Paediatrica Indonesiana

p-ISSN 0030-9311; e-ISSN 2338-476X; Vol.64, No.2(2024). p.184-92 ; DOI: https://doi.org/10.14238/pi64.2.2024.184-92

Case Report

# Challenges in diagnosing pediatric pericarditis and the etiology in a remote area during the COVID-19 pandemic: a socio-clinical dilemma

**Armand Setiady Liwan[1], Ni Putu Veny Kartika Yantie[2], Eka Gunawijaya[2]**

Inflammation of the visceral and parietal surfaces of the pericardium is defined as pericarditis. It can evolve to excessive production of pericardial effusion if the speed of fluid accumulation is faster than the absorption. Acute pericarditis is rare in children but it can lead to circulatory collapse and death. It accounts for <0.2% of the emergency visits of children without prior heart diseases in tertiary pediatric emergency settings. The etiology of acute pericarditis varies depending on geography, and the most common etiology in children are bacterial infection, viral pericarditis, inflammatory or connective tissue diseases, malignancies, metabolic diseases, and post-cardiac surgery. Idiopathic pericarditis is presumed to have viral or post-viral etiology. It accounts for 37-68% of admissions in children with pericardial effusions or acute pericarditis. **[Paediatr Indones. 2023;64:184-92; DOI: 10.14238/pi64.2.2024.184-92** ].

***Keywords:*** *pericarditis; pericardial effusion; children; diagnosis*

There are currently no definitive diagnostic criteria for pediatric pericarditis. A combination of typical signs and symptoms with supporting examination is needed to diagnose pericarditis. Typical symptoms of acute pericarditis are chest pain (sharp/stabbing pain), positional, radiating pain, and sometimes relieved by sitting upright. Several non-specific symptoms are cough, fever, dyspnea, abdominal pain, and other symptoms related to the underlying systemic diseases.[1-4] Electrocardiography (ECG), chest x-ray, and transthoracic echocardiography are required to confirm the diagnosis. Transthoracic echocardiography is the most sensitive and useful tool for identifying pericardial effusions; however, it is not widely available in remote hospitals in Indonesia. It is also an operator-dependent tool, and a pediatric cardiologist or trained doctor is required to operate it.[1,4,5]

Oksibil Hospital is located in Pegunungan Bintang district, Papua, the easternmost province in Indonesia. Lies in the middle of Papua Island, it is part of a mountain range that extends into Papua New Guinea in the east. The lowest temperature reached in Oksibil is 12°C at night (altitude 2.567 m above sea level). The only way to reach Oksibil is by plane, a one-hour flight from Jayapura (3777 km from Jakarta), which is available three times a week. Neither connecting roads nor waterways are available to exit the district. The Oksibil area (capital of the district) is not supplied with 24 hours of electricity; it runs only for 7 hours starting from 06.00 pm. There is no telephone line. The primary source of water supply is rainwater, which frequently results in a shortage of water, even in our hospital. The Oksibil Hospital is equipped with an emergency unit, polyclinic, 40 beds, and one operating room (substandard). The intensive care unit, minimum standard laboratory, and radiology

From Oksibil Hospital, Pegunungan Bintang District, Papua[1] and Department of Child Health, Sanglah Hospital, Denpasar, Bali[2], Indonesia.

**Corresponding author:** Armand Setiady Liwan. Oksibil Hospital. Jalan Poros Kabiding no. 02, Oksibil, Pegunungan Bintang District, Papua 99573. Phone (+62) 85244062020. Email: armandliwan@gmail.com.

Submitted May 20, 2022. Accepted February 21, 2024.

unit are not available. In the setting of our hospital, we report an unusual case of a Melanesian girl who presented with acute pericarditis and pericardial effusion.

## The case

A 15-year-old girl came to the emergency unit of Oksibil Hospital in Pegunungan Bintang district, Papua, on February 1, 2022, with shortness of breath, fever, cough, fatigue, and loss of appetite. The patient reported experiencing shortness of breath one day prior to admission, fever for three days leading up to admission, and has been complaining of cough, fatigue, and loss of appetite for the past month. There was no history of chest pain before. The patient had a history of several visits to policlinic because of epigastric pain since the previous year. There was no history of similar complaints, swelling, fainting, or any history of being out of the district before. She had not received basic immunizations in the past, including the COVID-19 vaccination. There was no record of prior surgeries or trauma. The patient was born spontaneously at full term with no asphyxia. The patient's developmental history was normal, but she could not speak Bahasa Indonesia fluently. The patient lived in a small, traditional, densely populated wooden house with eight people and used firewood for daily cooking.

