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JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Case Report

Open Access, Volume 6

Point-of-care ultrasound (PoCUS) for the diagnosis of purulent cardiac tamponade in Benin: A case report

Attannon Arnauld Fiogbé[1]*†; Veronique Suttels[2]†; Ablo Prudence Wachinou[1]; Brice Guendehou B[1]; Alovokpinhou Frederique[1]; Capo-chichi D[1]; Noémie Boillat-Blanco[2]; Opota O[3]; Annie Hartley[4]; Gildas Agodokpessi[1]

[1]National Teaching Hospital for Tuberculosis and Respiratory Diseases (CNHU-PPC), Cotonou, Benin.

[2]Infectious Diseases Department, Lausanne University Hospital (CHUV), 1011, Lausanne, Switzerland.

[3]Institute of Microbiology, Lausanne University Hospital (CHUV) and University of Lausanne (UNIL), 1011 Lausanne, Switzerland.

[4]Intelligent Global Health Research Group, Swiss Intitute of Technology (EPFL), 1015 Lausanne, Switzerland.

†Equal contribution.

***Corresponding Author: Fiogbe AA**

National Teaching Hospital for Tuberculosis and Respiratory Diseases (CNHU-PPC), Cotonou, Benin.

Tel: 0022967183933; Email: afiogbe@yahoo.fr

Received: Jun 24, 2025

Accepted: Jul 21, 2025

Published: Jul 28, 2025

Archived: www.jcimcr.org

DOI: www.doi.org/10.52768/2766-7820/3706

Abstract

We report a rare case of massive purulent pericarditis with cardiac tamponade in a 19-year-old immunocompetent patient, diagnosed using portable Point-of-Care Ultrasound (PoCUS). The ultrasound revealed a swinging heart, right ventricular collapse, and a large pericardial effusion with a positive plankton sign-features consistent with purulent pericarditis. Ultrasound-guided pericardiocentesis yielded abundant, viscous yellow pus. Microbiological analysis identified Staphylococcus aureus as the causative organism. This case highlights the critical role of PoCUS in the timely diagnosis and management of pericardial effusions, particularly in resource-limited settings.

Keyword: PoCUS; Purulent pericarditis; Benin.

Introduction

Point-of-Care Ultrasound (PoCUS) is becoming an increasingly accessible skill, decentralizing to non-specialist health workers to guide routine clinical decision making. The advent of ultrasound-on-a-chip has transformed the technology into an affordable and portable pocket-sized mobile health device, while retaining the versatility and diagnostic performance of their costly and cumbersome predecessors [1]. The modest consumables, low maintenance and ease-of-use, make PoCUS an attractive skill in resource-limited settings. PoCUS is well known to expedite the accurate diagnosis of many cardiopulmonary conditions and is particularly useful for the detection of pericardial effusion [2,3]. In high-income countries, bacterial pericarditis is diagnosed in approximately 1 in 18,000 hospitalized patients and mostly limited to small fluid collections diagnosed by ultrasound and microbiology [4,5]. Indeed, massive purulent effusions are seldom encountered since the antibiotic era. The prevalence and severity of this condition are expected to be much higher in TB-endemic regions, and to increase with growing antibiotic resistance.

We present a rare case of massive purulent pericarditis with tamponade in an immunocompetent West-African adult, diagnosed with handheld ultrasound.

Case presentation

A 19-year-old male from a remote rural region of Benin presented to the local TB referral hospital with dyspnea, lower limb oedema and shock. The dyspnea and oedema developed gradu-

