CASE REPORT

Cardiac Tamponade: A Rare Complication of *Plasmodium Knowlesi* Malaria

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ABSTRACT

Plasmodium knowlesi has been discovered as the fifth species causing malaria in humans. It is a major public health problem in South East Asia especially in Borneo. We report a case of pericardial effusion that rapidly progressing to cardiac tamponade, an atypical presentation of *P. knowlesi* malaria. Our patient had no underlying known medical illness, presented with high grade fever with chills and rigors, epigastric pain, nausea, vomiting and with poor oral intake. Initial bedside cardiac ultrasound showed minimal pericardial effusion. Within a few hours, she became hypotensive, deteriorated rapidly despite fluid resuscitation requiring mechanical ventilation and inotropic support. Bedside cardiac ultrasound showed cardiac tamponade and pericardiocentesis was done. We highlight the importance of having high level of suspicion for this atypical presentation of cardiac tamponade when a patient is hypotensive in *P. knowlesi* infection. Prompt diagnosis and management may prevent potentially fatal complication.

Keywords: Cardiac tamponade, *P. knowlesi*, Malaria

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INTRODUCTION

Humans are the natural hosts for *P. falciparum, P. vivax, P. malariae* and *P. ovale*. Documented spectrum of disease ranges from simple fever to complicated forms which may include multiorgan involvement usually due to *P. falciparum* and *P. vivax* (1). The natural host for *P. knowlesi* which is long-tailed macaques (Macaca fascicularis) was initially thought to be extremely rare until 2004 when there was a large focus of infections in the state of Sarawak, Malaysia Borneo. Since then, *P. knowlesi* is considered the fifth species of *Plasmodium* causing malaria in humans.

CASE REPORT

A 45-year-old lady, with no known medical illness, presented to our hospital with a complaint of fever, epigastric pain, vomiting, generalized body weakness and giddiness for four days duration. Upon arrival to the casualty, she appeared lethargic and hypotensive with her systolic blood pressure ranges from 60-80 and her diastolic blood pressure ranges from 30-40 mmHg. She was tachycardic with the heart rate (HR) of 130-160 beat per minutes. Her respiratory rate was 22 breaths/min. Her oxygen saturation was 98% under room air. Systemic examination revealed tenderness over epigastric region with muffle heart sound. Otherwise there was no other remarkable findings. Bedside cardiac ultrasound showed very huge pericardial effusion with good left and right ventricular contractility (Figure 1). Her inferior vena cava (IVC) collapsibility index was more than 50% and almost kissing. Blood investigation sent revealed negative result for both dengue non-structural protein1 (NS1) antigen and the dengue IgM. She was thrombocytopenic with the platelet count of 11. A blood film for malaria parasites (BFMP) was performed and reported to be positive. At this point of time, the *Plasmodium* type was not confirmed yet. She was diagnosed with severe malaria infection and started on intravenous artesunate 170 mg daily for 2 doses and tablet doxycycline 100mg 12 hourly for 5 days. A combination of normal saline and gelafundin was given by the emergency department (ED) team up to 30ml/kg/hr for fluid challenge. After fluid boluses were given in ED, her blood pressure picked up to 92/50 mmHg and

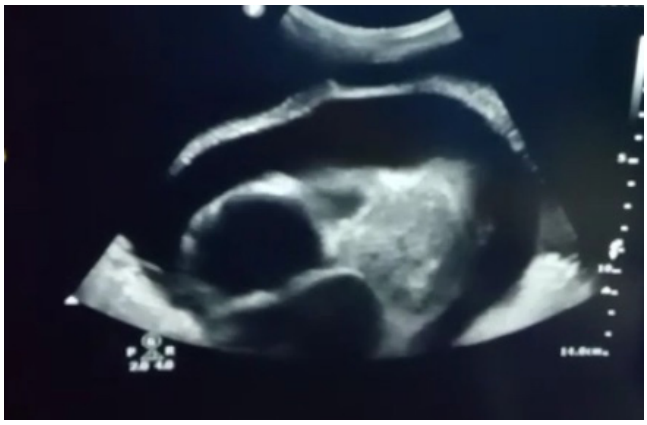

Figure 1: Pericardial effusion (black area) with right ventricular compression (triangle hyperdense area)