The patient was alert but in a weak condition. Her blood pressure (BP) was 95/70 mmHg, pulse rate was 124 beats per minute (good quality), respiratory rate was 40 times per minute, the axillary temperature was 37.8°C and oxygen saturation ($SpO_2$) was 88% (before oxygen supplementation). The patient's weight and height were 41 kg (<5$^{th}$ percentile, CDC 2000; severely underweight) and 147 cm (<5$^{th}$ percentile, CDC 2000; severely stunted), but good nutritional status (based on Waterlow 102.5%). Paleness, bilateral neck lymphadenopathy, and rales from both the lungs and subcostal retraction were noticed. Liver and spleen were not palpable. No *Bacille Calmette-Guerin* (BCG) scars were found. She was diagnosed with community acquired pneumonia (CAP), suspected lung tuberculosis (TB), suspected COVID-19 infection, moderate anemia, severely underweight, severely stunted, and well-nourished. The summary of clinical changes, supporting examinations (available/not available), and therapy during hospitalization are provided in **Table 1**.

On the 2$^{nd}$ day of admission, the patient was clinically stable but complained about epigastric pain. Pitting edema was observed in both legs. On the 3$^{rd}$ day of admission, the patient was hypotensive with a BP of 75/65 mmHg, pulse rate of 136 beats per minute (narrow pulse quality), respiratory rate of 48 times per minute, and axillary temperature of 36.6°C with warm extremities, SpO2 was 98% (1 Liter/minute); capillary refill time (CRT) was 2 s; she was also noted to be anuria for 11 hours. She was assessed with hypovolemic shock and identified as being at risk for acute kidney injury (AKI), with a differential diagnosis of chronic kidney disease (CKD). On the 4th day of admission, the patient's clinical condition worsened, and shortness of breath increased. Epigastric pain improved, but she complained about backache. She looked more comfortable in sitting upright than lying down on the bed. Blood pressure fluctuated from 80/60-95/65 mmHg, pulse rate was 132-144 beats per minute (narrow pulse quality), respiratory rate was 58-62 times per minute, $SpO_2$ decreased to 87%, and the body temperature was normal. Breath sounds were decreased in both lungs and shifting dullness was observed. Pitting edema persisted and CRT was 3 seconds. Electrocardiography showed a low voltage indicating pericardial effusion. The patient was suspected to have severe systemic lupus erythematosus/SLE (probable-SLICC 2015) with differential diagnosis of nephrotic syndrome (NS). High-dose methylprednisolone (HDMP) was administered for three days as a life-saving purpose in a suspected severe SLE. The patient was intended to be referred to a secondary hospital at Jayapura (capital of the province), but the transfer could not be arranged because of financial constraints.

On the 5$^{th}$-7$^{th}$ day of admission, the patient's condition was dramatically improved, and shortness of breath decreased. There was no epigastric or back pain, no fever, and the patient started to eat and walk by herself. Blood pressure was 95/65-105/70 mmHg, pulse rate was 100-114 beats per minute (good quality), respiratory rate was 32-36 times per minute, and $SpO_2$ was 99% (1 L/min). The edema on each leg had disappeared. On the 8$^{th}$ day of admission, since early morning the patient experienced severe

