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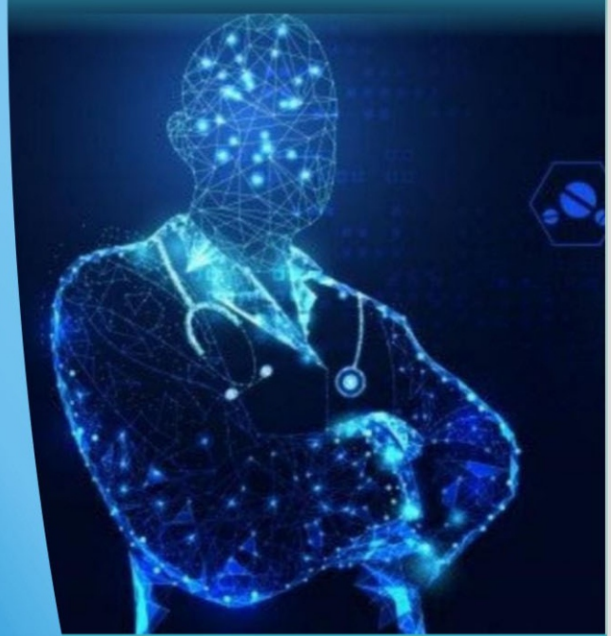

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Contents

EDITORIAL

"Beyond the Bedside: A Comprehensive Look at ICU Admission and Discharge Criteria" and Illuminating the Crucial Role of the Intensivist in ICU Management

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INTRODUCTION

As of October 8, 2023, the Ministry of Health and Family Welfare, Government of India, has issued detailed guidelines for intensive care unit (ICU) admission and discharge, including specific eligibility criteria for intensivist.[1]

COMMITTEE FORMATION

The Ministry of Health and Family Welfare, Government of India, has constituted a committee comprising 24 experts in critical care medicine from various hospitals and ICUs across the nation.

These guidelines were formulated through a collaborative effort involving all experts in critical care medicine, led by the Ministry of Health and Family Welfare, Government of India.

The guidelines articulate the ICU as a specialized environment dedicated to the multidisciplinary and focused management of individuals facing life-threatening dysfunction of vital organs, whether partially or fully reversible. Continuous monitoring and prompt interventions are deemed crucial, necessitating collaboration among multidisciplinary teams, including doctors, nurses, and especially trained staff such as physiotherapist, clinical pharmacist, and respiratory technician. Moreover, the ICU may necessitate specialized instruments and equipment to optimize patient care.

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ICU ADMISSION CRITERIA

The criteria for admitting a patient to the ICU should depend on the presence of organ failure, the necessity for organ support, or the anticipation of deterioration in the medical condition.

In the complex landscape of critical care, the decision to admit a patient to the ICU is guided by specific indications that demand heightened attention to altered physiology and specialized interventions to correct the abnormalities (as far as possible). New-onset altered consciousness triggers the need for vigilant monitoring and expert care within the ICU, ensuring a prompt response to evolving conditions. Hemodynamic instability (pulse, blood pressure, or other vital parameters) positions individuals as prime candidates for ICU admission, where continuous cardiovascular support is readily available. Respiratory failure, characterized by varying oxygen support requirements or the need for ventilation, underscores the critical role of the ICU in advanced respiratory care. Patients grappling with severe acute illnesses, necessitating intensive monitoring and organ support, find a tailored environment for their complex needs in the ICU. Anticipation of deterioration in any medical condition becomes a compelling reason for ICU admission, allowing for proactive management and timely interventions.

Moreover, major intraoperative complications, such as cardiovascular or respiratory instability, warrant immediate attention and resources, which the ICU is equipped to provide. Similarly, those who have undergone major surgeries, including trauma patients at a heightened risk of post-operative complications, benefit from the specialized

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care and monitoring available in the ICU. Recognizing and comprehending these indications are paramount, ensuring that individuals receive the precise and timely care required during critical phases of their medical journey. The ICU stands as a pivotal environment, offering a multidisciplinary approach to address the intricacies of these high-risk scenarios and optimize patient outcomes. Table 1 shows examples of indications for ICU admission.

THE CRITICALLY ILL, WHO SHOULD NOT BE ADMITTED TO ICU

In emergency room among all the critically ill patients, we have to decide who should not be admitted in critical care even though these patients have hemodynamic instability.

Certain critically ill patients are identified as not suitable for admission to the ICU, and these specific criteria guide the decision-making process. First, patients or their next-of-kin expressing a clear refusal to be admitted to the ICU are respected, acknowledging individual preferences and choices in medical care. In addition, individuals with diseases accompanied by treatment limitation plans are not directed to the ICU, aligning with established care directives. Those possessing living wills or advanced directives explicitly against ICU care also fall within this category, ensuring medical decisions align with the expressed wishes of the patient. Terminally ill patients, where medical judgment deems further interventions futile, are considered unsuitable for ICU admission. Furthermore, in scenarios of resource limitation, such as during a pandemic or disaster situation (mass casualties exceeding the current capacity of casualty), individuals meeting low-priority criteria during triage are not directed to the ICU. These exclusion criteria collectively contribute to a nuanced and ethical approach in allocating ICU resources and aligning medical care with the individual's informed preferences and broader health-care priorities.

Table 1: Clinical scenario warranting ICU admission

1	New-onset altered consciousness
2	Hemodynamic instability
3	Respiratory failure
4	Patients with severe acute (or acute-on-chronic) illness requiring intensive monitoring and/or organ support
5	Any medical condition or disease with anticipation of deterioration
6	Patients with major intraoperative complication
7	Patients who have undergone major surgery (e.g., thoracic, thoracoabdominal, upper abdominal operations, trauma) who require hemodynamic monitoring or at a high risk of developing post-operative complications.

ICU: Intensive care unit

These guidelines aid in determining whether a critically ill patient requires ICU admission or can be effectively managed with treatment in a general ward. Guidelines helps for determining which critically ill patients should not be admitted to the ICU, but they fall short in individualization or personalization of the management of these patients. Intensivist faces limitations in treating critically ill patients outside the ICU due to the absence of secure environment, necessary equipment, and monitoring tools in non-ICU settings.

The existing guidelines have limitations as they do not address the management of critically ill patients who necessitate ICU admission but face refusal from family members. "Relatives who refused ICU admission but want treatment for their patient" form a particularly vexatious situation remarks whether these patients should be given care equivalent to ICU in non-ICU setting or they should be given care which is appropriate to the setting in which they are admitted. There is a need for comprehensive guidelines specifically outlining the management approach for such patients. Table 2 to see examples of patient who should not be admitted in ICU

ICU DISCHARGE CRITERIA

Before discharging, the critically ill patients from ICU intensivist have to consider the following criteria.

The decision to discharge a patient from the ICU hinges on several factors aimed at ensuring optimal recovery and resource allocation. First, the return of physiological aberrations to near– normal or baseline status marks a significant milestone in determining readiness for discharge. This indicates that the acute illness necessitating ICU admission has reasonably resolved, and the patient has achieved stability.

Guidelines have not specified about near normal physiological status whether 20–30% of standard deviations is acceptable and should be considered as near normal or much more. Furthermore, baseline status of physiological status may not be known so return to baseline near normal may not be always determined.

Another crucial aspect involves the agreement of the patient or their family for ICU discharge, especially when a treatment-limiting decision or a transition to palliative care is contemplated. In India where majority of health care is administered by private institutions, economic status may creep in inadvertently when such decisions are being taken by family members.

