VOLUME 36 | ISSUE 1 | February 2024

Sri Ramakrishna
Hospital (MultiSpeciality)

pulse

Happenings at Sri Ramakrishna...

48 Years of Excellence

4TH, FEBRUARY

WORLD Cancer DAY

Shri.D.Lakshminarayanaswamy
Managing Trustee

We stand committed in constantly raising the bar to deliver best – in-class healthcare. We recognize the vital role that technology plays in delivering superior healthcare services and endeavor to be at the forefront in procuring the best of equipment to serve better.

Sri Ramakrishna Hospital is committed to fostering a community where compassion meets action. Through support, we strive to provide essential resources, promote early detection, and champion advancements in cancer research. Together, let's illuminate the path towards a future free from the burden of this formidable disease.

This Cancer Day February 4th, 2024 we unite in a collective effort to raise awareness, inspire hope, and contribute to the ongoing fight against cancer. Together, we can make a lasting impact on the lives touched by this challenge.

Dr. S. Rajagopal
Medical Director

Sri Ramakrishna Hospital has always been a forerunner in conducting diverse academic programs alongside its clinical achievements. The emphasis on clinical club meetings, where discussing interesting cases adds an enriching dimension to the professional development of the team.

The specific focus on Oncology, Dental & Maxillofacial surgery this month demonstrates a commitment to staying updated with medical advancements and addressing a broad spectrum of healthcare needs. This proactive approach not only benefits the medical professionals involved but also enhances the overall quality of patient care.

February 4th, World Cancer Day serves as a platform to highlight the importance of early detection and ongoing research. Together, let us progress in our collective journey towards a cancer-free future.

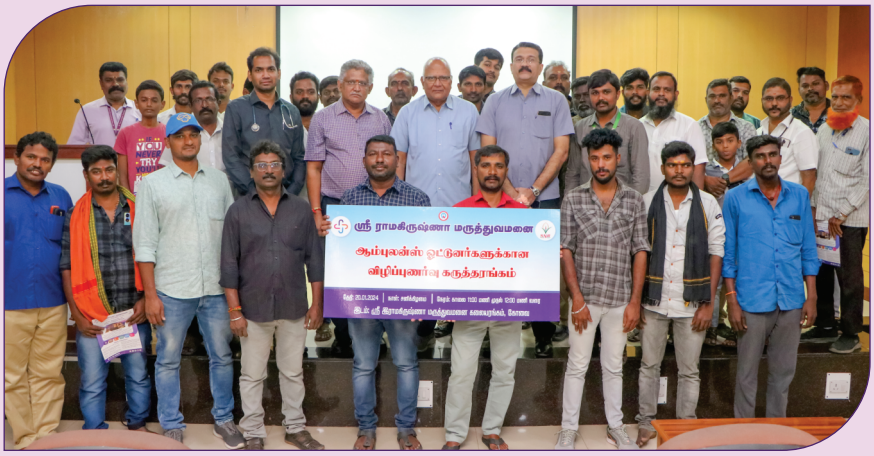
Sri Ramakrishna Hospital

ROAD SAFETY WEEK - AWARENESS CAMPAIGN - 12.01.2024

Sri Ramakrishna Hospital with Sri Ramakrishna Educational Institution's National Service Scheme took a proactive step towards promoting road safety and organized a rally on 12-01-2024 with an aim to raise awareness about the importance of adhering to road rules and adopting safe driving practices.

The Managing Trustee Shri. D. Lakshminarayanaswamy presided over and flagged off the road safety awareness rally from Government Women's Polytechnic College Signal. CEO Shri. C. V. Ramkumar, Medical Director Dr. Rajagopal, Medical Superintendent Dr. Alagappan, Emergency Consultants Dr. Manjunathan, Dr. Parthiban and Principals of Sri Ramakrishna Educational Institutions participated in the event. The NSS volunteers from Sri Ramakrishna Educational Institutions along with their NSS Coordinators, Staff Nurses from hospital enthusiastically participated in the rally, carrying vibrant awareness banners that highlighted key aspects of road safety.

AMBULANCE PILOTS AND OPERATORS AWARENESS PROGRAMME - 20.01.2024



The Ambulance drivers play a crucial role in ensuring rapid and safe arrival of emergency medical services at trauma spot. Department of Emergency Care organized an awareness programme for the ambulance operators and pilots on 20.01.2024(Saturday).