**Table 1**. Summary of clinical changes, supporting examinations, and therapy during hospitalization

| Period of admission | Clinical changes | Supporting examinations (blue colour: plan examination but not available) | Additional therapy |
|---|---|---|---|
| 1st day | Shortness of breath, cough, fever (Pneumonia, suspected TB, suspected C19 infection)<br><br>(Plan for Covid-19 Antigen test, CBC, sputum acid-fast bacillus (AFB) test, gene Xpert MTB, blood culture, chest x-ray: not available) | • WBC 11.300/µL (manually count),<br>• Hb 8.3 g/dL (Sahli's method)<br>• Malaria rapid test (-)<br>• HIV rapid test (-) | • IVFD D5½NS 40 mL/hour<br>• $O_2$ cannula 1 L/m<br>• Broad spectrum empirical antibiotic (ampicillin, gentamicin IV)<br>• Oral paracetamol 10 mg/kg every 8 hr<br>• NaCl 3% nebulization 5 mL every 8 hr<br>• Oral acetylcysteine |
| 2nd-3rd day | Lower extremities edema, epigastric pain, hypotension | • ECG: T inversion on V2-V6, day 2 (**Figure 1a**)<br>• Tuberculin test: (-) day 3 | • IVFD RL loading 20 mL/kg<br>• Omeprazole 1 mg/kg every 24 hr (IV)<br>• Furosemide 0.5 mg/kg every 8 hr (IV) |
| 4th day | Increased shortness of breath and back pain were complained, epigastric pain improved, and transient hypotension<br><br>(Severe SLE suspected) | • ECG: low voltage (**Figure 1b**)<br>• Proteinuria (+) dipstick<br><br>(Plan for ANA test, anti-ds DNA, complement, albumin serum, cholesterol: not available) | • $O_2$ mask 5 L/m (to maintained $Sp0_2$ >95%<br>• Ceftriaxone 75 mg/kg every 24 hr IV (antibiotic change)<br>• Albumin loading (IV) 20% 1g/kg over 1 hr<br>• High-dose methyl-prednisolone 1000 mg over 1 hr every 24 hr (3 days) |
| 5th-7th day | Epigastrial pain improved, edema relieved, started to walk and feed by herself | ECG: T inversion on II,III, aVF, V4-6 (**Figure 1c**). | • $O_2$ canulla tapering to 1 L/m<br>• High-dose methylprednisolone changed to low-dose methylprednisolone 1 mg/kg/ day<br>• Furosemide stop<br><br>(Cyclophosphamide/mycophenolate sodium: not available) |
| 8th day | Severe abdominal pain, escpescially on epigastrial/ Mc-Burney point | • WBC 23.300/µL (manually count)<br>• Hb 8.7 g/dL (Sahli's method)<br><br>(Consultation with a general surgeon for the possibility of appendicitis: not typical appendicitis) | Paracetamol 15 mg/kg every 6 hr (analgetic) |
| 9th day | Abdominal pain was relieved, but severe chest pain appeared radiating to the back, shortness of breath increase, edema, hypotension, narrow pulse, distant heart sound<br><br>(Pericarditis suspected with pre-tamponade condition) | • ECG: low voltage, T inversion persisted (**Figure 1d**),<br>• Apical 4 chambers viewed by (USG) 2D with convex-abdominal probe: confirming pericardial effusion<br><br>(Consultation with a pediatric cardiologist in Bali for evaluation and treatment) | • Pericardiocentesis (advise from a pediatric cardiologist in Bali): not done because of lack of trained professionals,<br>• Oral colchicine 0.6 mg every 12 hr, 0.6 mg every 24 hr on the next day<br>• Albumin 20% (IV) maintenance dose 0.5 g/kg/ day<br>• Furosemide use with caution |
| 10th day | Chest and abdominal pain improved, and edema slightly decreased | | • Patient discharged against medical advice<br>• Oral colchicine 0.6 mg every 24 hr<br>• Oral methylprednisolone 1 mg/kg/ day |

abdominal pain, especially in the epigastric, left hypochondriac, and right iliac region. There was no fever, nausea, or vomiting. Blood pressure was 95/70 mmHg, pulse rate was 134 beats per minute (good quality), and respiratory rate was 52 times per minute. Mc-Burney point tenderness, Rovsing, and Blumberg