ally over the prior two months with multiple febrile episodes. The patient had no significant medical history and specifically no history of TB or HIV. A traditional healer was consulted two weeks before referral, where ritual scarification of his lower oedematous limbs was performed. Shortly after this procedure his condition worsened rapidly, triggering referral. On arrival at the outpatient TB consultation, the patient presented with a qSOFA (quick Sequential Organ Failure Assessment) of 2 with tachypnea (28 breaths per minute) and hypotension (95/55 mmHg), without confusion. The patient was tachycardic (115 beats per minute) but afebrile (35.8°C without chills), with a normal oxygen saturation on ambient air as measured by pulse oximetry (98%). Clinical examination revealed NYHA stage IV dyspnea with marked orthopnea, symmetric pitting oedema and ulcerative cellulitis of the lower extremities (Figure 1). Cardiopulmonary auscultation was marked by diffuse rhonchi and dull heart tones. Upon admission, chest X-ray revealed an extremely enlarged heart shadow and diffuse lung infiltrates (Figure 2). Basic laboratory work-up showed a normocytic normochromic anemia of 10.6 g/dL, normal platelets of 381 G/L, leukocytosis of 15.5 G/L. PoCUS performed by the on call pneumologist (AF) revealed a swinging heart with right ventricular collapse in a large pericardial effusion with several strands and a positive plankton sign, indicative of purulent cardiac effusion (Figure 3 & 4). Careful ultrasound-guided left lateral drainage confirmed the presence of abundant viscous, yellow-tinged purulent fluid (Figure 5). Culture revealed methicillin-sensitive Staphylococcus aureus (MSSA). Further polymerase chain reaction (PCR) testing confirmed a Panton-Valentine Leukocidine (PVL) toxin producing MSSA.

Mycobacterial culture (on a conventional Lowenstein-Jensen medium) and PCR of the pericardial fluid were negative for M. Tuberculosis. Sputum examination with GeneXpert MTB/RIF® assay was also negative. The patient suffered a cardio-respiratory arrest in the hour after drainage, and demised in the absence of advanced life support resources.

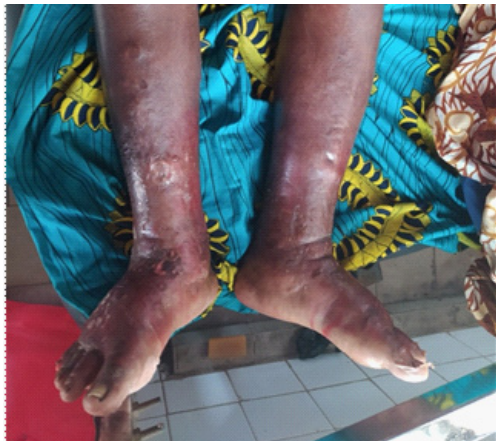

Figure 1: Lower limb pitting oedema and ulcerative cellulitis after ritual scarification.

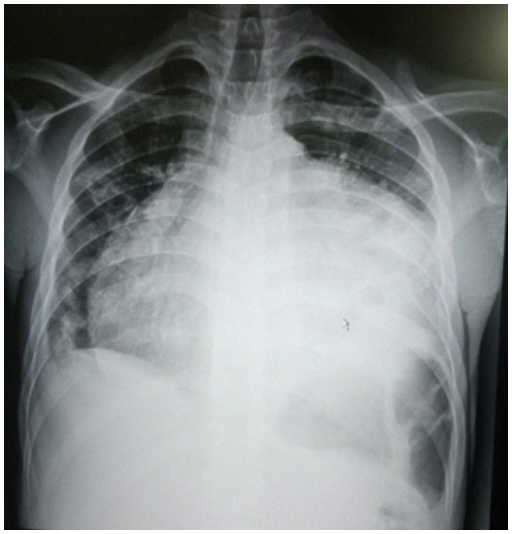

Figure 2: Chest X-ray face, revealing an extremely enlarged heart shadow along with diffuse pulmonary infiltrates.

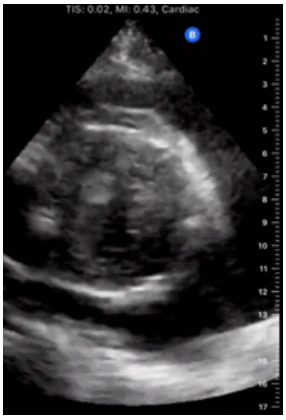

Figure 3: Ultrasound view with probe in position of the patient's left lateral lung quadrant demonstrating a swinging heart in a massive pericardial effusion with several strands and a positive plankton sign indicative of a purulent effusion. The echogenicity of the effusion is almost equal to that of the spleen. Images recorded in an abdominal pre-set to overcome depth and gain of the cardiac pre-set.
LV Left Ventricle: PPE Purulent Pericardial Effusion; S: Spleen. RIGHT: In comparison, parasternal short axis view example of a large anechogenic pericardial effusion without the plankton sign as seen in classic TB pericarditis.

Figure 4: Thick yellow fluid drained from the pericardium. Culture was positive for methicillin- sensitive Staphylococcus aureus (MSSA).