In addition, decisions to cease aggressive care and discharge should primarily stem from a medical perspective, avoiding reliance on economic constraints and ideally not mandating family agreement. In certain cases, ICU discharge may be prompted by infection control measures, with a careful plan to ensure appropriate care in a non-ICU setting. Furthermore,

Table 2: Patients who should not be admitted in ICU

1	Patient's or next-of-kin informed refusal to be admitted in ICU
2	Disease with a treatment limitation plan
3	Anyone with a living will or advanced directive against ICU care
4	Terminally ill patients with a medical judgment of futility
5	Low priority criteria in case of pandemic or disaster situation where there is resource limitation (e.g., bed, workforce, equipment).

ICU: Intensive care unit

Table 3: ICU discharge criteria

1	Return of physiological aberrations to near normal or baseline status
2	Reasonable resolution and stability of the acute illness that required ICU admission
3	Patient/family agrees for ICU discharge for a treatment-limiting decision or palliative care.
4	Based on lack of benefit from aggressive care (should be a medical decision, not obligating family agreement and as far as possible should not be based on economic constraints).
5	For infection control reasons with ensuring appropriate care of the given patient in a non ICU location
6	Rationing (i.e., prioritization in the face of a resource crunch). In this event, there should be an explicit and transparent written rationing policy that should be fair, consistent, and reasonable.

ICU: Intensive care unit

Table 4: Monitoring in awaiting area

1	Heart rate and blood pressure (continuous/intermittent)
2	Hemodynamic monitoring (e.g., pulse rate, respiratory rate, breathing pattern, etc.)
3	Oxygen saturation – SpO2 (continuous/intermittent)
4	Capillary refill time
5	Urine output (continuous/intermittent)
6	Neurological status, e.g., GCS, AVPU scale, etc.
7	Intermittent temperature monitoring
8	Blood sugar

GCS: Glasgow coma scale, AVPU: Alert verbal pain unresponsive

during periods of resource scarcity, a transparent and fair written rationing policy becomes pivotal. This policy aims to prioritize patient care judiciously, promoting consistency and reasonableness in decision-making. The multifaceted considerations for ICU discharge underscore the importance of a balanced and patient-centered approach, aligning medical decisions with the best interests of the individual while navigating challenges posed by resource constraints.

Table 5: Stabilization in emergency room

1	Ensuring a secure airway (i.e., tracheal intubation if the patient has a GCS ≤ 8)
2	Ensuring adequate oxygenation and ventilation.
3	Hemodynamics stability either with or without vasoactive drug infusion
4	Ongoing correction of hyperglycemia/hypoglycemia and other life-threatening electrolyte/metabolic disturbances
5	Initiation of definitive therapy for life-threatening condition (e.g., external fixation of a fractured limb, administration of antiepileptic's for recurrent seizures, antiarrhythmic drug infusion for unstable arrhythmias, etc., intravenous antibiotics for sepsis)

GCS: Glasgow Coma Scale

Table 6: Parameters to monitor during interfacility transfer

1	Blood pressure (continuous/intermittent)
2	Clinical monitoring (pulse rate, respiratory rate, breathing pattern, etc.)
3	Continuous heart rate
4	Continuous SpO_2
5	Neurological status (AVPU, GCS, etc.)

GCS: Glasgow coma scale, AVPU: Alert verbal pain unresponsive

The discharge criteria from the ICU are beneficial for patients who have responded to treatment and achieved hemodynamic stability after acute illnesses that necessitated ICU admission. However, limitations arise when discharging critically ill patients, especially when their families agree to discharge due to treatment constraints and the absence of benefits from aggressive treatment. In addition, these guidelines do not provide specifics regarding the transportation of critically ill patients after discharge, considering the limitations in treatment and the lack of benefits from aggressive care. Table 3 to see examples of ICU Discharge criteria.

THE MINIMUM PATIENTS MONITORING REQUIRED WHILE AWAITING AN ICU BED INCLUDE THE FOLLOWING

Emergency room should be well established to monitor the following parameters of the critically ill patients who require ICU admission and are awaiting an ICU bed.

The critical care environment mandates vigilant monitoring of various physiological parameters to ensure comprehensive patient assessment. Key indicators include continuous or intermittent tracking of heart rate and blood pressure,

offering insights into cardiovascular stability. Hemodynamic monitoring, encompassing pulse rate, respiratory rate, and breathing pattern, provides crucial information on the overall circulatory and respiratory status. Oxygen saturation (SpO_2) is monitored continuously or intermittently to assess respiratory function and the effectiveness of oxygen therapy.

Additional parameters, such as capillary refill time, serve as indicators of perfusion and circulatory health. Urine output, monitored continuously or intermittently, is essential for evaluating renal function and fluid balance. Neurological status is assessed using tools such as the Glasgow coma scale (GCS) and the alert verbal pain unresponsive (AVPU) scale, offering valuable insights into cognitive function and responsiveness. Intermittent temperature monitoring provides a snapshot of the patient's thermoregulation, aiding in the detection of fever or hypothermia. Blood sugar levels are also routinely monitored, ensuring glycemic control in critically ill patients. Blood sugar level monitoring is especially relevant in India where large proportion of population has clinically undiagnosed hyperglycemia.

Collectively, these monitoring parameters form a comprehensive approach to patient care in the awaiting area setting, facilitating prompt interventions and personalized treatment plans. Table 4 to see examples of parameters to monitor in awaiting area.

MINIMUM STABILIZATION REQUIRED BEFORE TRANSFERRING A PATIENT TO ICU INCLUDE THE FOLLOWING

Before transferring the critically ill patients to ICU, minimum stabilization of the critically ill patients should be done in emergency room.

Minimal stabilization for critically ill patients mandates prompt interventions. Ensure airway security (tracheal intubation if GCS ≤8), optimize oxygenation, stabilize hemodynamics, correct metabolic imbalances, initiate definitive therapy for life-threatening conditions, prevent accidental secondary emergency. Table 5 to see parameters to stabilise in emergency room.

MINIMUM MONITORING REQUIRED FOR TRANSFERRING A CRITICALLY ILL PATIENT (INTER-FACILITY TRANSFER TO HOSPITAL/ICU)

After stabilization of the patient in emergency room following parameters has to be monitored during transferring the patient.

Minimum monitoring required for transferring critically ill patients includes Continuous monitoring of vital signs includes blood pressure, clinical parameters (pulse rate, respiratory rate, etc.), and continuous heart rate and SpO_2. Neurological status should be assessed through AVPU and GCS for comprehensive care. Table 6 to see the examples of parameters to monitor during inter facility transfer.

To ensure minimal stabilization and monitoring of patients awaiting an ICU bed in the hospital's emergency room/ casualty, a multidisciplinary team is essential. This team should include doctors, nurses, and trained staff, along with specialized instruments and equipment to enhance patient care. In addition, having a critical care specialist in the casualty/emergency room with specific training and experience in treating critically ill patients is crucial.

According to the guidelines, an intensivist or critical care specialist is a professional with specific training, certification, and experience dedicated to the treatment of critically ill patients within the ICU.

