LETTER TO EDITOR

Staphylococcal Pyopericardium: A Rare and Fatal Complication Following a Common Viral Disease

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A 30-year-old male was presented to emergency department with 2 days history of high-grade fever and excruciating chest pain associated with chills and rigors. The pain was located in mid-chest and radiating to the left back. He is nonsmoker, nonalcoholic with no known comorbidity. Ten days earlier he had dengue seropositive (NS1 Ag) febrile illness and was discharged from hospital once fever subsided.

On admission, patient had BP of 140/90 mm of Hg, pulse rate 120/min (sinus rhythm), SpO₂-98% on ambient air, and temperature of 102°F. Systemic examination showed chest was clear, heart sounds were normally audible, and no jugular venous engorgement. His chest X-ray on admission was normal. However on second day after admission, he developed respiratory distress requiring oxygen supplementation. A pericardial rub was audible and electrocardiogram showed ST changes. X-ray chest showed symmetrical enlargement of cardiac silhouette suggestive of pericardial effusion. 2D-echo confirmed the diagnosis of pericardial effusion (anterior 16 mm, posterior 12 mm, lateral 13 mm, RV anterior 7 mm). HRCT (high-resolution computed tomography) chest showed left-sided

Figs. 1A and B: (A) X-ray chest showed symmetrical enlargement of cardiac silhouette suggestive of pericardial effusion (inverted water-bottle sign). (B) 2D-echo image showing moderate pericardial effusion (anterior 16 mm, posterior 12 mm, lateral 13 mm, RV anterior 7 mm)

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therapy, antibiotic was switched to oral cloxacillin 1 gm 6 hourly and continued for 4 weeks. The patient recovered completely during this period (Figs. 1 to 3).

Staphylococcal infection following dengue is infrequently reported.1–3 One of the proposed mechanisms to explain coinfections in dengue is the damage of endothelial barrier rendering it possible to enter pathogens into circulation.4 Acute pyopericardium is a potentially life-threatening illness associated with a mortality of up to 50%, with the majority of deaths due to cardiac tamponade (Table 1).

In our case, the diagnosis was made by echocardiography and biochemistry of aspirated pus from pleural and pericardium.

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References
Table 1: Pericardial and pleural fluid analysis has shown predominantly neutrophilic leukocytosis

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L, lymphocyte; N, neutrophil; TLC, total leukocyte cell