Discussion

Purulent pericarditis is a rare but life-threatening condition. Late presentation and increasing antibiotic resistance can cause rapid deterioration, which is particularly common and fatal in low resource settings. The infection can originate from a contiguous intra-thoracic source (pneumonia, empyema or deep mediastinal infections), or through hematogenous spread. Most commonly identified bacteria are Streptococcus pneumoniae, Staphylococcus spp. and Haemophilus spp [5-7]. In Sub-Saharan Africa, an estimated 64 to 70% of pericardial effusions are due to tuberculosis (TB) [8]. In HIV infected cohorts, disseminated TB is more prevalent and causes up to 85% of pericarditis [9]. The most common clinical presentation of TB pericarditis is a large pericardial effusion (clear anechogenic fluid on ultrasound) with chronic cardiac compression mimicking heart failure including pitting oedema of the lower limbs [10].

Confirming the diagnosis of TB pericarditis in resource limited settings is difficult due to the poor availability of microbiological assays (mycobacterial culture, polymerase chain reaction), specific biochemistry (increased levels of adenosine deaminase, interferon or pericardial lysozyme) and histology (presence of caseous granulomas) [11]. Moreover, the diagnostic performance of PCR and culture in pericardial fluid alone remains sub-optimal with a sensitivity of 54% [10,12] and 15% [13,14] for conventional Lowenstein-Jensen culture and PCR respectively. Thus, the diagnosis is most commonly suspected on a clinical basis.

The history of this young patient is most compatible with a large chronic TB pericardial effusion complicated by hematogenous MSSA PVL+ superinfection through a port of entry in the lower extremities (in this case, cellulitis following traditional leg scarification).

PoCUS performed at the bedside by the on-call pulmonologist immediately allowed for the diagnosis and management of purulent cardiac effusion at no cost to the patient. While complete echocardiography remains firmly in the domain of experts [15], the specific skill of identifying pericardial effusion has high diagnostic accuracy in PoCUS-trained physicians, with a sensitivity and specificity of 89-91% and 96% respectively [16].

In contrast with chest X-ray, PoCUS also enables a basic appreciation of heart function and a qualitative evaluation of the effusion, such as was determined in this case with visible strands and a positive plankton sign. This sign was first described by Lichtenstein in 2008 in pleural effusions [17]. It refers to slow moving, whirling internal echoes within an anechogenic effusion, buffered by cardiac or respiratory impulses. It excludes a transudate and is highly indicative of an exudative, hemorrhagic or purulent effusion (Figure 3).

Treatment of purulent cardiac tamponade ideally consists of prompt subxiphoid or lateral percutaneous pericardiocentesis followed by pericardiotomy by an experienced surgeon, antimicrobial treatment with gram positive coverage and advanced supportive care [11]. In this case, and commonly in resource limited settings, only percutaneous pericardiocentesis and antibiotics were available. PoCUS can significantly improve the safety and efficacy of pericardiocentesis and is easy to perform [18].

Purulent cardiac tamponade is a challenging, hyperacute condition requiring resource-intensive and invasive specialist care. In resource-limited settings, early recognition and safe, conservative intervention are crucial to avoid untreatable complications. When left untreated, it is always fatal. Even in high-income countries with intensive care and cardiac surgery readily available, mortality of treated bacterial pericarditis (with or without tamponade) still reaches 40% [4]. In the pre-antibiotic era, mortality of untreated TB pericarditis without superinfection reached 80-90% [19]. However, when recognized early before cardiac compression develops, medical treatment alone offers excellent long-term results where up to 87% of patients can survive without complications such as secondary constrictive pericarditis [5,20].

Conclusion

PoCUS integrated into routine clinical care offers the possibility to improve and expedite the diagnosis and safe management of pericardial effusions. In TB endemic regions, large effusions are more common and are very easy to recognize on ultrasound. Early recognition with handheld ultrasound and treatment can lead to better outcomes. The plankton sign offers a simple way to further distinguish between exsudative/purulent/hemorrhagic and clear fluid content.

Declarations

Acknowledgments: We thank the CHUV (Lausanne, Switzerland) and HUG (Geneva, Switzerland) humanitarian funds for financing POCUS devices and training at our university hospital.

The authors are grateful to the patient and his parents for their cooperation.

Conflict of interest statement: None to declare.

Ethics statement: The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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