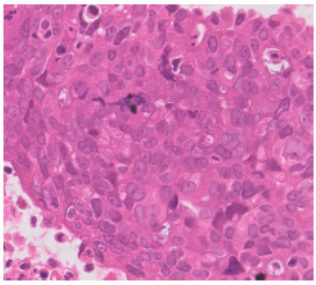
One lesion in Liver – Secondary
One lesion in Head of Pancreas – Primary

COURSE IN HOSPITAL

The patient was admitted to manage severe iron deficiency anaemia:

- There was no obvious source of blood loss in clinical history, but the faecal occult blood test was positive.
- Colonoscopy showed diverticular disease without evidence of bleeding.
- Upper GI endoscopy showed a large ulcer with bleeding. Hemospray was applied.
- Incidentally, the ultrasound of abdomen showed lesions in the liver which required further evaluation.

- Computerized Tomography scan showed a mass lesion in the head of the pancreas with multiple lesions in the liver suggestive of secondaries.
- It was probable that the duodenum ulcer was infiltrated by a mass lesion in the pancreatic head. And it proved challenging to biopsy due to its location; biopsy yielded insufficient tissue.
- CA 19-9 was markedly elevated, indicating Probable Pancreatic Adenocarcinoma, but tissue diagnosis was necessary to proceed further.
- EUS was performed, and biopsy could be obtained from the lesion in the liver as well as from the head of the pancreas.



EUS-guided Biopsy from Pancreatic Head Mass Lesion and Liver Metastasis showing Adenocarcinoma

DISCUSSION

Endoscopic Ultrasound (EUS) allows detailed examination of juxta-gastric structures, especially pancreas and certain liver segments. It also allows for detailed examination of posterior mediastinal structures close to the esophagus. It is considered more sensitive and specific than CT/MRI in certain situations.

EUS also allows for directed biopsy/tissue acquisition in real time with high levels of accuracy and a proven track record of safety.

This case highlights difficulties in obtaining tissue from pancreas through conventional means:

- Pancreas is located deep in abdomen and radiologically guided biopsy is technically difficult.
- Even though there was an infiltrating ulcer in duodenum, biopsy could not be obtained due to its difficult location.
- EUS was successful in getting the biopsy and establishing a diagnosis.

EUS is also more sensitive in detecting small liver secondaries in certain liver segments (seg 1,2,3,4), which may be missed on CT or MRI. This can sometimes alter cancer staging and make major changes in patient management.

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Uterine Fibroid Embolization

Management of Uterine Fibroids by Uterine Fibroid Embolization Procedure

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Aster Hospital, Mankhool & Al Qusais, Dubai

Uterine Fibroids (Leiomyomas) are the most common benign tumors of the female genital tract in women of reproductive age (1). Their occurrence increases with age, and they regress with menopause (1). Fibroids can be asymptomatic or cause numerous severe conditions such as disruption of surrounding pelvic structures, dysmenorrhea with or without abnormal uterine bleeding, and fertility problems (1).

The standard treatment for uterine fibroids involves surgical myomectomy and hysterectomy which leads to adequate symptom control (1). Myomectomy is sometimes associated with surgical removal of the uterus, and the fibroids grow back several years after the surgery, with 10 to 30% of the cases requiring a second surgery (2). In patients with symptomatic fibroids, uterine fibroid embolization (UFE) has proven to be an effective minimally invasive treatment alternative to surgery (1). Since the introduction of this procedure, strong evidence suggests the better safety and effectiveness of UFE in comparison to surgery (3). UFE has several advantages, including minimal blood loss, the preservation of the uterus, the use of conscious sedation rather than general anaesthesia, and a shorter recovery time (1).

HOW IS UFE PERFORMED?

- Typically, this procedure is performed by an Interventional Radiologist (IR).
- Before proceeding with UFE, a review of recent fibroid imaging is extremely important. MRI as compared to Ultrasound Sonography (USG) is advantageous as it evaluates fibroid location and the presence of ovarian artery (OA) supply to the fibroid, by enhancing the fibroid characteristics for better visualization (3).
- It also assists in determining other uterine pathologies which may imitate fibroid symptoms such as adenomyosis (3).
- The procedure is performed under local anesthesia with lidocaine 1% (1). This interventional radiology treatment is generally easier for the patient because it is minimally invasive, less painful, and is associated with shorter hospital stays (4). UFE is either done by femoral access or trans-radial access (5).

