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Management of postpartum preeclampsia complicated by idiopathic pericarditis: A case report

Lauren Fisher *, Eman Alnaggar

Fiona Stanley Hospital, Perth, 11 robin warren drive, Murdoch, WA 6150, Australia

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ABSTRACT

This case report discusses the rare occurrence of pericarditis with preeclampsia in the antepartum through to postpartum state. A woman in her 30s presented four days postnatally with positional central chest pain, elevated blood pressure and newly deranged liver function tests. Echocardiogram demonstrated new pleural effusion and she was diagnosed with preeclampsia and superimposed pericarditis. Her blood pressure was stabilised with a combination treatment regime of labetalol, enalapril and frusemide, whilst her pericarditis responded well to colchicine and ibuprofen. She was eventually discharge on enalapril and colchicine. By her 6-week follow-up she had made a full recovery and she had reported no recurrence of symptoms at the time of writing.

1. Introduction

Pericarditis is the inflammation of the pericardium, which typically presents with acute-onset shortness of breath and positional chest pain [1–3]. Diagnosis requires the presence of at least two of the following: chest pain (80–95%), typically positional, sharp, and relieved by sitting up and leaning forward; pericardial friction rub; electrocardiographic changes; and pericardial effusion [1–5]. Within developed countries, the cause of pericarditis is often idiopathic, with an assumed, but often unproven viral association (80%) [1–5]. A small proportion of cases are attributed to post-cardiac injury syndromes, connective tissue disease (5–10%), cancer (5%) and pericardial tuberculosis (<5%) [1–5]. One-third of cases have associated myocarditis (inflammation of the myocardium), resulting in elevated cardiac biomarkers (troponin I) [1–5].

The majority of those affected with idiopathic pericarditis go on to have good outcomes, with only 30% affected by disease recurrence [5–8]. There is variable evidence regarding gender predisposition. Women who are diagnosed often have associated autoimmune conditions (systemic lupus erythematosus or SLE amongst others), which increases their risk of recurrent pericarditis [7,8]. Whilst identifying predisposing factors for women in the antenatal and postpartum states is difficult given the limited body of literature on pericarditis in association with pregnancy, there is a growing body of literature on the cardiac complications of preeclampsia. Implications of the disease in both the antepartum and the postpartum state are further reaching than has been historically reported [9,10].

Preeclampsia is a syndrome characterised by development of hypertension (blood pressure >140/90) and end-organ dysfunction, typically proteinuria, at or after 20 weeks of gestation [9]. Hypothesised to be driven by abnormal development of placental vasculature, the subsequent release of antiangiogenic factors and resulting systemic endothelial dysfunction increases maternal blood pressure and drives dysfunction in other organ systems, including kidneys, liver, brain, cardiac, pulmonary and haematological systems [9]. Whilst some of these presentations are more common, recent research has shown a greater impact of preeclampsia on cardiac function than initially suspected [10]. These complications include but are not limited to hypertrophy, global dysfunction, congestive heart failure, and acute coronary syndromes [10,11]. A small but growing series of case reports document preeclampsia in the setting of pericardial diseases [12]. Like all obstetric conditions, diagnosis and management of pericarditis have implications for both mother and baby.

2. Case Presentation

A woman in her 30s presented with acute central chest pain four days after non-elective lower-segment caesarean section. On presentation to the emergency department, she described the chest pain as central, and first noticed it whilst lying flat. On further enquiry, she reported shortness of breath and clarified that the pain was relieved by sitting upright and leaning forward, and that the chest pain radiated to her neck

* Corresponding author.
E-mail address: lauren.fisher@health.wa.gov.au (L. Fisher).

