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RESEARCH ARTICLE

FOCAL PYOMYOSITIS PRESENTING AS SEPTIC SHOCK.

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Abstract

Introduction:-

Tropical Pyomyositis, usually a disease reported from tropical countries, is characterized by pus formation within skeletal muscles, manifesting as single or multiple abscesses. Scriba in 1885 described this entity for the first time[1]. Levin et al in 1971 reported the first case from a temperate region[2]. Since then many cases have been reported from various geographical regions of the world[3-8]. Tropical pyomyositis is also being increasingly reported from temperate regions .This increase in incidence is attributed to heightened awareness of this disease, an increase in the number of immunocompromised patients, and improvement in diagnostic techniques. Tropical pyomyositis is common throughout the tropics and accounts for 1–4% of all hospital admissions in some tropical countries[2,9].In India it has not been widely reported but there have been sporadic case reports[9,11]. We hereby present a 35 year old lady presenting as the rare disease in Indian subcontinent.

Case:-

A 35 year old otherwise healthy female was admitted in Postgraduate Department of Medicine, SMHS hospital with history of fever for last 5 days which was Continous, associated with sweating and generalized weakness of body without accompanying rigors and chills. She had taken cefixime for 3 days prior to hospital admission and paracetemol 500mg bid for these days without any relief. She did not give any history of altered sensorium , abnormal body movements, headache, urinary/ fecal incontinence, weakness of any part of body, cough/sorethroat, breathlessness, chest pain, hemoptysis/hematemesis, yellowish discoloration of skin, pruritis, abdominal pain/distension, constipation/diarrhea, travel outside, dysuria/hematuria, decreased urine output, swelling of body(generalised/ localised), arthralgias/myalgias, conjuctival congestion/lacrimation, rhinorrhoea/sneezing, tinnitus/vertigo, ear ache/ear discharge, eye discharge, skin rash/skin pigmentation, dysphagia/odynophagia, blood transfusion, similar illness in family, Insect bite or drug allergy. She gives a history of multiple skin eruptions, pustular, non pruritic on her right leg, multiple times in past few months, has taken some oral antibiotics from a local practitioner.

On examination, Patient had GCS-15/15, Pulse: 108/min, regular, feeble , B.P : 80/50 mmHg(supine), Temp:100°F , RR:24 and a normal thorough systemic examination.

Investigations on Day 1 sowed Hb-9.5, TLC-9000, DLC-$P_{80}L_{20}$, Platelet Count-1.6 Lac, Sr. Urea -51mg/dl, Sr. Creatinine-1.5 mg/dl, Na-134 mEq/L, ,K-3.4 mEq/L ,Blood Sugar-86mg/dl , routine urine examination-normal, ECG- sinus tachycardia, chest XRAY – normal, USG abd/Pelvis- normal. Blood gas analysis was showing mild metabolic acidosis.

She was given i.v fluids and was started on dopamine and Ceftriaxone 1gm i.v bd was given and was shifted to MICU. Keeping in view the history and temporal profile of the patient, additional investigations were ordered instantly: urine and blood cultures, Troponin T, D-dimer, Sr. amylase/Sr. lipase, USG Doppler, Sr. cortisol, Xray abdomen standing with both domes of diaphragm. Patient persisted with same condition for next day; however investigations now showed polymorphonuclear leucocytosis with TLC -1960 with 81% neutrophils. Other investigations received were Sr.amylase:70.5U/L, Sr.lipase:35.72(U/L), Sr. cortisol: normal, LFT: 1.9/6.5/3.1/193/249/197, D-dimer: 0.6(<0.3), Echocardiography: normal study ,Trop-t (qualitative): negative. CT angio was normal. Treatment was modified and she was put on noradrenaline infusion in addition to dopamine and piperacillin tazobactam was started empirically with heparin prophylaxis.

