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Symmetric peripheral gangrene secondary to sepsis

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Abstract

Symmetrical peripheral gangrene (SPG) is characterized by sudden onset of peripheral, frequently symmetrical, gangrene in the absence of major vascular occlusive disease with a high mortality rate. [1] We report a case of Upper limb SPG caused by septic shock in MODS with acute myocarditis.

It had been treated with inotropes.

This case shows that SPG may be present as a complication of sepsis due to systematic derangement that affects a wide range of organ systems, including coagulation and microcirculation.

Early recognition and prompt management of sepsis and optimization of the process of weaning off the inotropes at the earliest opportunity are necessary to avoid SPG.

Keywords: symmetric peri pheral gangrene, MODS, sepsis, myocarditis.

Introduction

Symmetric peripheral gangrene is a rare clinical syndrome characterized by symmetrical distal ischemic damage leading to gangrene of two or more sites in the absence of large vessel obstruction or vasculitis, sometimes used synonymously as purpura fulminans. Disseminated intravascular coagulation (DIC) and hemorrhagic infarction of skin with uninvolved proximal arteries are hallmark of this condition.[2]

Case study

A 30-year-old female, with no history of co morbidities presented to EMD with complaints of fever and breathlessness. Clinical examination revealed pulse rate of 106 bpm, BP-110/70 mm Hg, saturation -86% at room atmosphere, RR-38cpm, febrile, normal random blood sugar levels, pallor present. Systemic examination of RS showed bilateral rhonchi were present and other

systemic examination normal. patient was intubated in EMD I/V/O tachypnea and respiratory distress type 1.

Investigations

ECG Shows poor R wave progression.

SOB profile: CKMB:60.60g/ml, MYO:329 ng/ml, TROP-I:7.32ng/ml, BNP-222pg/ml, DDIM:3230ng/ml.

2d echo-normal chamber dimensions, no rwma, (lvef-55%), adequate lv systolic function, thin ias, mild Mr. Mild ar, mild pr. Mild tr, no clot, no veg for the above complaint's patient was treated with antibiotics, IV fluids, bronchodilators.

Investigations	DAY 1	DAY 2	DAY3	DAY5	DAY 9	DAY 20
HB(g/dl)	7.6	6.6	6.80	7.80	8.10	9.8
TC (per cumm)	5.54	9.14	17.55	13.70	15.37	7.51
PLT (lakh)	50	73	76	88	399	345
BU (mg/dl)	38	42	70	44	45	23
SC (mg/dl)	1.0	0.9	0.9	0.8	0.7	0.7

Liver function test

	TB	DB	SGOT	SGPT	ALP	ALB	A:G	TP
DAY 1	2.29	1.83	110	43	119	2.2	1.0	4.6
DAY 5	1.3	1.2	208	109	131	2.3	1.1	5.9

chest x ray

DAY 1: B/L diffuse non homogenous opacities left & right

DAY 3: B/L diffuse non homogenous opacities left<right

DAY 6: B/L diffuse non homogenous opacities left (resolved), right (present)

On 2nd day evening patient started with inotropes i/v/o hypotension. repeat Chest x ray suggestive of acute pulmonary edema and Inj.

furosemide 5mg/hrwas started. On day 3 patient developing bluish discoloration of digital tips of both upper limbs. Lipid profile was with in normal limits, we changed to higher antibiotics for raising total leukocyte counts.

HbA1C	S.Phosphorous	S. Calcium	S.Magnesium
5%	6.3mg/dl	10.6 mg/dl	1.5 mg/dl

On examination all peripheral pulses were palpable, c-reactive protein is positive, erythrocyte sedimentation ratio is 150mm/hr, PT 22.3sec (control 14sec) and INR 1.93aPTT31.6sec (control 28sec). urine routine showing traces proteins and 1-2 pus cells in urine, S.LDH-843.