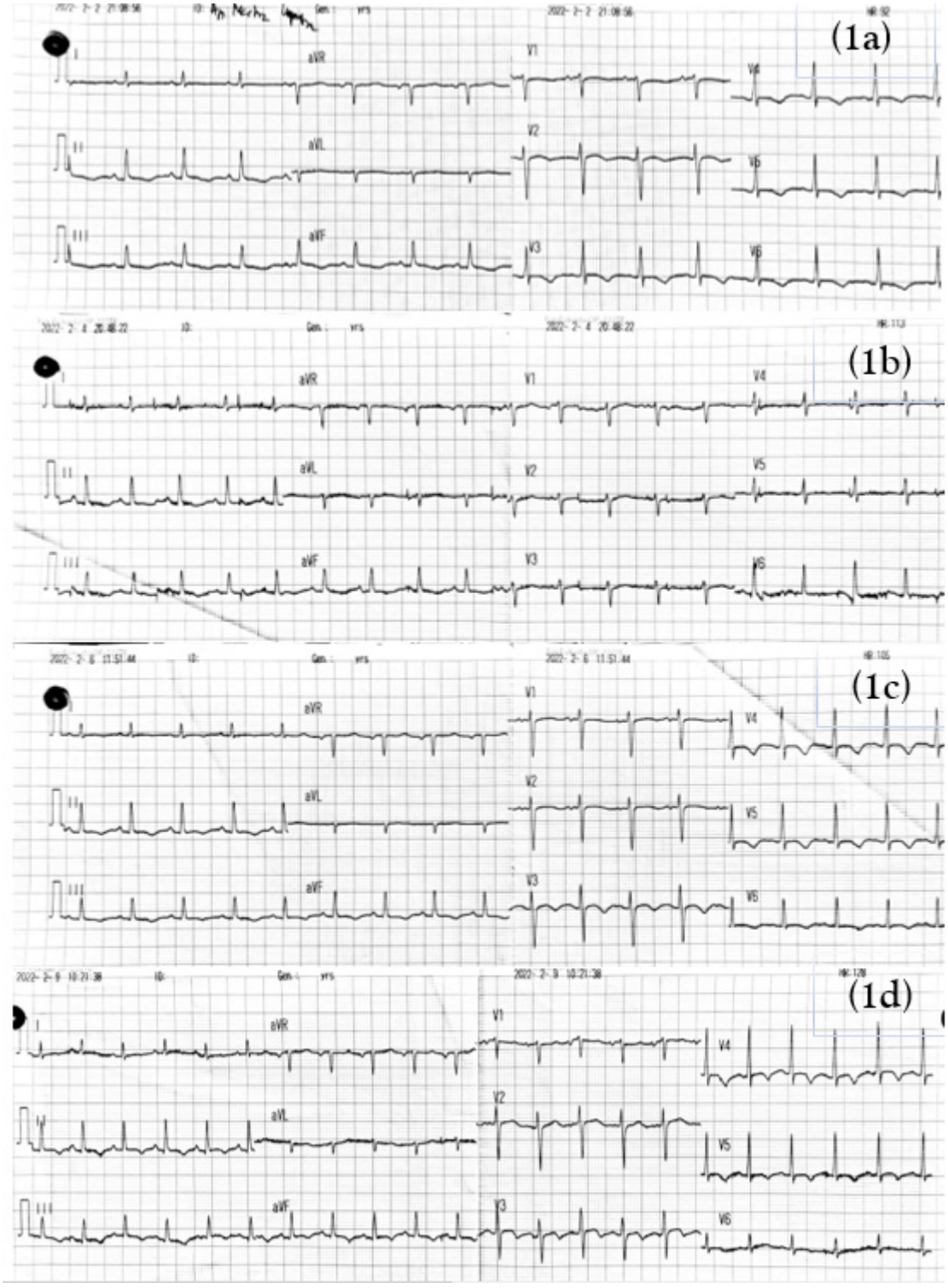


**Figure 1**. a) T inversion on V2-V6 (2nd day), b) Low voltage noticed (4th day), c) T inversion appeared on II, III, aVF, V3-V6 (6th day), d) T inversion persisted (9th day)

signs were positive. The patient was consulted by the team to a general surgeon to evaluate the possibility of acute appendicitis and treatment. However, the surgeon disagreed with the diagnosis of acute appendicitis and suggested conservative therapy.

On the 9th day of admission, there was no abdominal pain, but the patient experienced severe chest pain radiating through the back. The patient appeared restless with increased shortness of breath. Blood pressure was 80/60 mmHg, pulse rate was 146 beats per minute, respiratory rate was 58 times per minute (narrow quality) and temperature was normal. The oxygen saturation was 85% with oxygen nasal cannula of 1 lpm. Physical examination revealed distant heart sounds, narrow pulse pressure, and pitting edema on the hands and legs. Murmur, pericardial friction rub, distended jugular vein and hepatosplenomegaly were absent. The patient's extremities were warm, and the CRT was 2 seconds. Conventional ultrasonography (USG) 2D with a convex-abdominal probe (apical four chamber view) showed pericardial effusion with normal heart contractility without right atrial/ventricle collapse (**Figure 2**). The patient was diagnosed with acute pericarditis and pericardial effusion (PPE) pre-tamponade et causa suspected SLE with differential diagnoses of tuberculous (bacterial)/COVID-19 (viral) pericarditis. Thus, the patient was consulted to pediatric-cardiologist in Bali using a satellite Wi-Fi telephone to confirm the assessment and treatment. She was advised to undergo pericardiocentesis, albumin correction if hypoalbuminemia/albumin maintenance, furosemide use with caution, and colchicine for pericardial inflammation pain. Pericardiocentesis was not performed due to the absence of parental consent and trained professionals were unavailable.

On the 10th day of admission, the chest and back pain were relieved, and the patient did not complain about any pain, but shortness of breath persisted. The blood pressure was recorded at 95/70 mmHg, pulse rate was 120 beats per minute, respiratory rate was 48 times per minute, and temperature was normal. Although distant heart sounds with narrow pulse