WHO IS AN INTENSIVIST? CRITERIA FOR INTENSIVIST

The intensivist is required to possess a postgraduate qualification in broad specialties of internal medicine, anesthesia, pulmonary medicine, emergency medicine, or general surgery, along with super special degree in critical care or certificate course of national society of critical care medicine or post-doctoral fellowship in critical care (PDCC/fellowship) or equivalent course from foreign universities.

a. An additional qualification in Intensive Care, such as DM Critical Care/Pulmonary Critical Care, DNB/ FNB Critical Care (National Board of Examinations), Certificate Courses in Critical Care of the Indian Society of Critical Care Medicine (ISCCM) (Indian Diploma in Critical Care Medicine and Indian Fellowship in Critical Care Medicine), PDCC/Fellowship from an NMC recognized University, or equivalent qualifications from abroad, such as American Board Certification, Australian or New Zealand Fellowship (FANZCA or FFICANZCA), UK (CCT dual recognition), or its equivalent from Canada.
b. Alternatively, a minimum of 1-year training in a reputable ICU abroad. Candidates from the ISCCM Certificate Course (CTCCM) who have completed a 3-year training program in Intensive Care after MBBS are also acknowledged as Intensivist. Moreover, individuals with the mentioned qualifications or training should possess a minimum of 2 years of experience in an ICU, with at least 50% of their time spent in the ICU.

In cases where doctors lack the aforementioned qualifications or training, extensive experience in Intensive Care in India post MBBS is required, amounting to a minimum of 3-year experience in ICU, with at least 50% of their time spent in the ICU.

As per the guidelines, European Diploma in Intensive care is not considered as a qualification for an Intensivist. Furthermore, although the guidelines recognize MBBS with 3 years of ICU experience, requiring a minimum of 50% time spent in the ICU for an Intensivist, it is emphasized that experience and a 50% ICU time commitment may not be adequate for effective management of critical care patients.

It is difficult to fathom the equivalence or otherwise if these criteria as on the one hand, there is 3 years of training followed by an exit examination for the DM/DrNB critical care medicine course while only 1-year training in a reputable ICU abroad. Furthermore, for the post MBBS 3-year training, 50% time spent in ICU and may be too little so modifications in criteria need to be considered.

CONCLUSION

The newer guidelines provide a reliable framework regarding admission/ discharge decisions, monitoring of the critically ill patients and eligibility of intensivist. There is a scope of improvement and further modifications need to be considered for maximum benefit of these guidelines.

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CASE REPORT

A Case of Reactivation of Acute Rheumatic Fever in a Known Case of Rheumatic Valvular Heart Disease

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ABSTRACT

Acute rheumatic fever (ARF) is an inflammatory disease that can develop as a complication of untreated or inadequately treated streptococcal throat infection, specifically caused by group A Streptococcus bacteria (Streptococcus pyogenes). Diagnosis of ARF is based on the Jones Criteria, which include major and minor criteria related to clinical presentation, laboratory findings (such as elevated anti-streptolysin O titer), and evidence of preceding streptococcal infection.

Key words: Acute rheumatic fever, rheumatic valvular heart disease, Jones criteria

INTRODUCTION

Rheumatic fever is an acute, post-streptococcal, immune-mediated, multisystem inflammatory disease. It occurs as a sequel to Group A streptococcal pharyngitis. It develops after a latent period of 2–6 weeks. [1,2] The most common age of presentation is between 5 and 15 years.

CASE REPORT

A 19-year-old female presented in our casualty with chief complaints of chest pain and breathlessness for 2 weeks, palpitations, and right hip joint pain for 2 days. The patient was apparently alright 2 weeks ago when she experienced breathlessness which was insidious in onset initially at doing routine activity and later aggravated at rest and was associated with orthopnea and PND. She was relieved on getting up from bed. Her chest pain was sudden in onset, left-sided, constricting, and squeezing in nature and it was non-radiating. There was no diurnal variation. It was continuous in nature and settled after taking beta-blockers. She was having palpitations since 2 days, and it was sudden onset, fast, persistent, and was not associated with aggravating or relieving factors. She also started having right joint pain for 2 days which was sudden onset agonizing and severe pain continuous in nature non-radiating so much so that the patient was unable to get up from bed due to pain at the hip joint; it was without redness and local swelling.

There is also a history of high-grade fever, a single episode 2 weeks ago which was continuous in nature and settled after taking medication.

There was no history of cough, syncope, pedal edema, decreased urine output, or cyanosis. She has had a known case of rheumatic valvular heart disease since the age of 6 and has been taking injection of benzathine penicillin 1.2 million IU IM once in 3 weeks.

The patient has missed the dose of injection since the past 4 months and with the above complaints, she came to our emergency department.

The patient was conscious and oriented to time, place, and person but she was tachypenic and tachycardia was there with palpitations which were now continuous in nature.

On Investigations

In the emergency room, her ECG was done which has been shown below showing the junctional rhythm.

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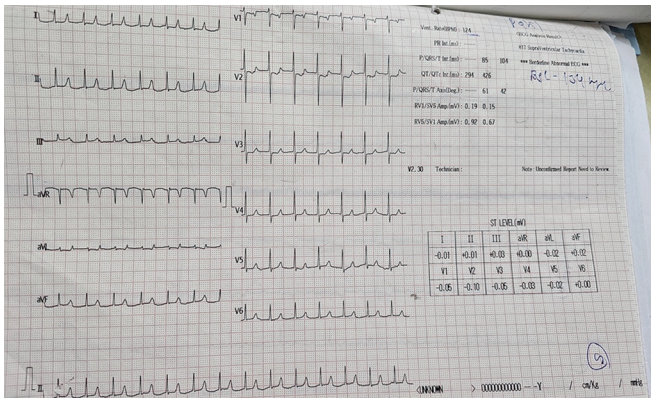


At this time, the patient received an injection of amiodarone and was started on amiodarone infusion.

For the next 24 h until her heart rate settled.

The patient's sample for ASO titer and high sensitivity C reactive protein (hsCRP) had been sent of which both came positive with a significant titer.[3]

Her 2D ECHO was done and was suggestive of mild-to-moderate mitral regurgitation with rheumatic affection on mitral valve leaflets and AML doming.

For her hip joint pain, she was started on NSAIDS and was continued for a period of 1 week. Her pain had settled by this time. The next day patient started complaining of left shoulder pain and now was started on injection dexamethasone and was on it for a week until the pain settled and then shifted to an oral aspirin tablet.

The patient was also given an oral tablet of penicillin G 4 lakh units twice a day for a period of 2 weeks and was also on beta-blockers and a low dose of diuretics.

Diagnosis

Jones Criteria for Rheumatic Fever

Major Criteria	Minor Criteria
Pancarditis (pericarditis, endocarditis, myocarditis)	Fever
Polyarthritis	Arthralgia
Sydenham Chorea	Prolonged PR interval
Subcutaneous Nodules	Increased ESR or CRP*
Erythema marginatum	Leukocytosis

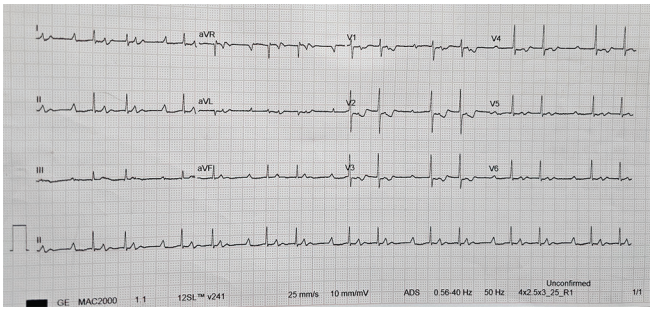
*Erythrocyte sedimentation rate or c-reactive protein LearnTheHeart.com
**Two major or 1 major and 2 minor must be present to diagnose rheumatic fever

Our patient was fitting into the Jones criteria[4] for acute rheumatic fever.