On 3rd day, Patient started pain in her right leg on lateral aspect; dull boring type of pain, Continous and didn't interfere with the movements of right leg with no sensory symptoms and no weakness and was associated with slight swelling on lateral aspect of right leg. Parameters recorded were; GCS: 15/15, Pulse: 110/min, B.P: 70/40 mm Hg , R.R: 26/min, U.O: 900ml (12hrs), Temp: 99° and a normal thorough systemic examination. Local examination of right leg showed a small swelling visible on lateral aspect of middle part, slightly reddish , ellipsoid in shape about the size of a coin, with no visible pulsations, no visible scar marks, sinus, varicosity or ulceration seen on the leg and palpation showing a small firm raised area palpable, about 1.5 cm in length and 1 cm breadth, in the anterolateral part of the right leg a few cms above its middle part, local temperature raised as compared to other side, regular surface and well defined margins, tender on deep palpation ,non –compressible ,non-pulsatile, non-fluctuant with no translucency and no bruit or murmur on auscultation.

Xray right leg was normal. USG leg showed a small fluid collection with edematous muscle planes with no evidence of thrombosis. Needle aspiration was done, a small amount of pus was drained and was sent for culture and sensitivity.

Other investigation received were : HIV and Hepatitis serology:negative, T4:4.68(5.1-14.1),T3:29(52-198), TSH: 2.06 (0.4-5.5), CPK:397(24-170)IU/L. Urine and blood cultures were Sterile.

Pus culture and sensitivity showed MRSA isolated sensitive to vancomycin , linezolid , Amikacin and Resistant to ceftriaxone, ceftazidime.

MRI showed altered signal intensity involving right posterolateral soft tissues and calf muscles on T2 weighed sequences with no bony lesions and normal signal void in the vessels suggestive of focal myositis and a final diagnosis of focal pyomyositis was made.

Treatment was modified and besides being on ionotropic support, she was put on Vancomycin and ciprofloxacin and was kept in ICU for two more weeks. She gradually improved and was discharged on 17th day of admission.

Discussion:-

Pyomyositis is an intriguing disease of unclear pathogenesis. Skeletal muscle tissue is intrinsically resistant to bacterial infections under normal circumstances[12]. It is understood that sequestration of iron by myoglobin results in slower growth of bacteria, allowing cellular and humoral defences to enter infected zones and thereby preventing establishment of infection. In a series of autopsied cases of staphylococcus septicemia, abscesses in skeletal muscle were found in fewer than 1% of cases[13]. Even on direct inoculation in the muscles of dogs, staphylococcus failed to produce an abscess[14]. Normal muscle, if damaged, is susceptible to Hematogenous invasion by bacteria with subsequent abscess formation[15]. In 20–50% of cases, a history of blunt trauma or vigorous exercise of the involved group of muscles is forthcoming[16]. Other postulated mechanisms, though not proven, include nutritional deficiencies and viral and parasitic infections. It is postulated that abundant iron is available after trauma, resulting in profuse growth of bacteria.

Predisposing conditions include diabetes mellitus, HIV (human immunodeficiency virus) infection or AIDS (acquired immunodeficiency syndrome), malignancy, chronic liver diseases, and intravenous drug abuse

The disease is seen in all age groups, although young males are the most susceptible group. Maximum incidence is seen at 10–40 years of age with a male to female ratio of 1.5:1. Muscles frequently involved are quadriceps, glutei, pectoralis major, serratus anterior, biceps, iliopsoas, gastrocnemius, abdominal and spinal muscles. Usually, a single group of muscle is affected, but in 12–40% of cases multiple groups are involved either sequentially or simultaneously[5,6,9].

Primary pyomyositis has three distinct stages, which represent a gradual progression from diffuse inflammation to focal abscess formation to a septic state [5,17,,18,19,20,21].

Stage1: Primary pyomyositis typically begins with the insidious onset of dull, cramping, progressive pain associated with a low-grade fever, general malaise, and muscle ache. Sealed by the muscle aponeurosis and deep fascia, an inflammatory process within a muscle does not reach the subcutaneous tissues and overlying epidermis in the early course of the infection. As a result, there is a short period, usually one or two weeks, during which local signs of inflammation are absent but pain and even systemic signs of infection are present.