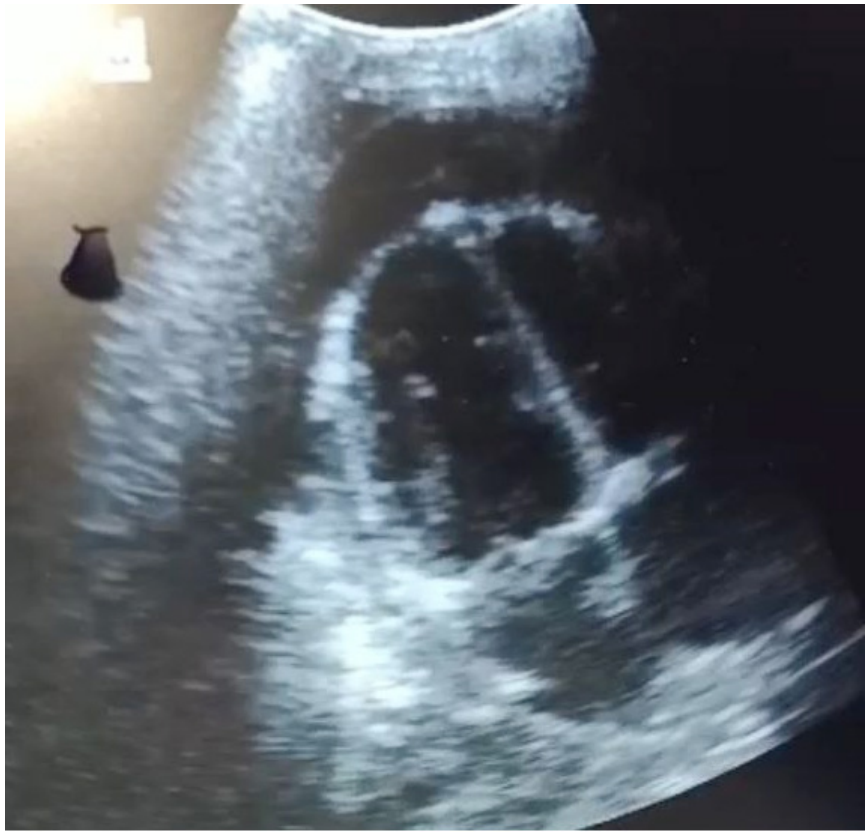

**Figure 2**. Apical 4 chambers view using convex-abdominal probe (USG 2D)

quality persisted, edema decreased. The intravenous administration of albumin maintenance dose was discontinued due to unavailability. In the afternoon, the patient requested discharge because of the financial problem. Consent was given from both the parent and the attending doctor. The patient received colchicine and oral methylprednisolone before leaving and did not revisit the hospital thereafter.

## Discussion

Children with acute pericarditis do not always come with typical and specific symptoms, such as sharp/stabbing chest pain, thus it can be difficult to distinguish it from other diseases. Chest pain can be seen in up to 6 in 1000 children and the incidence of cardiac chest pain is very low (0.2-1% of cases). The most common cause is idiopathic, musculoskeletal, psychological, gastrointestinal, respiratory, and the least common cause is cardiac etiology.[1,3,4] In our case, typical symptom presented late on the 9$^{th}$ day, marked by chest pain radiating to the back. Prior to the chest pain, the patient already complained epigastric pain on day 2, and had history of several episodes of epigastric pain since last year. Backache was complained on 4$^{th}$ day of admission. The epigastric pain and backache were relieved on the 5th day (after high dose of methyl prednisolone/HDMP). Severe abdominal pain (Mc-Burney tenderness) was complained on the 8$^{th}$ day of admission, which increased our suspicion of appendicitis. Epigastric pain, backache, and Mc-Burney tenderness were likely radiating pain from the heart. Shortness of breath, cough, fever, and fatigue were non-specific symptoms of acute pericarditis, which lead to an initial misdiagnosis of pneumonia.

Muffled/distant heart sounds, tachycardia, narrow pulse pressure, jugular venous distention and pericardial friction rub provide clues to the diagnosis of acute pericarditis. Excessive fall of systolic blood pressure >10 mmHg during inspiration (pulsus paradoxus) is a clue to cardiac tamponade.[1,3,5] In our case, distant heart sounds with narrow pulse pressure were appeared late on the 9$^{th}$ day and leg edema was noticed on the 2$^{nd}$ day. Tachycardia was observed throughout hospitalization. Children with pericarditis tend to be fussy and have decreased feeding with tachycardia being an important physical sign. Particular attention to resting tachycardia is needed to identify the diseases.[1] The most common presenting signs are tachycardia (90%), followed by tachypnoea (69%).[3] Hypotension and narrow pulse pressure were initially misinterpreted as a shock. Pulsus paradoxus may be misinterpreted as a transient hypotension. Leg edema with ascites was obviously noticed on the 4$^{th}$ day. It raised our suspicion toward heart problems.

The diagnosis of PPE can be challenging, especially for clinicians in remote hospitals with limited facilities. Chest x-ray, ECG, cardiac markers, transthoracic echocardiography, cardiac CT scan, and MRI were usually needed, but not widely available. Pericardial fluid analysis is often required to identify the etiology. In our case, serial ECG were performed during hospitalization, and low voltage and T inversion were noticed on the patient. The first ECG was performed on the 2$^{nd}$ day because of the appearance of leg edema to look for the possibility of heart hypertrophy indicating a heart failure. Chest pain, along with ECG changes and inflammatory markers might be helpful in the diagnosis of pericarditis.[1] Sixty percent of patient with paediatric pericarditis shows abnormal ECG.[5] Serial ECG which provide a clue to our diagnosis. Instead of using transthoracic echocardiography, we used conventional ultrasonography (USG) 2D with a convex-abdominal probe to determine the apical four chamber/ parasternal long axis view to looked for pericardial effusion and eye-bawling the contractility of the heart. It showed pericardial effusion without ventricular collapsed. Although transthoracic echocardiography appears as a class I recommendation for adult patients, it is not uncommon for children with suspected acute pericarditis to be treated empirically without echocardiography.[1] Total pain relieved after colchicine administration confirming our diagnosis, although the etiology of PPE was still unknown.