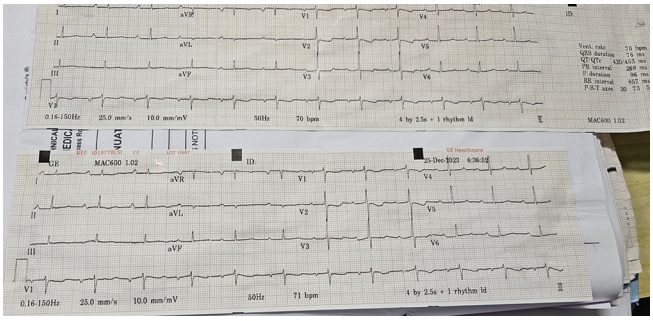
There was evidence of pancarditis with fever, arthralgia, prolonged PR interval, leukocytosis, and increased hsCRP.

There was evidence of preceding streptococcal infection within the past 45 days which was supported by elevated ASO titers.

Her ECG on day 2 was done which was showing atrial bigeminy with prolongation of PR interval.



After 1 week, her ECG showed normal sinus rhythm with PR prolongation being constant.



The patient after receiving adequate treatment was symptomatically better and has been discharged on diuretics, beta-blockers, and injection of benzathine penicillin which she will require lifelong as a prophylactic measure.

CONCLUSION

A missed dose of benzathine penicillin can lead to the reactivation of acute rheumatic fever in a known case of rheumatic valvular heart disease. The prophylactic regimen states to continue injection of benzathine penicillin 1.2 M IU once in 3 weeks for 21 years of age.[5-7] In our patient who has a high risk of reactivation, the patient will require the therapy for lifelong.

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CASE REPORT

A Rare Case of Unilateral Ovarian Sex Cord Tumor with Annular Tubules

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ABSTRACT

Sex cord tumor with annular tubules (SCTATs) is a rare ovarian tumor accounting for <1% of sex cord stromal tumors. It is of two types sporadic and syndromic. Syndromic SCTATs are regarded as benign, are usually bilateral, and are associated with germline STK11 mutations on chromosome 19p13.3 characteristic of Peutz–Jeghers syndrome. Sporadic SCTAT cases are unilateral and about 20% of them are regarded to manifest extra ovarian spread. Here, we intend to put on record a rare case of sex cord stromal tumor with annular tubules in a 49-year female presenting with left adnexal mass.

Key words: Ovarian tumor, hyperestrinism, sex cord tumor with annular tubules, inhibin

INTRODUCTION

Sex cord tumor with annular tubules (SCTATs) is a rare ovarian tumor accounting for less than 1% of sex cord stromal tumors. It was first described in 1970 by Scully.[1] It is of two types sporadic and syndromic.[2] Syndromic SCTATs are regarded as benign, are usually bilateral, and are associated with germline STK11 mutations on chromosome 19p13.3 characteristic of Peutz–Jeghers syndrome (PJS).[3] Sporadic SCTAT cases are unilateral and about 20% of them are regarded to manifest show extra ovarian spread.[4] Here, we intend to put on record a rare case of sex cord stromal tumor with annular tubules in a 49-year female presenting with left adnexal mass.

CASE REPORT

A 49-year, Gravida-2-Para-2-living-2, married female presented to gynecologic outpatient department with complaints of abdominal pain for a week and menorrhagia for 3 years. She had a history of death of one fetus at 36 weeks of gestation and lower segment cesarean section with twin pregnancy. Laboratory tests showed CA-125 of 13 U/mL (normal range: <35 U/mL). Ultrasound (USG) showed a well-defined complex solid-cystic lesion measuring 6.5 × 5.4 cm in left adnexa. The patient underwent total abdominal hysterectomy with left salpingo-oophorectomy. Resected specimen was sent for histopathological evaluation.

Grossly, globular soft to firm tissue mass measuring 8 cm in diameter and weighing 750 g was seen. The capsule was intact. Cut surface was solid, yellowish with few tiny cystic areas. Microscopically tumor cells were arranged in sheets and in simple and complex tubular areas. Few tubules were ring shaped with peripherally oriented nuclei around central hyaline material while few showed interconnecting rings revolving around multiple hyaline bodies. Individual tumor cells were medium sized, round to oval with moderate amount of cytoplasm with vesicular chromatin with few nuclei showing grooves. There was minimal atypia and mitosis of 1/10 HPF. There was no lymphovascular invasion noted. Histopathological diagnosis of unilateral sex cord stromal tumor with annular tubules was offered. Immunohistochemistry (IHC) done with inhibin

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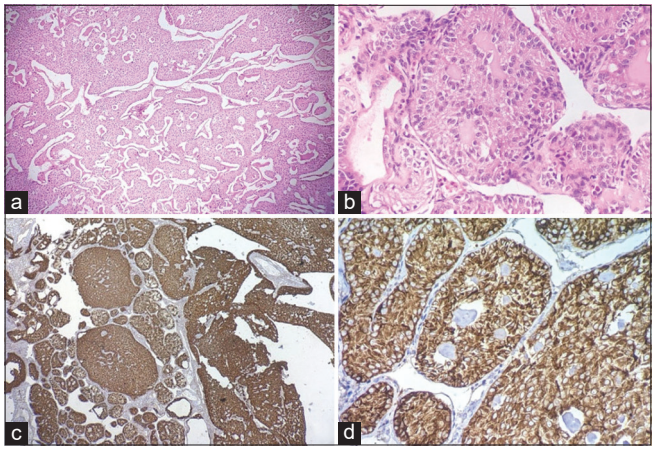

Figure 1: Photomicrograph showing (a) sharply delineated simple and complex tubules containing basement membrane-like material on ×100 and (b) ×400 magnification (c) tumor cells expressing strong cytoplasmic inhibin positivity on ×100 and (d) ×400 magnification

showed strong cytoplasmic immunoreactivity confirming the diagnosis (figure 1). The endometrial study showed disorderedly proliferative endometrium with tiny endometrial polyp. There was seen intramural leiomyoma of 1 cm in diameter. The patient was reexamined and radiologically reinvestigated to rule out PJS. General physical examination and gastrointestinal endoscopy ruled out syndromic SCTAT. On follow-up, the patient is disease free for 1½ years.

DISCUSSION

The SCTAT is a distinctive ovarian neoplasm exhibiting morphologic features intermediate between those of the granulosa cell tumor and those of the sertoli cell tumor.[1] About one third of the patients with SCTAT are associated with PJS characterized by mucocutaneous pigmentation and gastrointestinal polyposis.[2] Sporadic SCTATs occur most commonly among women of reproductive age group.[5] Our patient was 49 years and did not have PJS on clinical and radiological evaluation. Other associations reported with SCTAT include adenoma malignum of cervix, Turner's syndrome, dysgerminoma, gonadoblastoma, endometrial carcinoma, and endometriosis of fallopian tube.[1] On histopathological evaluation, there was seen disorderedly proliferative endometrium, tiny endometrial polyp, and intramural leiomyoma in this case. This can be explained as signs of hyperestrinism in present case. However, serum estradiol levels were not performed in this case.