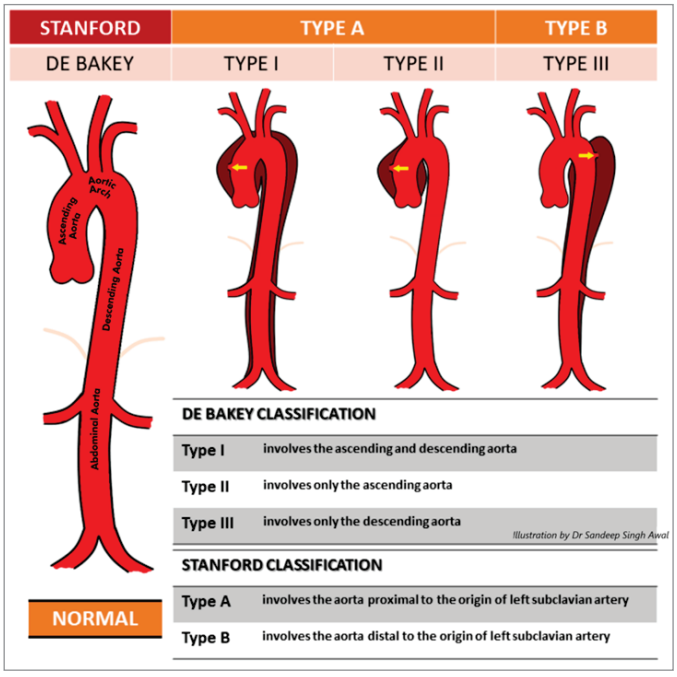
Around 70 Ambulance operators in Coimbatore attended the programme, Medical Director Dr.S.Rajagopal and Medical Superintendent Dr. Alagappan were present during the programme and briefed about the role of ambulance operators during trauma. Consultant Dr.N.Manjunathan explained the measures to be taken during "Intial Management of Mass Casualty" and ER Consultant Dr.M.Parthiban explained about the importance of "Golden Hour" after a trauma.

Eyes, Ears and Limbs deceive; Analysis reveals Truth

Case Report

62 year old male, known hypertensive and chronic smoker presented with complaints of left lower limb weakness and numbness since past 4 days. There was no complaint of any chest pain/back pain .On clinical examination, distal pulses were absent in left lower limb with cold peripheries and power of 0/5 .

After initial assesement, while preparing for USG Arterial doppler to rule out Acute Ischemic Limb, patient was found to have elevated blood pressure. Following which bilateral upper limb BP recording was done and it revealed huge variation (Right UL:180/100 mmHg, Left UL:120/60 mmHg).



Applying Aortic Dissection Detection Score algorithm, with a score of 2 patient was planned for CT aortogram. Meanwhile, bedside Echo revealed dissection flap.

CT aortogram confirmed Aortic dissection Stanford type A. Patient taken up for dissection repair surgery. Dacron graft was placed and patient discharged in stable condition.

Discussion

An Aortic dissection is a serious condition in which a tear occurs in the inner layer of body's main artery (Aorta).

Blood rushes through the tear, causing the inner and middle layers of the aorta to split(dissect).