INDICATIONS FOR UFE

The most common indications for UFE include (3):

- Heavy or prolonged menstrual bleeding (menorrhagia)
- Severe menstrual cramping (dysmenorrhea)
- Pelvic pressure, excessive bloating, discomfort, fullness, or bothersome abdominal wall distortion
- Pain during intercourse (dyspareunia), pelvic pain, and urinary urgency, frequency, nocturia, or retention

LONG-TERM OUTCOMES OF UFE

- The long-term outcome of the procedure is measured by symptom status and re-intervention, including hysterectomy, myomectomy, dilatation and curettage, endometrial ablation, hysteroscopic resection of fibroids or repeat UFE (4). Failure is defined when a patient requires major re-intervention, definitive myomectomy, including hysterectomy, or repeat UFE for any reason, or experienced no improvement in symptoms after embolization (4). Around 80-90% of the patients have long-term symptom control and hysterectomy rates between 1-1.5% (7).
- Women without infertility factors who underwent UFE have a substantial rate of subsequent fertility (3).
- UFE gives the best results for the treatment of small fibroids, however, it is also safe and effective for fibroids larger than 10 cm (1).

CLINICAL SUCCESS AND COMPLICATIONS:

- The expected results after a UFE include 50 to 60% fibroid size reduction, 40 to 50% uterine size reduction, 88 to 92% reduction of majority symptoms, greater than 90% elimination of abnormal uterine bleeding, and 80 to 90% patient satisfaction (3).
- Complications after UFE are further defined as minor or major events within each category with major events resulting in an unplanned increased level of care or extended hospitalization (6). Overall, major complications generally occur in fewer than 4% of patients and minor complications occur in fewer than 23% (6).

Minor complications of UFE (6):

- Immediate complications may relate to infection and pain management.
- Small hematomas and anteromedial thigh pain.

Major complications of UFE (6):

- Uterine infections, delayed superinfection of necrotic, dominant fibroids.
- Uterine Necrosis (UN).
- Expulsion or discharge of small tissue fragments.

Key Highlights

UFE is a safe, minimally invasive alternative to surgical myomectomy, resulting in fewer and less severe adverse events, shorter hospital stays, and similar rates of technical and clinical success (6).

- Recently, advances in methods with the addition of novel forms of periprocedural patient care have resulted in enhanced postprocedural pain management (3).
- Awareness of the known complications of UFE may allow quick diagnosis and effective therapeutic responses to complications when they occur (6).

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Bullous Pemphigoid

A Rare Skin Condition of Bullous Pemphigoid treated successfully at Aster Clinic, Discovery Gardens, Dubai

Dr. Dalton Singh Kanwar
Dermatology / Cosmetology (Specialist)

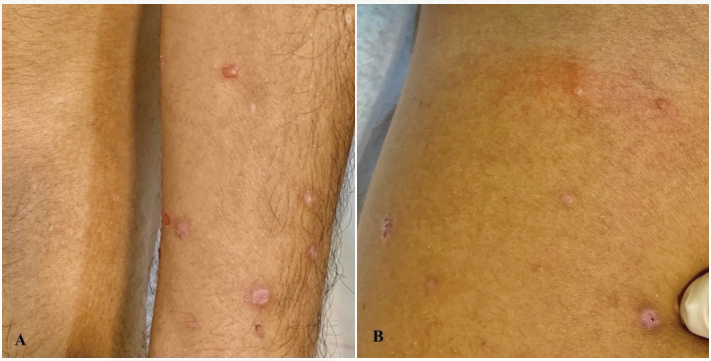
PRESENTATION

- 26 year old male
- No medical history
- No family history of medical illness
- Complaints of multiple fluid filled skin lesions over trunk, both upper and lower limbs and genitals for 2 weeks
- No associated local symptoms like itching or pain and systemic complaints

FINDINGS

During Examination:

- Vitals were normal
- Multiple vesicles and bullae were present over the abdomen and left forearm. Some lesions had erythematous macular to urticarial background, but others were on normal skin.
- Multiple dried-up erosions or superficial ulcers were present over the trunk and upper limbs. Multiple slightly violaceous to hypopigmented mildly elevated papules were present over both upper limbs and genitals.
- Multiple healed hypopigmented to pink appearing macules and some skin colored papules were present over both upper limbs.
- In addition, 3 erythematous ill-defined mildly elevated urticarial small plaques were present over the back.
- Multiple vesicles and one bulla, multiple superficial ulcers with yellow slough and red haemorrhagic crust, healing hypopigmented macules and mildly elevated papules with erosions were present over prepuce and glans.
- Oral, eye, pharyngeal, and nasal mucosa were normal.



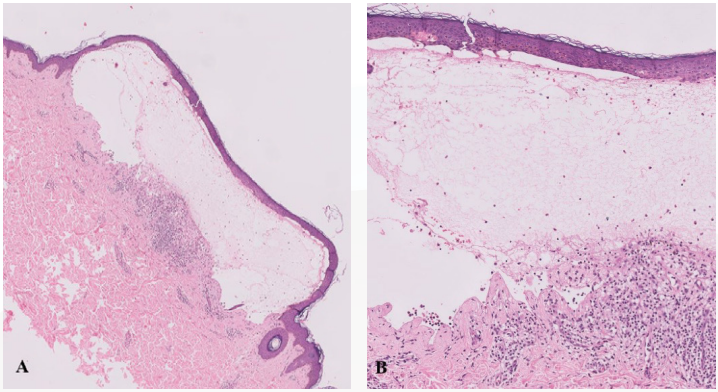
A. Vesicle with normal background skin.