The most common cause of pericarditis and pericardial effusion (PPE) in developing countries is infection (especially tuberculosis); however, in developed countries, the non-infection cause is more prominent.[1,3,5] Tuberculous pericarditis accounts for only 8% of all TB cases, and in endemic country like Indonesia, the number could be higher. The mortality rate reached 17-40% depend on the treatment

adequacy.[6,7] In our case, the patient was initially suspected to have lung TB, and complained cough with fatigue since the last month, which increased our awareness of TB. Unfortunately, the patient could not be tested for TB in our hospital, neither by bacteriological examination nor chest x-ray. There was no immunization history in this patient, including BCG vaccination (no BCG scar). No clear contact history of the patient with active TB cases; negative tuberculin test; and a total score of TB scoring system was only 3 (cough >3 weeks, cervical lymphadenopathy, severely underweight) reduced our suspicion of TB.

Image of USG 2D examination using a convex-abdominal probe of apical 4 chambers view was confirming our suspicion that the patient had a serous effusion/echo-free appearance (which could be a non-bacterial cause). Bacterial etiology typically results in serohemorrhagic or purulent effusion, characterized by an echo-dense or echo-genic appearance.[2] Leg edema was also observed in our patient. Edema is not a specific manifestation of pericarditis, but it may be a symptom of constrictive pericarditis where the pericardium becomes hard and thick (limited heart expansion), especially if it is a chronic manifestation. Tuberculosis is one of the most common causes of constrictive pericarditis.[6,7] History of edema before admission was denied by the patient.

The patient was suspected to have severe SLE (probable SLE-SLICC 2015) with two clinical manifestations: moderate anemia (although hemolytic process could not be confirmed); and pericardial effusion/ascites as 2nd criteria. Renal impairment was suspected because the patient was diagnosed with AKI (risk stage) with a differential diagnosis of CKD. Positive proteinuria further indicates renal involvement. The patient was also suspected with nephrotic syndrome (NS) but had not been confirmed. It is worth noting that nephrotic syndrome with cardiac involvement is an uncommon manifestation. Supporting examinations for establishing the diagnosis of SLE and NS are not available in our hospital (serum albumin, cholesterol level, ANA test, anti-dsDNA, antiphospholipid antibody, serum complement, etc), but remarkable clinical response after 3 days of HDMP administration strengthening our suspicion of severe SLE (ex-juvantibus diagnosis). Cardiovascular involvement in SLE varies widely; it has been reported to range from 4-78% and the incidence of pericarditis is 11-54% in SLE patients.[8] Severe cardiorespiratory involvement in children with SLE is less than 10%.[9] Pericarditis with pericardial effusion although uncommon, has been reported as an initial clinical manifestation.1[0-11] Hemolytic anemia, proteinuria, lymphadenopathy and anti-Smith are predictive of pericarditis.[8] Moderate anemia, proteinuria, and lymphadenopathy were present in the patient.

Children were initially considered less affected by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2)/ Covid-19 infection with milder diseases compared to adults, but current literature reported several children with multisystem involvement; thus, special attention should be paid to extrapulmonary manifestation.[12,13] Recent studies have reported that cardiac involvement is not rare in children, including ventricular dysfunction, pericarditis, coronaritis, and arrhythmias.[14,15] Some cases reported pericarditis as the main clinical manifestation of Covid-19 in children.16 In our case, the patient was initially suspected to have COVID-19 infection, but it could not be excluded because the swab antigen test for COVID-19 was unavailable due to a shortage, whereas the polymerase chain reaction (PCR) test was also not accessible. Additionally, a serology test for COVID-19 antibodies was also unavailable. The patient was hospitalized during the peak of 3rd wave of the COVID-19 pandemic in Indonesia. She had not received a COVID-19 vaccination, along with the majority of the local community. The patient's family did not have similar symptoms, and she denied any travel history outside the district.

