JOURNAL OF CLINICAL AND BIOMEDICAL SCIENCES

CASE REPORT

OPEN ACCESS

Received: 18.03.2024
Accepted: 06.07.2024
Published: 24.07.2924

Citation: Mani MRKP, Srinivasa SV. Symmetric Peripheral Gangrene Secondary to Sepsis. J Clin Biomed Sci 2024; 14(2): 56-58. https://doi.org /10.58739/jcbs/v14i2.23.12

* **Corresponding author**.

Manimohanhp@gmail.com

Funding: None

Competing Interests: None

Published By Sri Devaraj Urs Academy of Higher Education, Kolar, Karnataka

ISSN
Print: 2231-4180
Electronic: 2319-2453

Symmetric Peripheral Gangrene Secondary to Sepsis

Mani Mohan Reddy K P[1]*, Srinivasa S V[2]

1 Postgraduate, Department of General Medicine, Sri Devraj Urs Academy of Higher Education and Research, Kolar, Karnataka, India
2 Professor & HOU, Department of General Medicine, Sri Devraj Urs Academy of Higher Education and Research, Kolar, Karnataka, India

Abstract

Symmetrical peripheral gangrene (SPG) is described as rapid onset of peripheral and symmetrical gangrene in the lack of large vessel occlusive disease with high mortality rate.[1] Hereby reporting an event of Upper limb symmetrical gangrene caused by septic shock in MODS, Acute myocarditis and was managed by inotropes. The case shows Symmetrical peripheral gangrene as a complication of sepsis owing to systematic imbalance that disturbs the organ dysfunction mainly coagulation and microcirculation. Primary recognition and quick management of sepsis and weaning off the inotropes at the initial opportunity are essential to evade Symmetrical peripheral gangrene.

Keywords: Symmetric peripheral gangrene; MODS; Sepsis; Myocarditis; Inotropes

1 Introduction

SPG is a rare condition categorized by ischemic damage distally leading to symmetrical gangrene of digits with absence of large vessel vasculitis or obstruction. Disseminated intravascular coagulation (DIC) and hemorrhagic infarction of skin where proximal arteries are not involved as trademark in this condition.[2]

2 Case Study

A 30yr elderly female, with no history of co morbidities came 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.

- **Physical examination**

Only pallor present.
N/k/c/o smoker, alcohol.

2.1 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.

Malaria, leptospira, dengue: negative.

Table 1. Case parameters

HB (g/dl)	7.6	6.6	6.8	7.8	8.1	9.8
TC (per cumm)	5.54	9.14	17.55	13.7	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	0.9	0.9	0.8	0.7	0.7

Table 2. L iver 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. (B/l bronchopneumonia)

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 Bronchopneumonia and Inj. furosemide 5mg/hr was 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.

Table 3. Clinical parameters

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

On examination all pulses which are peripheral are palpable, c-reactive protein is positive, erythrocyte sedimentation ratio is 150mm/hr, PT 22.3sec (control 14sec) and INR 1.93 aPTT 31.6 sec (control 28 sec), urine routine showing traces proteins and 1-2 pus cells in urine, S.LDH-843.

- **Further investigations**

Ultrasound abdomen showing normal size kidneys and echotexture. Arterial color Doppler study of bilateral upper limb

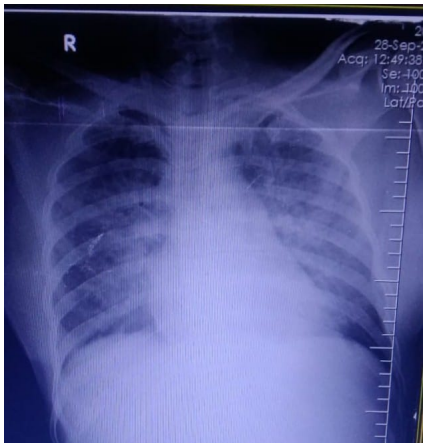

Fig 1. Day 1: B/L diffuse NHO on left & right

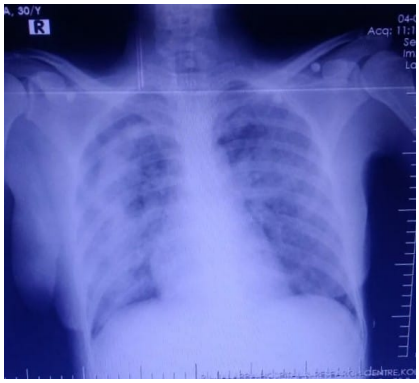

Fig 2. Day 3: B/L diffuse NHO on left < right

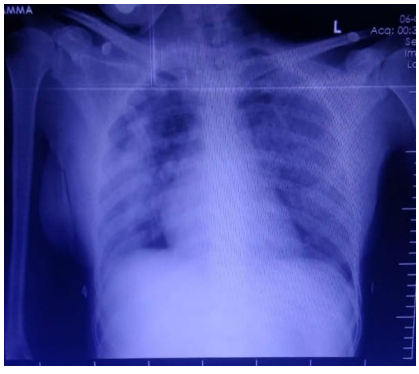

Fig 3. Day 6: B/L diffuse non homogenous opacities left (resolved), right (present)

shows triphasic spectral flow with normal PSV. Subclavian artery could not be assessed due to uncooperative patient.