her HR came down to 120 beats per minute. She was then started on inotropic supports, noradrenaline 0.1- 1 microg/kg/min and dobutamine up to 10 microg/kg/min that were titrated to achieve a mean arterial pressure of 65 mmHg. She was admitted to intensive care unit (ICU). Repeated arterial blood gas showed worsening metabolic acidosis; pH 7.4, PCO^2 of 20.1, PaO^2 of 381, HCO^3 of 12 and base excess (BE) of -12 with reduced urine output. Other inotropes such as dopamine and adrenaline were added in addition to the ongoing inotropes. She was then intubated. The induction drugs used were intravenous fentanyl 100micg, midazolam titrated up to 4 mg followed by suxamethonium 100 mg. Bedside cardiac ultrasound was repeated in ICU and revealed poor cardiac contractility due to cardiac tamponade (Figure 1). Urgent pericardiocentesis was done. A total of 220 mls serous fluid was drained. The fluids were sent for microbiological investigations. Blood pressure became normotensive on four types of inotropic support after the pericardiocentesis procedure. Later, we were able to taper down the inotropic support from four inotropes to single inotrope which was noradrenaline. She underwent hemodialysis later in view of severe metabolic acidosis.

Her BFMP revealed *Plasmodium* infection which was confirmed later caused by *P. knowlesi* using PCR. The type of PCR used was multiplex real-time PCR using abTES Malaria5 qPCR kits. This multiplex real-time PCR was proven to be an acceptable method for detection of malaria species as reported by Krishna et al in 2009 (2). The gene targeted in this real time PCR were *P. falciparum, P. vivax, P. ovale, P. malariae* and *P. knowlesi*. The parasite count was 160000/µl. She responded well to artesunate and doxycycline. The parasite counts rapidly reduced to half, which was 84210/ul. The patient showed clinical improvement despite oliguric acute renal failure. Ventilation and oxygenation were good with no signs of bleeding even though she was thrombocytopenic.

Daily parasite count was reduced until becoming undetectable on day four of ICU admission. We were able to wean off the noradrenaline one day later. Bedside cardiac ultrasound did not show any evidence of recurring pericardial effusion. She was extubated on day 7 of ICU admission and transferred back to the general wards two days later.

DISCUSSIONS

Pericardial involvement due to malaria is considered as an atypical presentation. The most common presentation is pericarditis manifesting as conduction abnormalities and myocarditis. Both are due to *P. falciparum* infection. There have been rare cases reported of pericardial effusion progressing to tamponade in *P. falciparum* infection (3). In this case study, we would like to highlight a rare manifestation of *P. knowlesi* infection that rapidly progressed from mild pericardial effusion to cardiac tamponade within a matter of hours. This is the first case of cardiac tamponade occurring in *P. knowlesi* infection being reported in Malaysia to our knowledge.

The mechanism of pericardial effusion development and how it progressed to cardiac tamponade in this case is not fully understood. One possible explanation is probably the effect of pro-inflammatory cytokines released during infection and its effect systemically. Clark et al. concluded that excess inflammatory cytokines was showed to cause cardiomyocyte mitochondrial depression that are highly expressed in *P. falciparum* malaria (4). Day et al showed that cytokines produced in a particular tissue may be regulated by the degree of parasitic sequestration. Overzealous fluid resuscitation leading to overload is unlikely as there are no peripheral edema and oxygenation impairment (5).

CONCLUSION

We concluded that *P. knowlesi* malaria may present with pericardial effusion that can progress to cardiac tamponade rapidly. Cardiac tamponade should be actively ruled out in a hypotensive patient presenting with *P. knowlesi* infection. Prompt diagnosis and management may prevent morbidity as well as mortality.

ACKNOWLEDGEMENT

I am taking this opportunity to thank everyone in Hospital Lahad Datu, Sabah who has involved in this patient management. Our sincere appreciation goes to Department of Anesthesiology Hospital Lahad Datu and Department of Anesthesiology Universiti Sains Malaysia for collaboration in writing this report.

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