In this case, we could not differentiate whether the patient had a current COVID-19 infection, post viral infection symptoms, or a multisystem inflammatory syndrome in children (MIS-C). Children age range 0-19 years old and fever ≥ 3 days; symptoms with minimal 2 organ involvement; increased inflammatory marker (ESR, CRP, Procalcitonin); the absence of bacteriological etiology; and evidence of recent or history of COVID-19 infection are needed for establishing the diagnosis of MIS-C.[17,18] In the patient, fever with hypotension, pericarditis/ pericardial effusion, and abdominal pain were observed. However, inflammation markers, blood culture, and COVID-19 infection markers could not

be conducted. Serial ECG examinations showed low voltage amplitude with T-inversion. A previous report stated that MIS-C is associated with ECG changes over the course of the diseases, with low amplitude ECGs on presentation, followed by transient T-wave inversion, particularly in the precordial leads. There was a low prevalence of ST-segment changes and tachyarrhythmias.[17] Majority of patients with MIS-C had a reduced left ventricular ejection fraction, and many of them were accompanied by PPE.[18]

As the largest archipelago in the world, Indonesia is facing healthcare problems. Disparities in the healthcare of rural residents compared to non-rural residents reflect a significant concern in our country. Access to care for rural residents is hindered by shortages of clinicians, facilities, as well as sociocultural, geographical, and climatic barrier. Clinicians and hospital management further challenged by ethical issues that impede the delivery of optimal quality care. Effective leadership in rural hospitals is crucial to provide accurate, clear, and promptly delivered information that aligns with the needs of both patients and clinicians.[19]

In our case, there was an intended referral of the patient to a secondary hospital on the 4th day of hospitalization. Unfortunately, this plan failed because of financial problems. The patient and her family had no national insurance coverage, and the referral budget in our hospital had not been disbursed by the government. A combination factor of low socio-economic status, level of education, language barrier, absence of immunization history, extremely remote location, local government policies, hospital facilities, and management, contributed to the patient's predicament. It is important to have patient advocates, individuals within communities who can facilitate communication and discuss about basic knowledge, innovative uses of technology, medical issues, policy applications, and best practices.[20]

Pericardiocentesis is considered a vital procedure in treating pre-tamponade PPE. In our case, pericardiocentesis was planned, but it was not performed due to parental consent issues and a lack of trained professionals. As a clinician, we are faced with a dilemma between limited facilities, financial issues, the patient's medical condition, and the absence of a trained pediatric cardiologist. Question arise regarding it is permissible for a general pediatrician to perform pericardiocentesis in such circumstances. Similarly, uncertainties surround the administration of HDMP in suspected severe SLE (ex-juvantibus) in remote area and whether general paediatrician can give anti-viral (COVID-19) or oral anti-tuberculosis drugs in this condition. Telemedicine may be helpful but many rural communities do not have access to technologies that use for telemedicine.[20] The best ways to support rural healthcare system is still a mystery, especially during a crisis such as the current pandemic. Collaboration between central and local governments is required to immediately solve this problem.

Paediatric patients with pericarditis do not always have typical signs and symptoms; thus, the diagnosis making process becomes difficult and treatment could be delayed especially in limited facilities. Proper basic knowledge about typical and untypical pericarditis symptoms in children should be mastered. In remote hospitals, in addition to precise history taking and physical examinations, serial ECG changes over the course of the disease and conventional USG 2D convex-abdominal probe could be used to diagnose PPE. Although the definitive etiology could not be determined, response therapy after HDMP administration increased our awareness toward SLE (ex-juvantibus diagnosis). Telemedicine may be useful in supporting the rural healthcare system. Multi-collaboration between central/local governments, leaders in communities, hospital management, and clinicians are needed to provide the best quality care, especially in extremely remote areas.

## Conflict of interest

None declared.

### Acknowledgement

I would like to express my special thanks to my supervisor Dr. dr. Ni Putu Veny Kartika Yantie, M.Sc, Sp.A(K) who gave me the opportunity to do this project on the topic of pediatric pericarditis, and also helped me manage this patient.