B. Vesicle on urticarial erythematous background.

Both images shows skin lesions in different stages of evolution. Skin lesions are healing without medication with post inflammatory hypopigmentation, which over time are gaining normal skin colour. There are no scars or milia formation at the headed sites

WORK-UP

- Biopsy was performed for H & E histopathological examination and direct immunofluorescence.
- H&E stain showed sub-epidermal blister with scattered plasma cells, eosinophils, and neutrophils within it. Superficial dermis showed a moderated degree of peri-vascular lymphocytic infiltration with scattered eosinophils. Adnexal structures, deep dermis & subcutaneous fat were unremarkable. Negative for granuloma or malignancy.



H&E stained slides

A. Sub-epidermal blister

B. Scattered eosinophils in the blister cavity. Superficial dermis showed peri-vascular lymphocytic infiltration with scattered eosinophils

Direct immunofluorescence was positive for linear deposits of IgG, and C3 complement in the dermo-epidermal junction. It was negative for IgM, IgA, and C1q deposits. N-serrated or U-serrated pattern was not reported, which could have been a clue to differentiate between Bullous pemphigoid on the one hand and Anti p200 pemphigoid, an inflammatory variant of Epidermolysis Bullosa Acquisita, Bullous SLE on the other hand.

- Clinical and laboratory findings ruled out Linear IgA disease, Cicatricial (Mucous Membrane) Pemphigoid, Classical Epidermolysis Bullosa Acquisita (mechano-bullous variant), Lichen Planus Pemphigoid, and Bullous SLE.
- Anti-p200 pemphigoid could not be ruled out with certainty on clinical rounds alone.
- In this case, all clinical and laboratory findings supported Bullous Pemphigoid. The treatment started with only oral Doxycycline and topical mometasone ointment.
- All lesions healed completely without scar or milia formation within 15 days of treatment. Later, once all lesions healed, only oral Doxycycline was used for maintenance. There was no relapse while patient was on oral Doxycycline.
- The associated neurological diseases and malignancies were checked based on clinical history and basic investigations.
- The patient's CBC, RFT, LFT, USG whole abdomen, peripheral blood film, routine urine microscopy, occult stool blood, X-ray chest PA view, and 24 hours of urine protein all came normal.
- The patient's ANA came positive, but he did not satisfy ACR/EULAR criteria for SLE.
- There was no predisposing cause like medicine, another disease, or recent vaccination.

A. Genital lesions with vesicles, ulcers with yellow slough and red crusts, healing lesion with erosion, healed lesion with post inflammatory hypopigmentation. Skin lesions can be seen in different stages of evolution.

B. All lesions healed with oral Doxycycline and topical Mometasone ointment without scar and milia formation.

A. Genital lesions with vesicles, ulcers with yellow slough and red crusts, healing lesion with erosion, healed lesion with post inflammatory hypopigmentation. Self-healing lesions presenting as violaceous to hypopigmented mildly elevated papule and macules to slightly elevated plaques. Skin lesions can be seen in different stages of evolution.

B. All lesions healed with oral Doxycycline and topical Mometasone ointment without scar and milia formation.

DISCUSSION

Bullous Pemphigoid mainly affects elderly people although younger people may also be affected and often starts with pruritus, urticarial and erythematous lesions. Later, tense blisters are characteristic both on erythematous and normal skin. Mucosal involvement only develops in a minority of patients and is not predominant.

Histopathology of lesional biopsy reveals sub-epidermal splitting. Autoantibodies, chiefly IgG, recognize two proteins of Dermo-epiderma Junction (DEJ), BP 180 in almost all patients and, in about half of them, BP 230. It typically shows a sub-epidermal blister with dense eosinophilic rich infiltrate in papillary dermis and along the DEJ, that usually also includes neutrophils, macrophages, and T lymphocytes.

By electron microscopy, the split is shown to occur within Lamina lucida. In Direct immunofluorescence of perilesional skin linear deposits of IgG and/or C3, and to lesser extents of IgA and IgE, are seen along DEJ.

On pattern analysis of DIF at higher magnification and thin sections, Bullous pemphigoid shows an 'n-serrated' pattern. Circulating autoantibodies can be detected by Indirect Immune-Fluorescence, Commercial ELISA System, Immunoblotting, & Immunoprecipitation (1).

The case presented here is due to the rarity of this condition, with an onset at a young age and the absence of associated symptom of itching.

REFERENCES

- Rook's textbook of dermatology 9th Edition.

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