The characteristic histomorphology of variably size round nests of sharply delineated simple and complex tubules containing basement membrane-like material, which may also be present around the tubules helps arriving at an accurate diagnosis. However, gonadoblastoma (seen in indeterminate gonads, composed of germ cells, and sex cord stromal cells distorted by hyalinization and calcification), adult granulosa cell tumor (Call-Exner bodies, tumor cells with coffee bean nuclei), and sertoli cell tumor (tubules lined by columnar cells) may be regarded as histomorphological differentials.[4,6]

SCTATs are known to show immunoexpression of immunohistochemical markers such as inhibin, calretinin, and WT1 which are also seen in most sex cord stromal tumor. SCTAT do not express placental alkaline phosphatase, CD117, and alpha-fetoprotein.[2,5] Thus, characteristic histomorphological features together with IHC could help in arriving at accurate diagnosis and ruling out differentials.

USG remains the most sensitive and cost-effective imaging modality for initial assessment of adnexal masses.[7] Other imaging modalities such as CT, magnetic resonance imaging, and positron imaging tomography scans can be used for better characterization of ovarian SCTAT, detection of extraovarian disease, and identification of other possible primary neoplasms.[7]

SCTAT has been reported in literature to be treated by surgically (salpingo-oophorectomy) in non-syndromic cases. Complete removal is advised in recurrent cases. Prognosis is overall favorable as documented in literature. Other treatment modalities include adjuvant chemotherapy and radiotherapy or combination of surgical treatment and chemotherapy and/or radiotherapy.[2]

Malignant potential of SCTAT cannot be reliably assessed on histologic evaluation as high-risk features such as mitotic rate, lymphovascular invasion, or ovarian surface involvement are not consistently associated with poor outcomes. Malignant SCTAT may have early or late recurrences decades after initial presentation.[4] Thus, regular follow-up of the cases even if regarded as sporadic is warranted.

CONCLUSION

Our experience with the present case highlights the rarity of the lesion, its association with signs of hyperestrinism, awareness of the characteristic histomorphological appearance along with immuno-histopathological studying in arriving at an accurate diagnosis.

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CASE REPORT

A Case of Pancytopenia in a Young Female

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ABSTRACT

Aplastic anemia (AA) is a rare but serious hematologic disorder associated with hematopoietic failure that results due to the decrease or absence of hematopoietic precursor cells in the bone marrow. AA has equal distribution between race and gender, and can occur at any age. Patients usually present with generalized, non-specific complaints, and treatment depends on age, disease severity, donor availability, and clinical and laboratory presentation. Here, we report the case of a 21-year-old woman who presented to the emergency department with vague symptoms of dyspnea, fatigue, pedal edema, and fever, and on further evaluation, a laboratory panel revealed persistent pancytopenia with hypocellular bone marrow and an excellent response to immunosuppressive therapy.

Keywords: Pancytopenia, Aplastic Anaemia, Bone Marrow, Hypocellular

INTRODUCTION

Aplastic anemia (AA) or medullary aplasia is a rare and life-threatening disease, affecting all three hematological cell lines, and is characterized by pancytopenia (leukopenia, anemia, and thrombocytopenia) and bone marrow hypocellularity.[1] AA is more common in Asia than in Western countries.[2] Environmental factors such as drugs, chemicals, viral pathogens, and genetic predisposition play a role in the etiopathogenesis of the disease. Although the disease can occur in any age group, AA shows two incidence peaks: in the young (20–25 years) and the elderly (age of 60 and older).[3,4]

The three main mechanisms of AA are direct trauma, immune dysfunction, and marrow failure.

The most common cause of AA is idiopathic, accounting for 65% of patients.

Seronegative hepatitis accounts for about 10% of cases.[5] AA can also be secondary to viral infections (human immunodeficiency virus, cytomegalovirus, Epstein-Barr virus, parvovirus B19, hepatitis B or C virus), or to physical, chemical, pharmacological (chloramphenicol, sulphonamides, penicillamine, carbamazepine) agents, or ionizing radiation (the effect is dependent on the radiation dose). It can also be associated with pregnancy, or congenital anomalies like Fanconi anemia.[6-9]

AA presents with non-specific symptoms caused by pancytopenia, such as fever with neutropenia, easy fatiguability, exertional dyspnea due to anemia, and mucosal bleeding such as petechiae, heavy menstruation, gingival bleeding due to thrombocytopenia.[10]

In order to establish a diagnosis, bone marrow biopsy and exclusion of malignant or fibrotic infiltration are required. A bone scan can be done to check for bone aplasia or hypoplasia.

In addition, cytogenetic studies such as fluorescence *in situ* hybridization or next-generation sequencing help establish the diagnosis and exclude other hematological abnormalities causing pancytopenia.[10]

Treatment of AA in patients without reversible causes depends on the age of the patient and the severity of the disease. Allogeneic hematopoietic cell transplantation (HCT) should be performed before starting immunosuppressive

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therapy (IST) in young, healthy patients. Patients older than 50 years or incapable of HCT should be started on full-dose IST, including eltrombopag (a thrombopoietin agonist), antithymocyte globulin to deplete antigen-reactive T cells, cyclosporine A inhibiting IL-II, and prednisone that leads to the destruction of immature T-lymphocytes.[10]

Supportive treatment with transfusion of leukoreduced RBCs for hemoglobin <7 mg/dL or platelets <10,000/μL, along with appropriate infection prophylaxis and control are also indicated for patients with AA.

CASE REPORT

A 23-year-old female came with complaints of generalized weakness, decreased appetite, and progressive fatigue for 10 days, along with dyspnea on exertion for 5 days, which was insidious in onset and gradually progressed from NYHA class 1 to class 3, not associated with crepitations/cough/PND/palpitations/chest pain.

She also developed gradually progressive bilateral lower limb pitting edema over 5 days, intermittent low-grade fever, and had one episode of gum bleeding and a single 2 × 3 cm bruise on her right thigh, not associated with trauma. There was no history of yellowish discoloration of eyes or skin, no bleeding from any site, bone tenderness, or weight loss.

The patient was vitally stable, and a general examination revealed the presence of blood on an anterior pillar of the tonsils, and marked pallor in the lower palpebral conjunctiva and nails. There was no angular stomatitis, glossitis or cheilosis, koilonychia/platonychia. Systemic examination was normal and did not reveal palpable hepatosplenomegaly.

INVESTIGATIONS

Hemogram		
Hb-3.9	Neutrophils-25	Hematocrit-11.6
TLC-1370	Lymphocytes-66	MCV-100
Platelets-5000	Eosinophils-4	Mch-33.6
Retic count-0.3%	Monocytes-4	

USG (A +P) and 2D echo did not show any abnormality. Fundus examination revealed bilateral anemic retinopathy and dot blot hemorrhages.

The patient was started on antibiotics and given blood transfusions with little clinical improvement. Blood picture on day 3 revealed- CBC- Hb 7.1, TLC- 1450, and platelet- 18000 A bone marrow examination was done.

BONE MARROW REPORT

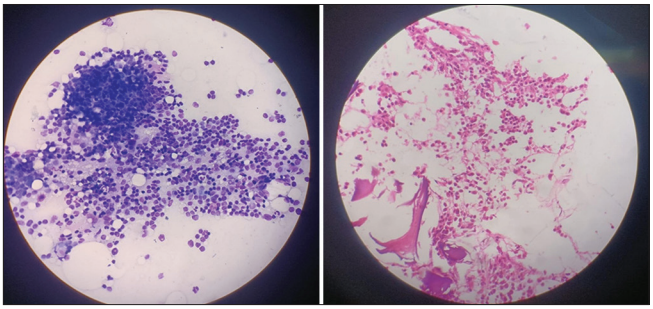
Peripheral Blood Smear Findings

Peripheral blood smear findings revealed predominantly normocytic normochromic RBCs with anisocytosis, leukopenia, thrombocytopenia and few giant platelets.