## References

1. Shankar B. Pediatric pericarditis. Expert analysis. American College of Cardiology. 2016 Jan [cited 2022 Feb 12]. Available from: https://www.acc.org/latest-in-cardiology/articles/2016/06/08/11/43/pediatric-pericarditis
2. Kucera F. Atlas of pediatric echocardiography. Amsterdam: Elsevier; 2020.
3. Abdel-Haq N, Moussa Z, Farhat MH, Chandrasekar L, Asmar BI. Infectious and noninfectious acute pericarditis in children: an 11-year experience. Hindawi: International Journal of Pediatric. 2018;1-12. DOI: https://doi.org/10.1155/2018/5450697
4. Loza S, Tallman B, Hanson K, Rainey S. A 15-year-old with chest pain: An unexpected etiology. SAGE Open Medical Case Reports. 2022;10:1-4. DOI: https://doi.org/10.1177/2050313X211069026
5. Shakti D, Hehn R, Gauvreau K, Sundel RP, Newburger JW. Idiopathic pericarditis and pericardial effusion in children: contemporary epidemiology and management. Journal of the American Heart Association. 2014;1-7. DOI: https://doi.org/10.1161/JAHA.114.001483
6. Paramitha W, Murni IK, Arguni E, Setyowireni D. Tuberculous pericarditis in adolescents: A case series. Paediatr Indones. 2020;60:111-6. DOI: https://doi.org/10.14238/pi60.2.2020.111-6
7. Isiguzo G, Bruyn ED, Howlett P, Nteskhe M. Diagnosis and management of tuberculous pericarditis: what is new?. Curr Cardiol Rep . 2020;22 (Suppl 2):1-8. DOI: https://doi.org/10.1007/s11886-020-1254-1
8. Dein E, Douglas H, Petri M, Law G, Timlin H. Pericarditis in lupus. Cureus. 2019;11:1-6. DOI: https://doi.org/10.7759/cureus.4166
9. Leary DO, Connor CO, Nertney L, Mac-Dermott EJ, Mullane D, Franklin O, *et al.* Juvenile systemic lupus erythematosus presenting as pancarditis. Pediatr Rheumatol . 2019;17 Suppl 71:1-4. DOI: https://doi.org/10.1186/s12969-019-0372-z
10. Morales VG, Magana EL, Ramirez ARL, Guerrero JMC, Quibrera J, Gaxiola GP, *et al*. Pericarditis and pericardial effusion as the first presentation of systemic lupus erythematosus. Rev Alerg Mex. 2019;66 Suppl 1:132-9. DOI: https://doi.org/10.29262/ram.v66i1.528
11. Bezwada P, Quadri A, Shaikh A, Ayala-Rodriguez C, Green S. Myopericarditis and pericardial effusion as the initial presentation of systemic lupus erythematosus. Hindawi: Case Reports in Medicine. 2017:1-4. DOI: https://doi.org/10.1155/2017/6912020
12. Raymond TT, Das A, Manzuri S, Ehrett S, Guleserian K, Brenes J. Pediatric COVID-19 and pericarditis presenting with acute pericardial tamponade. World Journal for Pediatric and Congenital Heart Surgery. 2020;11 Suppl 6:802-4. DOI: https://doi.org/10.1177/2150135120949455
13. Citoni B, Digilio MC, Capolino R, Gagliardi MG, Campana A, Drago F, *et al.* SARS-CoV-2 and pre-tamponade pericardial effusion. Could sotos syndrome be a major risk factor?. Genes. 2021;12 Suppl 1782:1-5. DOI: https://doi.org/ 10.3390/genes12111782
14. Cantarutti N, Battista V, Adorisio R, Cicena M, Campanello C, Listo E, *et al*. Cardiac manifestations in children with SARS-COV-2 infection: 1-year pediatric multicenter experience. Children. 2021;8 Suppl 717:1-8. DOI: https://doi.org/10.3390/children8080717
15. Lamprinos N, Ladomenou F, Stefanaki S, Foukarakis E, Vlachaki G. Pericarditis following recovery from COVID-19 Infection in a 15-year-old boy: a postinflammatory immune-mediated presentation or a new-onset autoimmune disease. Cereus. 2021;13 Suppl 11:1-5. DOI: https://doi.org/ 10.7759/cureus.19255
16. Dimapoulou D, Spyridis N, Dasoula F, Krepis P, Eleftheriou E, Liaska M, *et al.* Pericarditis as the main clinical manifestation of COVID-19 in adolescents. Pediatr Infect Dis J. 2021;40:197-9. DOI: https://doi.org/10.1097/INF.0000000000003096
17. Regan W, Byrne LO, Stewart KS, Miller O, Pushparajah K, Theocharis P, *et al.* Electrocardiographic changes in children with multisystem inflammation associated with COVID-19. J Pediatr. 2021;234:27-32. DOI: https://doi.org/10.1016/j.jpeds.2020.12.033
18. Henrina J, Putra ICS, Lawrensia, Marta DS, Wijaya W, Cool CJ, et al. Cardiac manifestations, treatment characteristics, and outcomes of paediatric inflammatory multisystem syndrome temporally associated with severe acute respiratory syndrome coronavirus-2: A systematic review. Progress in Pediatric Cardiology. 2021;63:1-8. DOI: https://doi.org/10.1016/j.ppedcard.2021.101365
19. Nelson W, Pomerantz A, Bushy A. A proposed rural healthcare ethics agenda. J Med Ethics. 2007;33:136-9. DOI: https://doi.org/10.1136/jme.2006.015966
20. Erwin C, Aultman J, Harter T, Illes J, Kogan RCJ. Rural and remote communities: unique ethical issues in the COVID-19 pandemic. The American Journal of Bioethics. 2020;20 (Suppl 7):117-20. DOI: https://doi.org/10.1080/15265161.2020.1764139