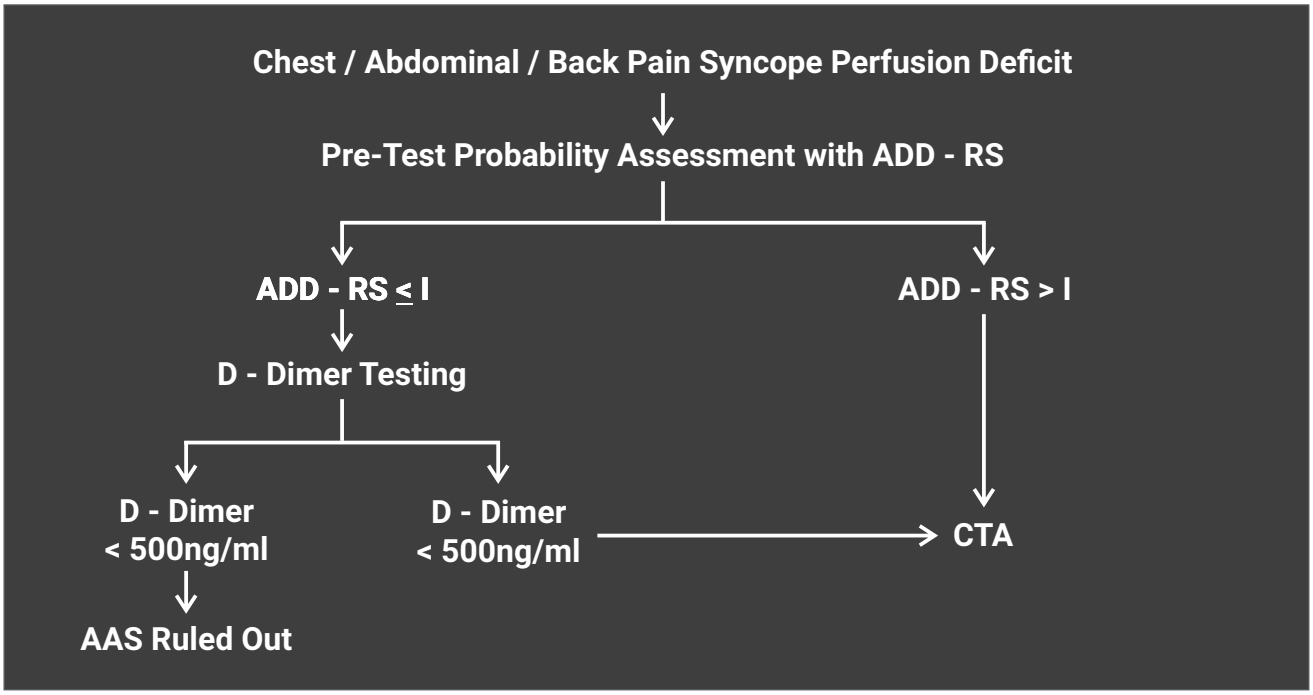
Risk Factors

- Long-standing hypertension
- Smoking and cocaine use
- Connective tissue disorder (eg, Marfans syndrome, Ehlers Danlos syndrome)
- Bicuspid aortic valve
- Coarctation of aorta
- Hereditary thoracic aneurysm
- Intracranial aneurysms
- Simple renal cysts
- Vascular inflammation (eg, GCA, Takayasu arteritis)
- Syphilis
- Decceleration trauma

Tools to detect Aortic dissection in ER

Pre-Test Probability Assessment (ADD-RS): Based on 12 risk markers classified in 3 categories Calculated by number of categories (0 – 3) where at least one risk marker was present

Aortic Dissection Detection Score		
Predisposing Conditions	**Pain Features**	**Physical Exam Findings**
Marfan Syndrome	Abrupt Onset of Pain	Pulse Deficit or SBP Differential
Family History of Aortic Disease	Severe Pain Inensity	Focal Neurological Deficit Pain
Known Aortic Valve Disease	Ripping or Tearing Pain	New Aortic Insufficiency Murmur * Pain
Recent Aortic Manipulation		Hypotension / Shock State
Known Thoracic Aortic Aneurysm		
If positive in any column, then add one point		



*Acute Aortic Syndrome

Conclusion

The above case study emphasis that not all absent pulses indicates Peripheral Vascular Disease, not every aortic dissection presents with chest pain/back pain and also the importance of dissection score algorithm which helped in narrowing to aortic dissection diagnosis.

Dr.S.MOHAMED SUGAIL
M.B.B.S., MEM.,
E.R.Consultant

Blue Hues And Toxic Clues!!!

A 30-year-old male patient was brought to the ER with alleged history of intake of unknown poisoning one hour back. On initial 10 second assessment,patient is alert, with fragmented speech, and increased work of breathing. So, patient is triaged to RED zone and connected to continuous cardiac monitoring.

	Findings	Interventions
AIRWAY	oral Secretions present, but maintainable	Suctioning done
BREATHING	RR-30 per min, Spo2-75% on RA. B/L air entry +., Rt basal Crepts +	O2 via NRB @ 15 L/min. Reassessment:pt was intubated using Inj. etomidate 20mg, inj.succinyl choline 100mgand on mechanical ventilation.
CIRCULATION	HR-100 /min, BP-130/90mmHg Peripheral pulses present, Peripheries - cyanosed.	IV line secured. IVF 0.9% NS @75 ml/hr.
DISABILITY	CBG-150 mg/dl, GCS –E4V5M6 B/L pupil 2 mm ERTL	
EXPOSURE	Temperature was within normal limits. Diaphoretic, Peripheries and tongue cyanosed	

Primary Survey Adjuncts: ABG -showing high anion gap metabolic acidosis and increased methaemoglobin levels.While drawing blood, blood was in dark chocolate brown colour. Ryles tube inserted and gastric lavage showing yellow coloured aspirate. Even after intubation and mechanical ventilation, saturation was not picking up and cyanosis was not resolving. With the above-mentioned findings, we had suspicion of poison causing methemoglobinemia, so Inj.methyleneblue 100 mg was given over 5 min followed by 5% DEXTROSE infusion @ 50 ml/hr.

Secondary Survey and adjuncts: Alleged history of unknown compound ingestion one hour back following which he had difficulty in breathing and vomiting. CBC, Electrolytes, RFT, LFT were WNL. After 45 min, patient saturation improved around 90%. Patient attenders brought a bottle of suspected poison of ingestion which showed nitrobenzene compound. Patient was on NPO and patient was treated with one more dose of INJ methylene blue 100 mg iv, INJ ascorbic acid 500 mg iv BD and INJ Taxim 1gm iv bd. On day 2 , patient was extubated and was shifted to ward from there pt was discharged after psychiatric opinion.

