

Septicaemia induced acute heart failure with elevated Trop I – a case report in Fourniers gangrene.

Introduction: Infection induced acute heart failure is sometimes remained undiagnosed cause of death in hospital admission. Any sort of initial systemic infection caused greatly increased oxygen consumption, decrease microvascular circulation and decrease oxygen delivery to heart, causing release of Trop i in systemic circulation. Severe Sepsis with acute heart failure in previous healthy heart patient has mimicking raised troponin like acute MI.

Abstract: A 42 yrs Diabetic, H/O healthy cardiac status male patient presented as scrotal abscess admitted in surgery ward and debridement done as a case of Fourniers gangrene under regional anaesthesia. Patient had symptoms of cough, dyspnoea, frothy secretions, high color urine and episodes of desaturation preoperatively. 4 hours following debridement patient develops marked dyspnoea, chest heaviness with increased frothy secretions and shifted to intensive care unit. **In critical care comprehensive management patient symptomatically improved after 3 days of ICU care and shifted to ward.**

**Keywords: Acute heart failure, Severe sepsis, elevated trop I, Fourniersgangrene .**

Case report : A 42 yrs Male, Diabetic had a history of 3 days of pain in lower scrotum and perineal area. On admission patient diagnosed as scrotal abscess and underwent surgery with regional anaesthesia. On exploration it was gangrenous and labeled as Fourniers. Preoperatively patient was febrile, mild dyspnoeic, frothy secretions and high colored urine. Perioperatively patient was desaturated and managed with high flow of oxygen. Following 4 hrs of Wide local excision and debridement of devitalised tissues, patient status was deteriorated, worsening dyspnoea, frothy secretions and restlessness. Immediately shifted for intensive care and parameters reveals ABG-mild respiratory alkalosis, markedly raised TLC(20,140/cumm), CRP(157 mg/l), **chest x ray non significant**, HBA1C(12.7). D dimer-6928.21 ng/ml, **pro BNP -2905 pg/ml (high probability of heart failure > 450)**. his urinary ketones also was positive. **Trop i 27.2ng/ml (<19 ng normal and acute MI >100)** and dyselectrolytaemia. Screening echocardiogram was non significant. **By comprehensive critical care including Enoxaparin, mild diuretics and collaboration of surgery team, patient status improved gradually and shifted to ward.**

Discussion : In Hospital admitted patient morbidity and mortality review, sepsis induced acute heart failure is a great contribution though we sometimes overlook. **In critically ill patient with sepsis the results are difficult to interpret and in evolutionary medicine. Pulmonary edema and fluid overload, pulmonary embolism is a major part in perioperative period eventful recovery. Previous healthy**

individual sudden respiratory complaints with severe sepsis , acute heart failure should kept in mind even in remote thinking.

Conclusion: Infection induced acute heart failure should kept In mind in treating sepsis. fluid overload , Pulmonary embolism is also not always for respiratory complains in perioperative sepsis. Definitive pathophysiology of acute heart failure in infection will provide basis for further definitive therapeutic strategies. Elevated trop I is always not the cause of acute MI in sepsis.

## References

- 1 Farmakis D, Parissis j, karavidas A et al (2015) In hospital management of acute heart failure : practical recommendations and future perspectives . int J Cardiol 201:231-236. <https://doi.org/10.1016/j.ijcard.2015.08.030>
2. Farmakis D, Parissis J, Lekakis J, Filipatos G (2015) Acute heart failure: epidemiology, risk factors, and prevention. Rev Esp Cardiol (Engl. Ed) 68(3):245-248. <https://doi.org/10.1016/j.rec.2014.11.004>
3. Alon. D. Stein GY, Korenfeld R, Fuchs S (2013) predictors and outcomes of infection –related hospital admission of heart failure patients. PLoS ONE 8(8):e72476. <https://doi.org/10.1371/journal.pone.0072476>
4. Shirazi LF, Bissett J, Romeo F, Mehta JL (2017) Role of inflammation in heart failure. Curr Atheroscler Rep 19(6):27. <https://doi.org/10.1007/s11883-017-0660-3>
5. Kakihana Y, Ito T, Nakahara M, Yamaguchi K, Yasuda T (2016) Sepsis- induced myocardial dysfunction: pathophysiology and management. J Intensive Care 4:22. <https://doi.org/10.1186/s40560-016-0148-1>
6. Minasyan H (2017) Sepsis and septic shock: pathogenesis and treatment perspectives. J Crit Care 40:229-242. <https://doi.org/10.1016/j.jcrc.2017.04.015>
7. Marik PE, Linde-Zwirble WT, Bittner EA, Sahatjian J, Hansell D (2017) Fluid administration in severe sepsis and septic shock. Patterns and outcomes; an analysis of large national database. Intensive Care Med 43(5):625-632. <https://doi.org/10.1007/s00134-016-4675y>

8.Parker MM , Shelhamer JH, Bacharach SL et al (1984) profound but reversible myocardial depression in patients with septic shock. Ann Intern Med 100(4):483-490. <https://doi.org/10.7326/0003-4819-100-4-483>

9.Hotchkiss RS, Karl IE (1992) Reevaluation of the role of cellular hypoxia and bioenergetics failure in sepsis. JAMA 267(11):1503-1510.