Bone Marrow Aspiration

Hypocellular bone marrow with M: E Ratio: 1:2. Normoblastic erythropoiesis, normal lymphoid series, and suppressed myeloid series with normal progressive maturation with prominent eosinophilic precursors. Prominent macrophages and mast cells were seen, and no parasites.

- Microscopic examination-3 intertrabecular spaces with intervening trilineage hypocellular hematopoietic tissue, normoblastic erythropoiesis, myeloid suppression with normal progressive maturation. No megakaryocytes/atypical cells/foreign body/granuloma/parasites were seen.



Differential diagnoses of hypocellular marrow were aplastic anemia, hypocellular MDS, and aleukemic leukemia.

LFT	**RFT**		
Tot bilirubin-0.6	Urea-29	S. Tot. Protein-6.2	ICT and DCT-Negative
Direct bilirubin-0.1	Creatinine-0.7	S. Albumin-3.7	PT-14
SGOT-28	Na-138	Dengue and RMT- Negative	INR-1.03
SGPT-29	S. K-4.7		
	S. Cl-103		

As there was hypocellular bone marrow and persistent pancytopenia, the diagnosis of aplastic anemia was made hematologist reference was taken and the patient was started on capsule danazol capsule cyclosporine for 3 months and injection romiplostim for 3 weeks. The patient showed significant clinical improvement on follow-up, with an improved hemogram of Hb-8.2/TLC-5620/platelet count-1,50,000

CONCLUSION

Aplastic anemia is associated with a high prevalence of mortality and morbidity. Every effort should be made for diagnosis, prompt initiation of therapy, and long-term monitoring.

The patient in the discussion responded excellently to injection romiplostim, capsule cyclosporine, and capsule danazol, recovering all three cell lines after 3 months of management, contributing significantly to prognosis and quality of life.

Therefore, a young patient with no significant past medical history and non-specific presentation with pancytopenia can be a case of hematological disorder like severe aplastic anemia, which, on careful evaluation, can be appropriately diagnosed and treated.

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CASE REPORT

A Case of Acyclovir-induced Encephalopathy in a Chronic Kidney Disease Patient

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ABSTRACT

Acyclovir (ACV)-associated encephalopathy is a very rare entity induced by ACV and valacyclovir, a prodrug of ACV. Clinically differentiating herpes zoster encephalopathy from ACV-induced encephalopathy is very challenging. Zoster encephalopathy should improve with ACV, whereas drug-induced neurotoxicity will get worse if the medication is continued. Manifestations of ACV neurotoxicity include altered consciousness, tremor, myoclonus, and dysarthria. However, headache, fever, convulsions, and focal symptoms are rare.

Key words: Acyclovir, Encephalopathy, Herpes zoster.

INTRODUCTION

Abnormalities in cerebrospinal fluid (CSF) analysis and magnetic resource imaging (MRI) are generally not observed in acyclovir (ACV)-induced encephalopathy.[1]

Furthermore, polymerase chain reaction for varicella zoster virus in the CSF plays an important role. Many patients with impaired renal function can present with both herpes zoster and ACV-induced neurotoxicity simultaneously.[2]

Discontinuation of ACV and dialysis will show improvement in 4–5 days. Here, we report a case of ACV-associated encephalopathy in a known case of Stage 5 chronic kidney disease (CKD) after oral ACV administration.

CASE REPORT

A 76-year-old female, a known case of stable CKD Stage 5 with type 2 DM status, presented with complaints of slurred speech, intermittent irrelevant talk, and generalized weakness.

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She had a typical rash of herpes zoster over the right side of her chest and back [Figures 1 and 2], which developed 3 days back. For this, she was started on a Tablet ACV 800 mg TDS along with Tablet pregabalin OD by local practitioner.

She presented after 3 days of starting of above treatment. There was no history of any head trauma, seizure episode, and neurofocal deficit. On admission, her vitals were stable and her renal function test revealed [Table 1].

ABG was normal, and there was no signs of CO_2 narcosis.

With time, her drowsiness increased, so pregabalin was stopped, and dialysis was done to rule out uremic encephalopathy. After the 1st dialysis, the patient showed some improvement not lasting for more than 6 h. Later, she also developed increased neck rigidity along with hypertonia in all joints with areflexia. To rule out CNS infections, specially herpes encephalitis, CSF analysis was done.

CSF study showed only 2 cells/mm^3 with normal proteins and slightly elevated CSF glucose levels [Table 2].

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Table 1: Renal function test

Components	Baseline levels	On admission
Sr. Urea (mg/dl)	80	130
Sr. Creatinine (mg/dl)	4.5	6.1
Sr. Na (mmol/l)	138	136
Sr. K (mmol/l)	4.5	4.9
Sr. Cl (mmol/l)	112	110

Table 2: CSF analysis

CSF study	
Color	Colorless
Appearance	Clear
Cobweb/Coagulum	Absent
CSF - Protein	54 mg/dl
CSF - Glucose	79 mg/dl
Total nucleated cell count	2/mm^3
CSF lymphocytes	100%

Table 3: CSF meningitis panel

Film array meningitis/encephalitis panel	
Organism	**Result**
Escherichia coli	Not detected
H. influenzae	
Listeria monocytogenes	
Neisseria meningitidis	
Streptococcus agalactiae	
Streptococcus Pneumoniae	

Table 4: CSF meningitis panel

Organism	Result
Cytomegalovirus	Not detected
Enterovirus	
Herpes simplex virus - 1	
Herpes simplex virus - 2	
Herpes simplex virus - 6	
Varicella zoster virus	

CSF meningitis panel was also negative [Tables 3 and 4].

Chronic small vessel ischemic changes with atrophy seen on MRI brain.

Diagnosis

Hence, after the exclusion of other differentials, the diagnosis of ACV-induced encephalopathy was considered.

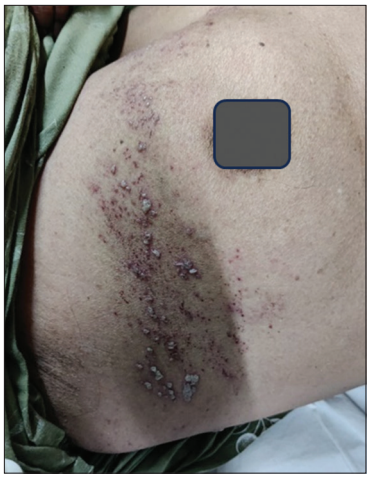

Figure 1: Herpes lesion on chest

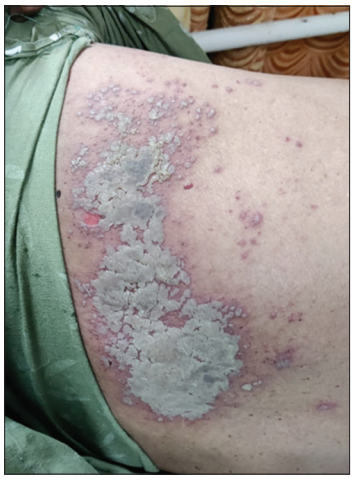

Figure 2: Herpes lesion on back

After 4th day of discontinuation of ACV, the patient started improving gradually. Her consciousness improved with a reduction in rigidity along with an improvement of speech and all higher mental functions.