Discussion

Methemoglobinemia: The ferrous state of iron (Fe2+) in Hb oxidized to the ferric state (Fe3+) under the action of oxidizers, e.g. Nitrite and nitrobenzene , leading to formation of MetHb.

Hbfe3+ loses the ability to carry oxygen. Human body can tolerate a very small amount <1% of metHb, but a higher level is likely to cause methemoglobinemia. Methemoglobinemia shift the O2 dissociation curve to theleft.

Methemoglobin accumulation is enzymatically prevented by the rapid reduction of the ferric iron back to the ferrous form. Cytochrome b5 reductase is primarily responsible for this reduction, in which reduced nicotinamide adenine dinucleotide donates its electrons to cytochrome b5, which subsequently reduces methemoglobin to hemoglobin.

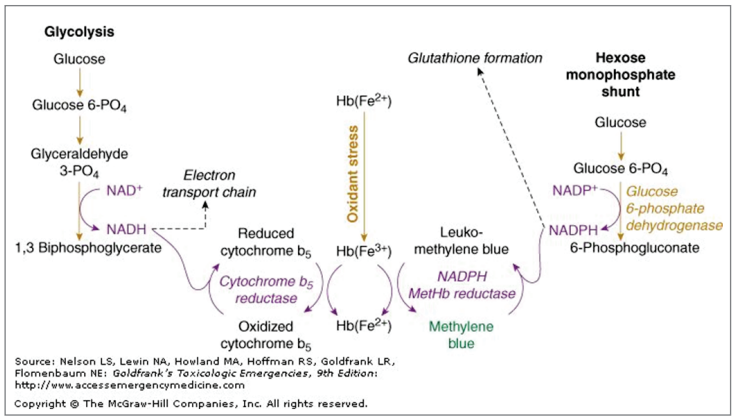
This pathway is responsible for reducing nearly 95% of methemoglobin produced under typical circumstances. Methemoglobinemia occurs when this enzymatic reduction is overwhelmed by an exogenous oxidant stress, such as a drug or chemical agent. Methemoglobin can also be reduced by a second enzymatic pathway using the reduced form of nicotinamide adenine dinucleotide phosphate (or NADPH) and NADPH-methemoglobin reductase. This pathway is normally of minimal importance and is responsible for only less than 5% reduction.

When to Suspect Methemoglobinemia:

- Sudden onset of cyanosis with symptoms of hypoxia after administration or ingestion of an agent that can cause methemoglobinemia.
- Hypoxia (Low Spo2 on pulse oximeter) that does not improve with an increased fraction of inspired oxygen.
- Abnormal dark red, chocolate or brownish coloration of the blood.

Symptoms of Methemoglobinemia

- <15% -skin discoloration (pale, gray, blue)
- 15 - 20% -Cyanosis,
- 25- 50% -Headache, dyspnoea, light-headedness, weakness, confusion, palpitations, chest pain
- 50-70% -Abnormal cardiac rhythms, altered mental status, delirium, seizures, Coma, Profound acidosis.
- >70% - death

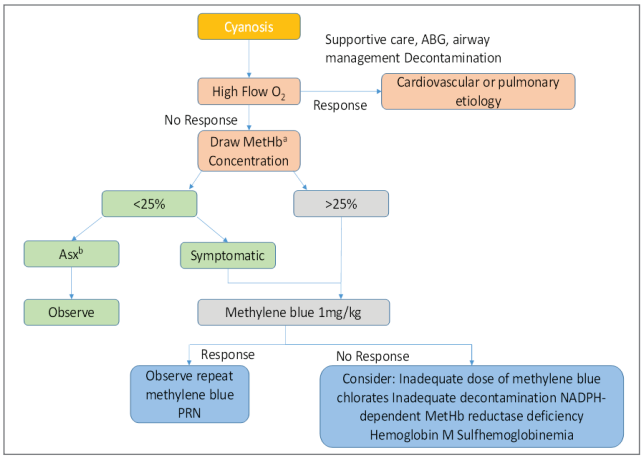


Laboratory investigations

- Oxygen carrying capacity of blood may be determined with the help of ABG, Co-oximetry, Pulseoximetry.
- To rule out hemolysis- CBC, reticulocyte counts, LDH, indirect bilirubin;
- Organ failure & end organ dysfunction: LFT, RFT, Serum electrolytes;
- Electrophoresis to identify Hemoglobin,
- Chocolate colored blood remains same even after exposure to room air or after aerating a tube of blood with 100% O_2