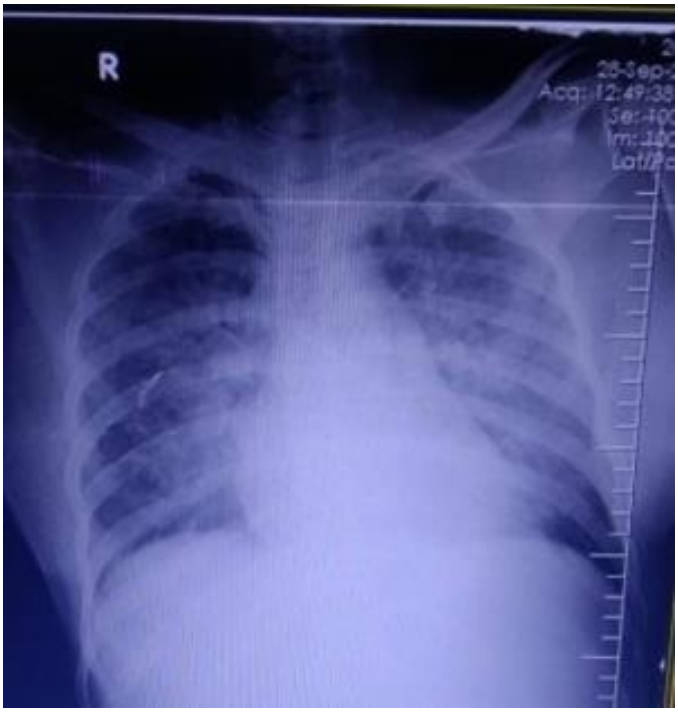

Figure 1: Day 1 : B/L diffuse NHO on left & right.

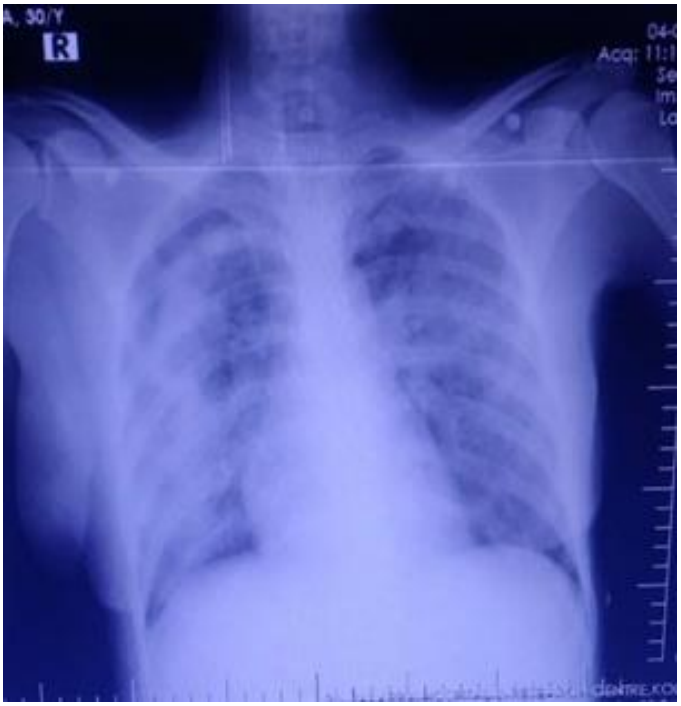

Figure 2: Day 3: B/L diffuse NHO on left<right.

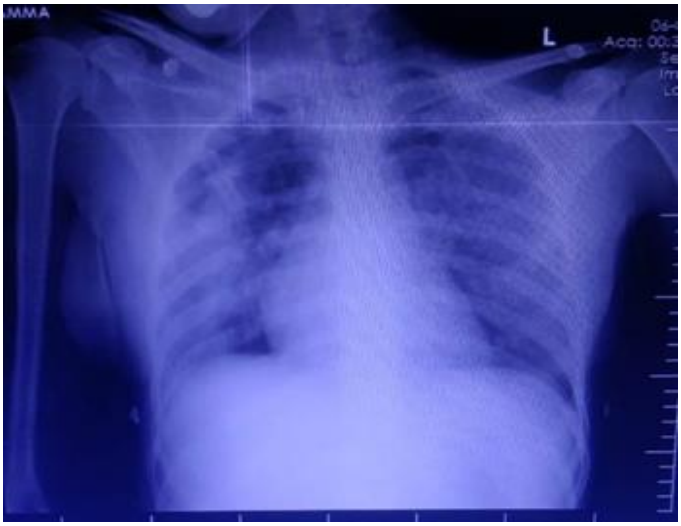

Figure 3: Day 6:B/L diffuse non homogenous opacities left (resolved), right (present)

Further investigations: Ultra sound abdomen showing normal size kidneys and echotexture

Arterial color Doppler study of bilateral upper limb:

Right upper limb (Axillary artery, Brachial artery, Radial artery, Ulnar artery): shows triphasic spectral flow with normal PSV

Left upper limb (Axillary artery, Brachial artery, Radial artery, Ulnar artery): shows triphasic spectral flow with normal PSV subclavian artery could not be assessed due to un cooperative patient.