Stage 2: Muscle abscess formation, the stage during which most patients are first seen, is associated with local and systemic manifestations of infection. A tender soft-tissue mass can be palpated, and the affected muscle is typically described as having a firm, wooden texture. The overlying skin is swollen, erythematous, and warm. The patient may have exquisite tenderness and fluctuance of the involved area, obvious functional disability, and occasionally frank septicemia.

Stage 3: If pyomyositis is not treated in the second stage, it may progress to the third stage, which includes signs of toxicity and septic shock. This late stage of the disease is characterized by severe pain, local signs of infection, and systemic manifestations of sepsis, all of which require urgent intervention.

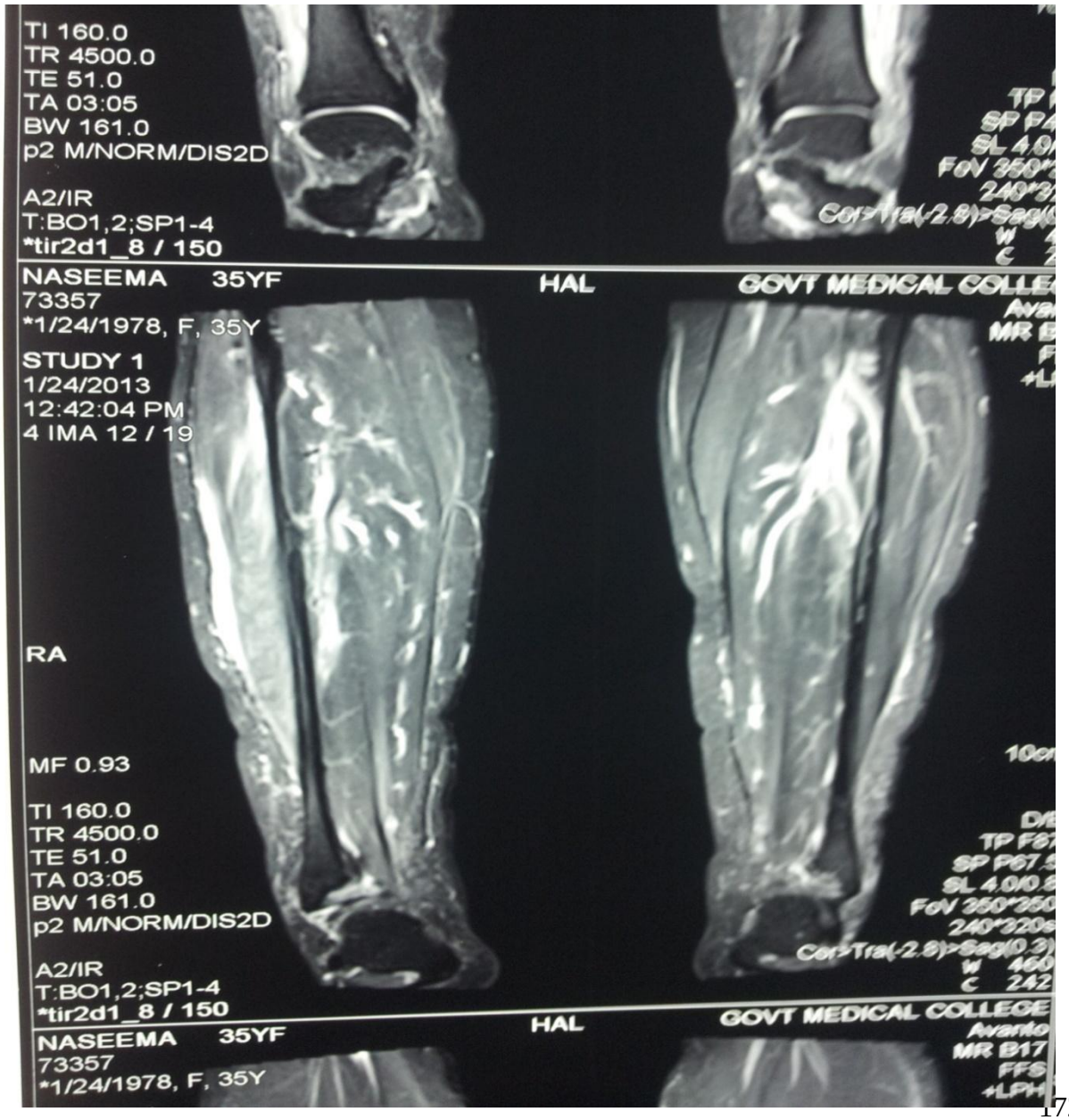
Because of its indistinct clinical manifestations, pyomyositis has been confused with muscle strain, thrombophlebitis, cellulitis, bursitis, contusion, hematoma, Perthes disease, septic arthritis, osteomyelitis, rheumatoid disease, and soft-tissue sarcoma[22,23,24]. Pyomyositis of the iliopsoas or involvement of the abdominal wall, especially the right lower quadrant, may be confused with acute appendicitis and lead to unnecessary laparotomy[22]. A comprehensive medical history, meticulous physical examination, and judicious use of imaging studies can establish the diagnosis in the majority of patients.