Agents Causing Acquired Methemoglobinemia

Drugs	Dapsone, Clofazimine, Chloroquine, Metoclopramide, Primaquine, Rasburicase, Sulphonamides
Local anesthetics	Benzocaine, Lidocaine, Prilocaine
Nitrites	Amyl nitrite, Sodium nitrite, Nitroglycerin, Nitric oxide
Others	Acetanilide, p-Amino salicylic acid, Aniline, aniline dyes, Benzene derivatives, Chlorates, Naphthalene, Nitrobenzene,Phenacetin, Phenazopyridine, Resorcinol



Management

- Methylene blue 1 to 2 mg/kg body weight infused intravenously over 5 to 10 minutes (0.1 to 0.2 mL/Kg of 1% solution). Repeat dose – in 1 hr intervals till a maximum cumulative dose of 7mg/kg
- Ascorbic acid:(1gm iv sat/ 500 mg b.d for 7 to 10 days)- Reverse methemoglobin by an alternate metabolic pathway, antidote for methemoglobinemia in G6PD deficiency patients.
- Dextrose infusion (50ml/hr) -As glucose supply is needed for reduction pathways to act
- Exchange transfusion- haemolysis, haematuria, refractory hypoxia.

Conclusion

Timely suspicion and early management of hypoxia, cyanosis not resolving with oxygen administration(PaO_2-SpO_2 gap)associated with unknown poisoning as a possibility of methemoglobinemia should be considered and it becomes an eye-opener for Emergency physician. Management with methylene blue as an antidote helps in early recovery & good outcome.

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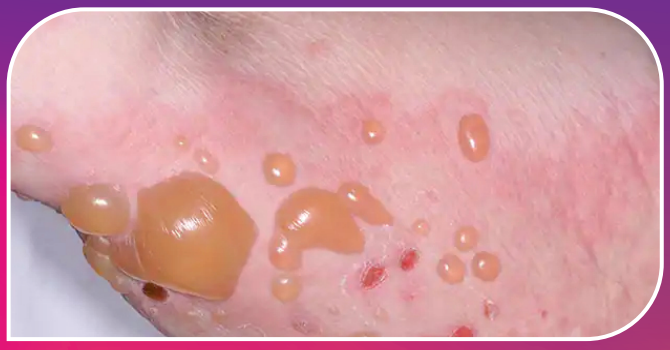

Drug associated bullous pemphigoid: A rare case report

A rare case report

Bullous pemphigoid (BP) is a common autoimmune blistering disease, characterized by sub epidermal separation and inflammation with abundant eosinophils leading to tense bullae.

Immunopathological findings of BP are linear deposits of C3 and IgG at the basement membrane zone (BMZ). The autoantibodies against two main structural proteins of the dermal-epidermal junction, BP antigen 1 (BPAG1 or BP230 antigen) and BPAG2 (or termed BP180 antigen), are involved in the pathogenesis of BP. 56 year old female presented with generalised vesciculobullous (Figure1) lesions of 2 months duration. She was on treatment for type II diabetes mellitus for past 3 years and was on metformin and glimeperide. Her fasting blood sugar (FBS) was 281mg/dl, post prandial blood sugar (PPBS) was 362 mg/dl and HbA1C was 8.9%. Complete haemogramshowed erosinophils 25%. Also patient had hyponaetremia, hypocalcemia and hypoalbuminemia.

Lipid profile, Liver and Renal function tests were within normal limits. Urine routine and microscopic examination showed glucose2+. USG Abdomen showed mild fatty liver and Chest X-ray detected no abnormalities. Skin biopsy was taken from lesion(figure2,3) for histopathologyand perilesionalarea for DIF. DIF showed a linear deposit of immunoglobulin IgG band C3 along the basement membrane zone (Figure 4) and confirmed our clinical diagnosis of BP. She was admitted and during hospital admission, treated with systemic steroids with 1mg/kg/dose, insulin and other supportive medications including correction ofhypernatremia, hypocalcaemia and hypoalbuminemia.

As patient had poor glycaemic status along with insulin glimepiride and metformin was continued and also added vildagliptin. Even after 1week of systemic steroids her skin lesions were not improving and started with azathioprine 2mg/kg dosage and still no improvement was noticed. So stopped all oral hypoglycaemic medications and managed her diabetes with insulin. Day to day examination showed improvement of her skin lesions after stopping oral hypoglycaemic agents and no new skin lesions developed in next 1 week. Patient relieved from symptoms and blisters started healing well and she was discharged with advice to continue oral prednisolone with tapering doses, azathioprine, and other oral supplements till follow up visit. After a week the blisters were healing well without any scarring. After 2 weeks on review RBS was 550 and again restarted oral hypoglycaemic agents suspecting insulin resistance. Patient was on glimepiride 1mg and metformin for her diabetes mellitus before onset of disease. Considering the strong association of BP with Gliptins and sulphonylureas and avery rare association with metformin we restarted metformin.