DISCUSSION

Acyclovir-induced encephalopathy is a rare but serious neurological condition that can occur as a side effect of acyclovir treatment. It typically occurs in patients receiving high doses of acyclovir, especially those with impaired kidney function who may not properly clear the drug from their system.

Encephalopathy occure due to accumulation of acyclovir or its metabolites in the brain, leading to neurotoxicity.

Factors that increase the risk of developing acyclovir-induced encephalopathy include:

- High doses of acyclovir, particularly in patients with renal impairment.
- Prolonged treatment duration.
- Advanced age.
- Underlying neurological conditions.

Diagnosis is primarily clinical, based on the symptoms observed in a patient receiving acyclovir. Imaging studies such as CT scan or MRI of the brain may be performed to rule out other potential causes of encephalopathy. Treatment involves discontinuing acyclovir immediately and providing supportive care. In severe cases, intensive care management may be necessary. Dialysis may be considered in patients with renal impairment to enhance clearance of acyclovir.[3]

CONCLUSION

Acyclovir is used in the treatment of herpes zoster universally. Presented case guide us that we should use Acyclovir cautiously in renal failure patient.

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CASE REPORT

A Case of Drug-induced Hypokalemic Paralysis

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ABSTRACT

Hypokalemic paralysis is a rare condition marked by sudden muscle weakness due to low potassium levels. . Prompt recognition and potassium supplementation led to symptom resolution, emphasizing the importance of early intervention in managing this potentially life-threatening condition.

Key words: Potassium, Hypokalemia , Paralysis

INTRODUCTION

Hypokalemia is characterized by a decline in plasma K+ concentration of <3.5 mEq/L. It can be driven by K+ loss from the kidneys and gastrointestinal system as well as by K+ redistribution between cells.[1] Drugs such as diuretics and steroids are common causes of hypokalemia. The possibility of compensated channelopathies with some trigger is considered an etiology for hypokalemic paralysis.[2]

CASE REPORT

A 73-year-old male presented with no known comorbidities resident of Mahad, Raigad, farmer by occupation, brought by relatives to the casualty in a drowsy, disoriented state with loss of power in all four limbs, and an absent neck holding. The patient was complaining of fatigue five days before admission, the onset of which is sudden with progressive in nature for which he had taken treatment at a local physician (injection of dexamethasone 2cc intramuscular). After which, fatigue improved, but the patient started complaining of pain in the bilateral lower limb.

Pain was associated with a loss of power in the bilateral lower limb while carrying out daily routine activities, which progressed to the bilateral upper limbs and upper body with absent neck holding. With all these complaints, the patient was shifted to casualty status and intubated in view of respiratory distress and airway protection. There is no history of any trauma, radiating pain, headache, diarrhea, nausea, vomiting, or bowel and bladder involvement.

On examination, the patient was drowsy, arousable not oriented to time, place, and person and was vitally stable. On neurological examination, hypotonia was present in all four limbs, with a power of 0/5 in all of them. The plantar reflex was bilateral extensor. Deep tendon reflexes were absent. Pupils of both eyes were mid-dilated and reacting to light. Sensory and cranial nerve examinations were unremarkable.[3] On per abdominal examination, bowel sounds were absent, suggesting hypokalemic paralytic ileus.

The electrocardiogram suggests an inversion of the T wave with prominent U waves in all chest leads (a sine wave pattern) suggestive of hypokalemia [Figure 1].

On Investigations

Hypokalemia with acute kidney injury and normal serum and urinary osmolality were present. As urinary potassium is low, extrarenal causes are considered.

Sr. sodium (mEq/l)	140
Sr. potassium (mEq/l)	1.2
Sr. chloride (mEq/l)	105
Sr. creatinine (mg/dl)	2.5
Sr. osmolality (mOsmol/kg)	319.9

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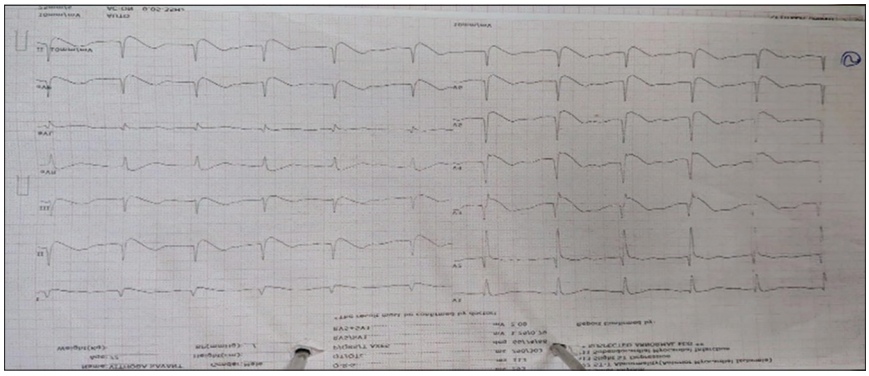

Figure 1: Sine wave pattern ECG

Ur. sodium (mmol)	69
Ur. potassium (mmol)	7.7
Ur. chloride (mmol)	77
Ur. osmolality (mOsmol/kg)	263.1

Perspiration for strenuous exercise
High carbohydrate diet
Injection dexamethasone intramuscular
Gastrointestinal loss
Diuretics
Laxatives

The patient was started on a potassium infusion at a rate of 10 mEq/l with serum potassium monitoring 6 hourly with an oral supplement of potassium-rich liquid diet.[4] Serum potassium (mEq/l) was 1.2 on day 1, which was increased to 3.8 on day 6. With a rise in serum potassium level, tone improved to normal and power improved to 5/5.[5]

DISCUSSION

We hereby present a 73-year-old male patient with hypokalemic paralysis, probably due to iatrogenic or injection dexamethasone. Hypokalemic paralysis was diagnosed due to the symptoms, short duration, and rapid improvement with potassium supplementation. The triggering factor for it was likely an iatrogenic dexamethasone injection.[6,7] Triggers are important for inducing a paralysis attack. The identification of specific triggers and their prevention are important and advised for patients with hypokalemic paralysis. We would like to keep steroids as an unusual precipitating factor while managing patients with hypokalemic paralysis, as per the results of this case study.

CONCLUSION

This case highlights the critical importance of considering hypokalemia as a differential diagnosis in patients presenting with acute muscle weakness. The presence of quadriplegia in this patient underscores the severity and debilitating nature of hypokalemic paralysis. Despite the challenges posed by quadriplegia, timely diagnosis and aggressive potassium supplementation remain paramount in managing this condition. Long-term rehabilitation and supportive care are crucial in optimizing the patient's functional outcomes and quality of life.

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CASE REPORT

Acroangiodermatitis of Mali: A Rare Case Report

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ABSTRACT

Acroangiodermatitis of Mali also known as pseudo-Kaposi sarcoma is an unusual, benign condition. It is clinically presents as purple-colored patches, which can be plaques or nodules. It is located mostly on the extensor surface of lower extremities and seen most commonly in patients with chronic venous insufficiency and arteriovenous malformation. It looks like aggressive conditions like Kaposi's sarcoma. Hence, histopathological examination is required for its diagnosis. Herein, we report a patient who presented with a chronic history of purple-colored papules and plaques over both legs which was later histopathologically confirmed to be case of acroangiodermatitis of Mali.