Routine laboratory evaluation is rarely helpful in the diagnosis of pyomyositis, as recognition of this condition is based on the clinical presentation and the results of imaging studies. Leukocytosis with a left shift and an elevated sedimentation rate are common[6,23]. Eosinophilia was reported in association with pyomyositis in the tropics, an observation that led to the hypothesis that these patients had an underlying parasitic infection[6]. However, eosinophilia has been rarely reported in North American patients with pyomyositis[21].

Serum levels of muscle enzymes are generally normal[25]. A review of the findings in 144 patients reported on in eight studies showed that cultures of blood and of purulent material are positive for only 16 to 38% and 21%to 41% of the patients, respectively[21,25.]

Ultrasound is the initial screening tool for reasons of economy and easy availability. Hypoechoic areas with an increase in muscle bulk are seen on Ultrasound[26].Computed tomography/magnetic resonance imaging (MRI) are the best imaging techniques for early diagnosis[27]. Computed tomography shows areas of low attenuation with loss of muscle planes and a surrounding rim of contrast enhancement as characteristic of pyomyositis. Computed tomography is also useful in differentiating tumours, haematoma, and thrombophlebitis from abscess[28].

However, at times a computed tomogram alone may be unreliable in distinguishing muscle abscess from swollen muscles[29].MRI shows hyperintense rim on T1 weighted images with peripheral enhancement on gadolinium DTPA scan[26]. Gallium scintigraphy is an extremely sensitive tool, but its inability to provide anatomic details, lack of specificity, and high cost reduces its usefulness. It is best reserved for a group of patients in which clinical suspicion is high but computed tomography/MRI is inconclusive. It is also utilised for detecting unsuspected but possible metastatic abscess[17]. Once diagnosis is established, attention should turn to aggressive management. Surgical debridement and drainage, accompanied by parenteral antistaphylococcal b-lactamase resistant penicillin (cloxacillin 1–2 g every six hours), is the initial recommended treatment. Diffuse myositis without abscess may respond to an antimicrobial agent alone but abscess may develop eventually requiring drainage. In most clinical settings, treating S. aureus infection with more than one drug, to which the organism is known to be susceptible, attains no significant benefit. Penicillin is the drug of choice for infections caused by penicillin susceptible staphylococcus. Drug combinations of penicillin with b-lactamase inhibitor are also effective but are best reserved for treatment of mixed infections. In case of penicillin allergic subjects, first generation cephalosporin (cefazolin) may be preferred for reasons related to cost, potency, and breadth of spectrum. For methicillin resistant staphylococcus, vancomycin in a dose of 15 mg/kg to a maximum of 1 g, given every 12 hours is a suitable alternative. Another glycopeptide, teicoplanin, in a dosage of 400 mg/day in a single dose is equally effective. For vancomycin intermediate sensitive staphylococcus, linzolid or dalfopristine-quinapristine are effective alternatives[30].



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