Two weeks later when patient reviewed lesions started reappearing and oral steroid was updosed and started dapsone after stopping azathioprine. Even after two weeks there was no considerable difference and new lesions were developing. Then we stopped metformin and increased the dose of insulin. A week later skin lesions started improving and new lesions also stopped appearing. Now the patient is on oral steroid, dapsone and supportive medications.

Bullous pemphigoid (BP) is an acquired cutaneous autoimmune vesciculobullous condition. The clinical manifestations of BP include pruritic tense bullae, eczematous and urticarial skin lesions. In 10 to 20% of patients oral mucous membrane

Bullous pemphigoid (BP) is an acquired cutaneous autoimmune vesciculobullous condition. The clinical manifestations of BP include pruritic tense bullae, eczematous and urticarial skin lesions. In 10 to 20% of patients oral mucous membrane erosions may be present. The incidence of BP appears to be equal in men and women and it is most common in older adults. There is no known ethnic, racial or sexual predilection for bullous pemphigoid5. The autoimmune reaction against the hemidesmosomal proteins bullous pemphigoid antigen 180 (BP180) and/or bullous pemphigoid antigen 230 (BP230) results in BP1-3. Many pathomechanisms have been proposed in the abnormal immune response, but the exact reason for this is unknown, although it sometimes can be triggered by taking certain medications, trauma, burns, radiotherapyand ultraviolet irradiation8. 89 drugs were implicated in DABP.

The strongest association for bullous pemphigoid is seen with gliptins, PD-1/PD-L1 inhibitors, loop diuretics, nonsteroidal anti-inflammatory drugs (NSAIDs; ibuprofen), captopril, phenacetin, penicillamine, etanercept, systemic antibiotics (pencillins), and derivatives. The knowledge about Drug associated BP (DABP) and its associated drugs enables clinicians to identify potential cases of DABP earlier and to stop the offending medication at the earliest.Drug associated BP is a term used to describe instances of BP demonstrating clinical, histological, or immunopathological features identical or similar to those of the idiopathic form of the disease. Systemic intake or topical application of particular drugs can lead to DABP.

Metformin induced BP was first reported in 2015. Although in most cases the causative agent remains unidentified, certain medications have been implicated in the pathogenesis of the disease. It is difficult to differentiate classic BP from DABP clinically. Hence, Drug induced BP must always be considered as a possible diagnosis in the elderly who have been started on new medications3. Metformin, being the first preferred and effective agent for the management of type 2 DM, is widely used.

Localized BP may be treated with highpotency topical steroid and extensive disease require use of systemic corticosteroids alone or combined with immunosuppressive drugs such as azathioprine, cyclophosphamide, mycophenolate or rituximab. Patients with moderate disease can be treated with dapsone, tetracyclines which may be combined with niacinamide, minimizing the use of sterioids. Metformin is not known to cause very severe adverse effects, apart from mild gastro intestinal upset and lactic acidosis. The literature search revealed only two previous case reports of metformin induced Bullous pemphigoid and this case will be third one.

Conclusion

Drug history of all BP patients is very important though DABP is a rare entity. Early identification and discontinuationof the culprit drug, helps in rapid treatment response and overall treatment outcome. Metformin induced BP in diabetes patient is a rare case reoprt in medical literature.

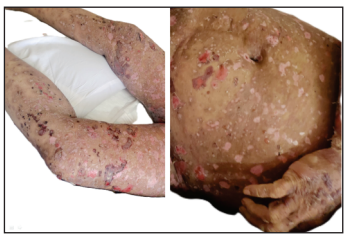

Figure 1

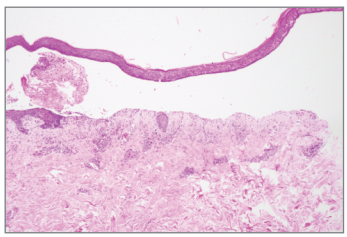

Figure 2
Subepidermal bullae

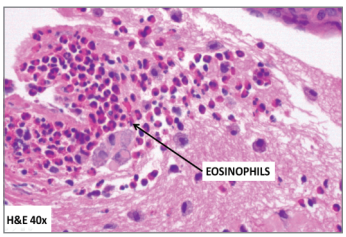

Figure 3
Eosinophilic infiltrate in dermis and bullae

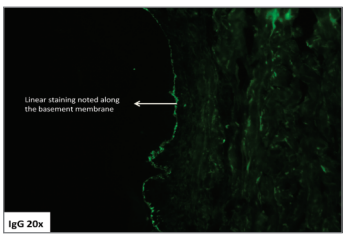

Figure 4
Linear staining of IgG and C3 noted in basement membrane

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Consultant Dermatologist

Trichotillomania(TTM)