ANA screening done and 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 Bronchopneumonia. On the aggressive management, the patient recovered on day 6 and extubated and shifted to ward on hospital day 10.

Fig 4. Image showing B/L symentrical gangrene of upper limb

3 Discussion

The mechanism under this condition is unknown but some studies show endothelial damage which is associated with hypercoagulable condition leading to occlusion of microcirculation.[3] The causes might be bacterial, parasitic, viral infection or other conditions like vasopressors usage, cardiogenic shock, paraneoplastic syndrome, pulmonary embolism, and malignancies.[2] Aggravating factors like decreased immunity condition, Absent spleen, old cold injuries to digits, renal failure, diabetes mellitus, increased sympathetic tone and vasopressors usage.[4,5]

Most of the cases are caused by bacterial septicemia are leading to death and diseased. DIC is related to majority of cases and common mechanism is occlusion of microcirculation.[6]

We should suspect immediately when digits undergo coldness, cyanosis, pain, pallor in the extremities, as the disorder rapidly progressive to cyanosis in digits and gangrene. As ischemic changes start distally and progress to the whole limb.[1]

These features are associated with intact distal pulses and sparing of large vessels which are ruled out by Doppler study. The commonly affected areas are distal extremities, Scalp, Tip of the nose and genitalia and having the condition of shock and hypotension. Lab investigations are focused on the underlying condition.

The amputated specimen under staining shows thrombi obstructing the small vessels and sparing the large vessels. The treatment should be focused on primary etiology.

There is no treatment which is effective. Recognition and early discontinuation of vasopressor therapy and active management towards the sepsis and DIC are the main mechanisms towards SPG management.[6]

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

4 Conclusion

Symmetrical peripheral gangrene (SPG) is the condition where there is high case mortality and morbidity. We have to highly think about SPG and rapid management towards SPG to save the life and limb saving from gangrene.[7]

References

1) Parmar MS. Symmetrical peripheral gangrene: A rare but dreadful complication of sepsis. *Canadian Medical Association Journal*. 2002;167(9):1037–1038. Available from: https://www.cmaj.ca/content/cmaj/167/9/1037.full.pdf.
2) Shimbo K, Yokota K, Miyamoto J, Okuhara Y, Ochi M. Symmetrical peripheral gangrene caused by septic shock. *Case Reports in Plastic Surgery and Hand Surgery*. 2015;2(3-4):53–56. Available from: https://doi.org/10.3109/23320885.2015.1041529.
3) Davis MDP, Dy KM, Nelson S. Presentation and outcome of purpura fulminans associated with peripheral gangrene in 12 patients at Mayo Clinic. *Journal of the American Academy of Dermatology*. 2007;57(6):944–956. Available from: https://doi.org/10.1016/j.jaad.2007.07.039.
4) Hayes MA, Yau EH, Hinds CJ, Watson JD. Symmetrical peripheral gangrene: association with noradrenaline administration. *Intensive Care Medicine*. 1992;18(7):433–436. Available from: https://doi.org/10.1007/bf01694349.
5) Colak T, Erdogan O, Yerebakan O, Arici C, Gurkan A. Symmetrical peripheral gangrene and dopamine. *Ulus Travma Acil Cerrahi Derg*. 2003;9(3):222–224. Available from: https://pubmed.ncbi.nlm.nih.gov/12923702/#:~:text=The%20SPG%20syndrome%20consists%20of,contributory%20cause%20in%20many%20cases.
6) Ghosh S, Bandyopadhyay D, Ghosh A. Symmetrical peripheral gangrene: A prospective study of 14 consecutive cases in a tertiary-care hospital in eastern India. *Journal of the European Academy of Dermatology and Venereology*. 2010;24(2):214–218. Available from: https://doi.org/10.1111/j.1468-3083.2009.03329.x.
7) Tripathy S, Rath B. Symmetric peripheral gangrene Catch it early! *Journal of Emergencies Trauma and Shock*. 2010;3(2):189–190. Available from: https://journals.lww.com/onlinejets/fulltext/2010/03020/symmetric_peripheral_gangrene__catch_it_early_.18.aspx.