ANA screening done anti-nuclear antibody levels 1.37 units(negative), then we switched to meropenem antibiotic on day 2.LFT returned to normal, leukocyte count return to normal on day 10. Urine culture and blood culture sensitivity showing no growth after 48 hrs of incubation.

Patient was diagnosed as mods (ards, sepsis, thrombocytopenia, acute liver injury) and acute pulmonary edema secondary to acute myocarditis. On the aggressive management, the patient recovered on day 6 and extubated and shifted to ward on hospital day 10.

Images

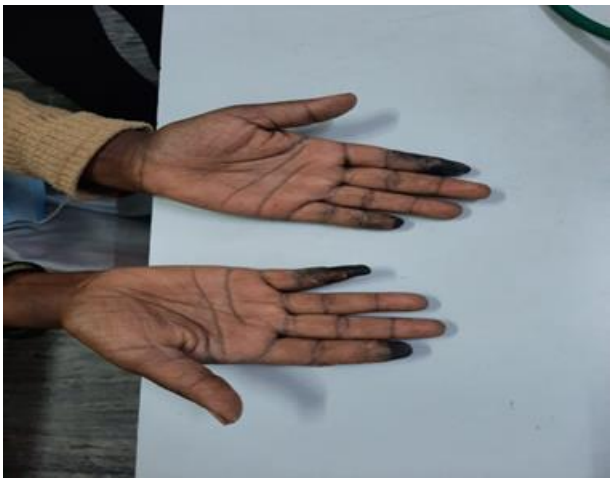

Figure 4: Showing B/L symentrical gangrene of upper limb.

Discussion

The exact pathogenesis is unknown but low flow states coupled with endothelial damage in association with hypercoagulable state leads to micro- circulation occlusion. The etio logies could be bacterial, viral or parasitic infection versus non infective causes like cardiogenic shock, massive pulmonary embolism, and use of vasopressors, paraneo plastic syndrome, auto immune disorders and various malignancies.

Aggravating factors include asplenia, immuno suppression, and previous cold injury to extremities, diabetes mellitus, renal failure, increased sympathetic tone and use of vasopressors.

By far bacterial septicemia is the most frequent cause and lead to considerable morbidity and mortality. DIC is associated in majority of cases and is the final common pathway of micro- circulation occlusion.

SPG should be suspected at the first sign of marked coldness, pallor, cyanosis or pain in the extremity, as the condition can progress rapidly to acrocyanosis and, if not corrected to gangrene. The ischemic changes begin distally but soon may progress proximally to involve the whole limb.

These changes are associated with intact distal pulses as confirmed by Doppler study because the large vessels are often spared. The peripheral extremities, tip of the nose, scalp and genitalia are areas most commonly affected. Patients usually have features of hypotension and shock. The laboratory investigation is directed towards determining the underlying cause. It may include septic screen, DIC and auto antibody panel and evaluation of arterial circulation with Doppler's.

Pathologic examination of amputated specimens often reveals thrombi concentrated in the small vessels with sparing of large vessels. The treatment is supportive and is focused to the underlying etiology.

No treatment is universally effective. Early recognition and immediate discontinuation or reduction, if possible, of vasopressor therapy (as it aggravates the low-flow state by enhancing vasoconstriction) and vigorous therapy of sepsis and DIC with intravenous antibiotic therapy and heparinization are essential components of SPG management [3].

In extreme cases amputation of gangrenous area may become necessary to save life.

Conclusion

SPG carries a high morbidity and mortality. A high index of suspicion and prompt management with usual measures may limit the progression of gangrene, limb saving and lifesaving, Here we present a case to SPG occurring in patients with Bronchopneumonia with sepsis with MODS with Septic shock.

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