Introduction

Trichotillomania is a psychodermatologic disorder characterized by uncontrollable urge to pull one's own hair, mostly from the scalp, but also from eyebrows, eyelashes, pubic area, beard or moustache. TTM onset in childhood or adolescence appears to be the norm. The etiology of TTM in children can be based on a wide range including parental issues at home, bullying and poor performance, whereas in adults it is commonly associated with anxiety disorders, mood disorders, substance abuse and personality disorders.

Case Report

A 15 year old male patient, accompanied by his mother, reported patchy hair loss over the scalp for the past 6 months. He had been previously diagnosed as Alopecia Areata(AA) and was treated with topical minoxidil and steroids . The mother noted improvement in existing patches and at the same time new lesions developed in nearby areas. There was no history of pulling of hair, abnormal behaviour or any other comorbidities. He used to be good in academics.

Cutaneous examination revealed, large, non scarring, alopecic patches over the scalp. Hair pull test was negative. Trichoscopic examination showed decreased hair density, broken hairs at varying lengths and red dots(follicular microhemorrhage). Exclamation hairs or yellow dots, suggestive of AA were not observed.

Histopathological evaluation could not be done as patient refused biopsy of the scalp. On probing again, patient admitted the habit of plucking his hair, and that, he lives with his mother and is separated from his brother and father. Based on the history, clinical findings and trichoscopic features, a diagnosis of TTM was made. The patient was referred to the psychiatric consultation and psychologic evaluation. He was started on SSRI(Fluoxetine) followed by behaviour therapy.

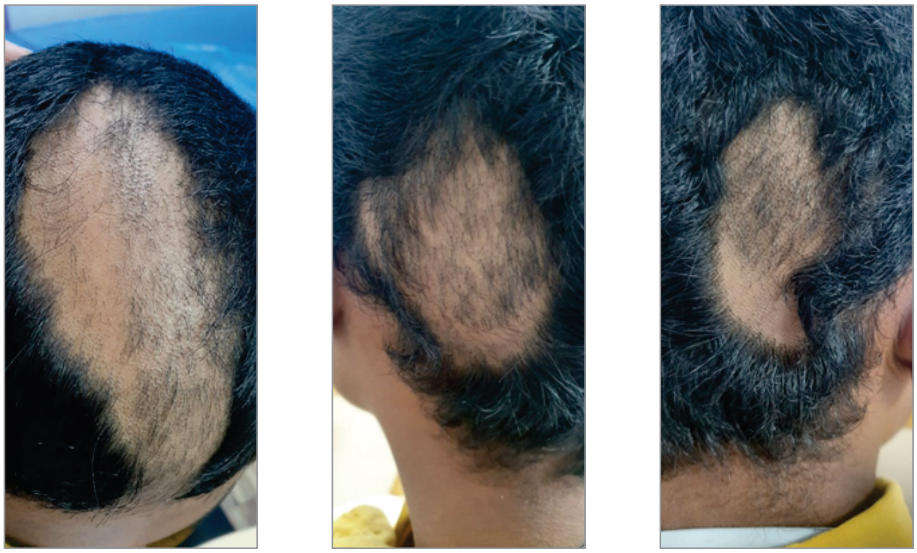
TTM is a chronic impulse control disorder, first described by Hallopeau in 1889. Age of onset varies from 9 to 13 years and is more common in females. Diagnosis is usually by exclusion after a thorough history and dermatological examination. But, in pediatric patients it is often tough, due to 2 reasons, a. Difficulty in eliciting right history b. Other disorders like AA and tinea capitis have similar clinical presentation.

Clinically, TTM presents with multifocal and irregular patches formed by multiple twisted and broken hairs. The favourite site is the easily reached fronto-parietal region of the scalp. In more severe forms, tonsure pattern of baldness, known as "Friar tuck sign"(alopecia of the vertex with sparing of occipital region) is seen. A hair ball, trichobezoar, is a rare accompaniment of TTM, in those who also eat the plucked hair(trichophagia). Such patients usually present to the emergency or surgery department with abdominal pain/discomfort. AA , in turn, reveals smooth and shiny patches with ragged edges.