Key words: Acroangiodermatitis of mali, Arteriovenous malformation, Venous insufficiency

INTRODUCTION

Acroangiodermatitis of Mali is a reactive angiodysplasia of cutaneous blood vessels. It is generally associated with venous insufficiency or with vascular anomalies like klippel–trenaunay syndrome or in amputation stump dermatosis. Chronic history of stasis dermatitis started as violaceous macules and patches. These lesions progress into papules, nodules, and indurated plaques. The lesions are usually located on the lower extremities, mostly bilateral and presented with edema. It is a benign condition but it may resemble other malignant conditions like Kaposi's sarcoma [Table 1]. Hence, histopathological examination is essential for its diagnosis and differentiation.

CASE REPORT

A 36-year-old male presented with multiple dark-colored small lesions over both legs since 1 year associated with itching. There was no other significant history and general examination was unremarkable. Cutaneous examination revealed multiple, well defined, hyperpigmented to violaceous papules with few papules coalescence to form plaques over anterior aspect of bilateral legs [Figure 1]. Similar lesions are not seen elsewhere on the body. Dermoscopic examination showed radially arranged whitish lines on a brownish or reddish background similar to of prurigo nodularis "star burst" pattern [Figure 2]. Histopathological findings showed moderately dense superficial perivascular infiltrate with focal atrophy of overlying epidermis. Within the thickened papillary dermis, there is an increased number of thick-walled capillaries in clustered pattern. These thick-walled capillaries are surrounded by mucin and a moderate perivascular lymphocytic infiltrate with eosinophils occasionally. Minimal amount of hemosiderin deposits seen around vessels [Figure 3].

DISCUSSION

Acroangiodermatitis (other names: pseudo-Kaposi's sarcoma, acroangiodermatitis of Mali-Kuiper, gravitational purpura, stasis purpura) was first coined by Mali in 1965.[1] The condition occurs due to proliferation of pre-existing vasculature which is seen in venous insufficiency, arteriovenous malformation, or acquired iatrogenic arteriovenous fistula. In a study by Mehta *et al*., they reported 2 cases of acroangiodermatitis one of which had venous insufficiency and other had a normal Doppler study.[2]

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Table 1 : The histopathologic differentials for acroangiodermatitis are Kaposi's sarcoma

Differentiating features	Acroangiodermatitis of Mali	Kaposi's sarcoma
Histopathologically	Small dilated vessels lined by plump endothelial cells with hyperplasia of pre-existing vasculature	Slit-like spaces and spindle cell proliferation are independent of the existing vasculature
Periodic Acid Schiff positivity for the vessels	Present	Absent
Factor VIII-associated antigen in the endothelial cells	Present	Absent
Immunolabeling for the CD34 antigen	Positively seen on endothelial cells of hyperplastic vessels	Positively seen on both endothelial cells and the characteristic spindle-shaped perivascular cells
Dermal fibrosis, RBC extravasation, hemosiderin	Present	Present

RBC: Red blood cell

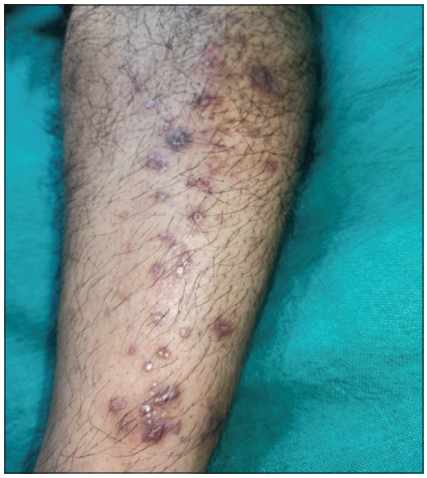

Figure 1: Clinical image: Multiple hyperpigmented to violaceous papules and plaques present over left leg

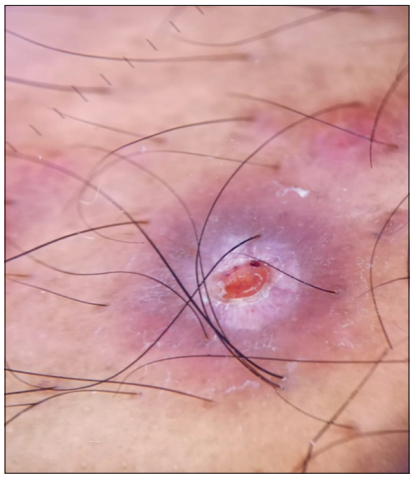

Figure 2: Dermoscopic images: ×10: "Star Burst" pattern

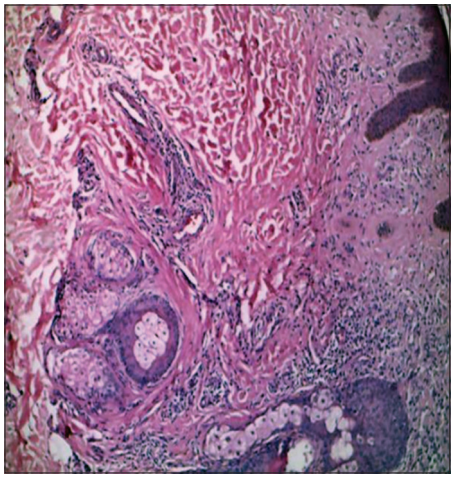

Figure 3: Histopathological image: ×40

Various variants of acroangiodermatitis are:

- Stewart-Bluefarb syndrome: It is a congenital arteriovenous malformation of the lower limb with multiple arteriovenous shunts. It begins in early life and presents with painful purple papules and macules, which can ulcerate. The condition is unilateral and presents over lower extremities.[1]
- Mali type: It is severe form of stasis dermatitis seen in elderly patients. The condition is generally bilateral. It is associated with chronic venous insufficiency. Located on dorsum of feet, hallux, and second toe or on medial aspect of lower limbs. It starts as violaceous macules and patches which gradually progress into soft, non-tender, red-to-purple papules and nodules or indurated plaques.[1]
- Acroangiodermatitis in first pregnancy: It is a gravity purpura also known as Dermite ocre of Favre. Located on lower legs over the site of varicosities of veins which may extend to the dorsa of feet and toes.[3]

- Angiodermatitis occurring after placement of the arteriovenous shunt: It is seen in patients of chronic renal failure and on hemodialysis. Angiodermatitis develops in these patients after placement of the arteriovenous shunt for hemodialysis.[3]

The exact etiology is not known, but it is suggested that severe chronic venous stasis with insufficiency of the calf muscle pump increases the capillary pressure and it leads chronic edema due to which there is chronic tissue hypoxia which causes neovascularization and fibroblast proliferation.[3]

Treatment of acroangiodermatitis contains first correction of the underlying vascular pathology with the help of compression stockings or a compression pump for venous stasis and it is the mainstay of therapy with local wound care for ulcers with daily dressing. Oral erythromycin 500 mg 4 times a day or dapsone 50 mg twice a day for 3 months in combination with compression stocking and pump therapy has been tried with good results.[4] Topical therapy with corticosteroid is also often helpful.[3] Laser ablation, such as pulsed-dye laser, may be useful to clear some localized lesions.[2]

CONCLUSION

In clinical practice, acroangiodermatitis of Mali can be presented in such features which may looks like aggressive malignant conditions like Kaposi's sarcoma. Hence, histopathology should be considered as a gold standard in the differentiation of such similar presenting conditions.

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