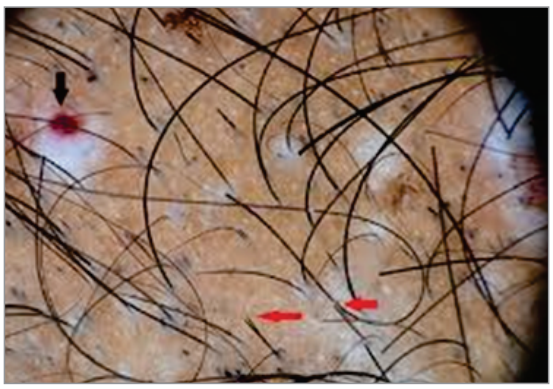
In doubtful cases, trichoscopy can be done, which shows decreased hair density, broken hairs at varying lengths, red dots(follicular microhemorrhage), irregular coiled hairs, trichoptilosis(split ends), flame hairs, V sign, tulip hairs and hair powder(sprinkled hair residue). Findings in AA include exclamation hairs, yellow dots, uniform black dots, broken hair, trichoptillosis and vellus hair.

In cases where overlapping features are found, histopathology plays a corroborative role in definitive diagnosis. Histologic features of TTM are empty anagen follicles, increased numbers of non inflamed catagen follicles and pigment casts in hair canals, whereas, AA shows reduction in number of hair follicles with evidence of miniaturization, bulbar and periadnexal lymphocytic inflammation and a catagen/ telogen shift.

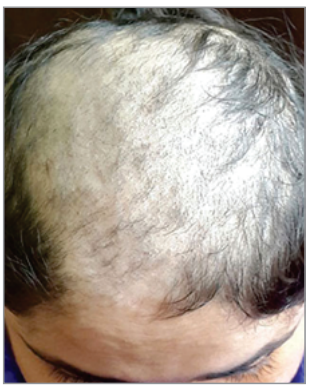
Any child presenting with non scarring hair loss needs to be evaluated in detail depending on the pattern and type of hair growth. AA and TTM are two diseases affecting similar clinical profiles with potential stigmatising implications. It is important to distinguish between the two and establish the correct therapeutic approach for each of the conditions.



Multiple Alopecic Patches over the scalp showing regrowth and peripheral extension



Trichoscopic features showing red dots. Reduced density hairs of varying length



Friar Tuck Sign

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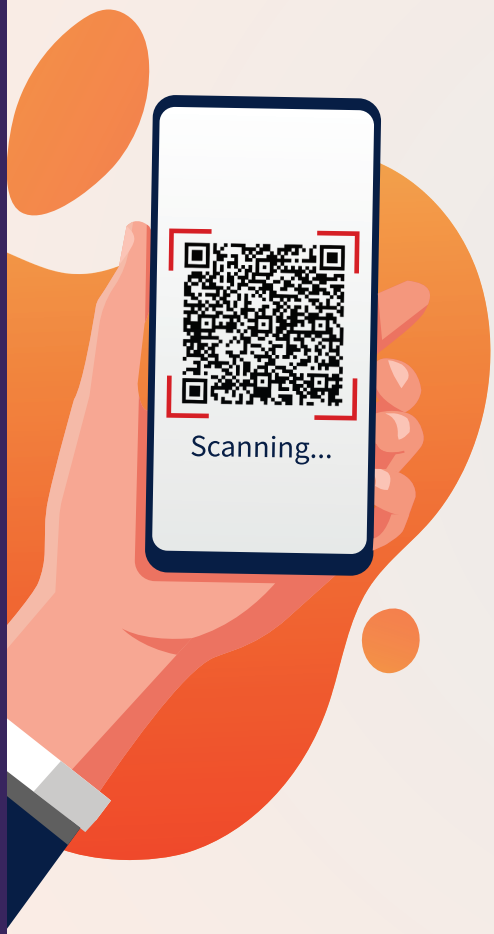

FOR THE FIRST TIME IN INDIA

LAUNCHING QR CODES

WITH COMPLETE CANCER AWARENESS INFORMATION

IN ENGLISH AND TAMIL

SCAN THE QR CODE
TO VIEW DETAILS IN YOUR SMART PHONE

- Providing information on What is Cancer, Types of Cancer, Diagnosis & Treatment, When to consult your Doctor with animated contents and Ebooks
- Exclusive Audio based and Video based information on Breast Cancer
- Connect to view Medical experts video talks on SRIOR Youtube channel

World Cancer Day 4 February

Close the care gap

#CloseTheCareGap #WorldCancerDay

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LAUNCHING FREE MAMMOGRAM

FOR ENTIRE YEAR FROM 2024 - 2025
(ONLY ON WORKING DAYS)

AWARENESS FOR CANCER **AWARENESS FOR LIFE**

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