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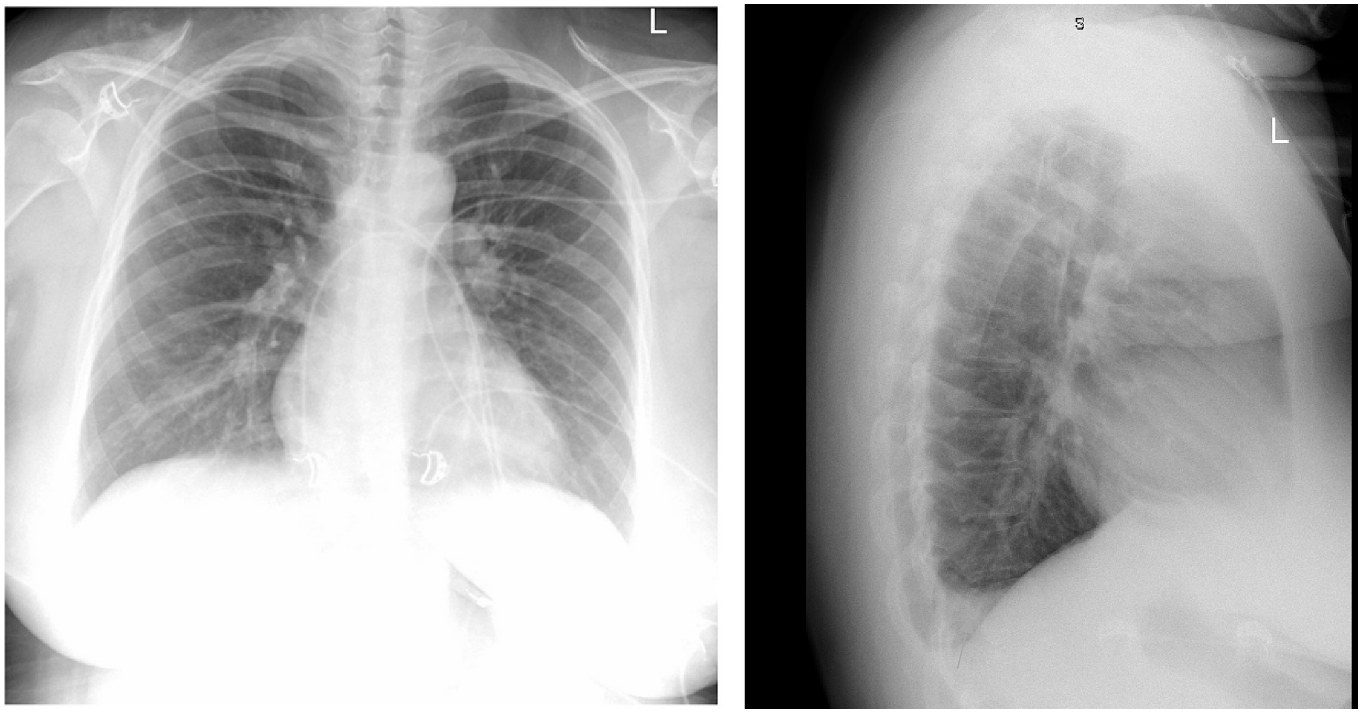

Fig. 1. CXR taken on arrival to the emergency department. Normal cardiac, mediastinal, and hilar contours reported. A small left pleural effusion can be seen, marked with thin arrow. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 1
Blood test results taken on arrival to the emergency department, in comparison with results at 6-week follow-up by the maternal heart clinic[18–19].

		On arrival to ED	6-week- follow-up	Normal range in third trimester of pregnancy	Units of measurement
Full blood picture	HB	100	126	110–150	g/L
	WCC	6.43	6.34	6–16	g/L
	Platelet	141	256	150–400	g/L
	MCV	88	89	81–99	g/L
	Haematocrit	0.36	0.38	0.23–0.4	g/L
Urea and electrolytes	Na	143	141	135–145	mmol/L
	K	3.9	4.2	3.5–4.5	mmol/L
	bicarbonate	26	28	22–25	mmol/L
	Urea	2.7	4.2	3–8	mmol/L
	EGFR	>90	>90	>90	
Liver function tests	Bilirubin	5	15	<22	g/L
	ALT	42		<21	g/L
	AST	43		<30	g/L
	GGT	26		<40	g/L
	Albumin	28	30	24–31	g/L
	LDH	325		115–211	IU/L
	Uric acid	0.36		<3.8	mg/dL
	D.dimer	1.54		0.4–2.2	mg/dL
Cardiac markers	Troponin I	2		<2	
	BNP	297		<200	Pg/mL

and shoulder. She denied any previous episodes of similar chest pain. She reported no recent viral illness, upper respiratory symptoms or sick contacts. Her past medical history included mild, unmedicated gestational hypertension, diagnosed antenatally, and hypothyroidism well controlled with thyroxine. She was otherwise well with no family or social history of note.

On arrival her observations were as follows; HR 72, BP 160–180/100, RR 16 with O2 97% on RA, afebrile. Her heart sounds were dual, with no additional sounds. There was no pain on palpation of the chest wall overlying the costochondral and costosternal joints. Auscultation of her chest demonstrated bilateral clear breath sounds. Peripheral vascular examination revealed bilateral pitting oedema measured up to her knees. An electrocardiogram (ECG) showed sinus rhythm and chest X-ray showed a small left-sided pleural effusion (Fig. 1).

Bloods taken on arrival demonstrated the following changes: a low haemoglobin of 100 g/L, normal platelets, white cell counts, and urea and electrolytes. Liver function tests, LDH and uric acid were mildly deranged in keeping with a new diagnosis of preeclampsia, though not severe enough to be concerning for haemolysis, elevated liver enzymes and low platelets (HELLP syndrome). A high-sensitivity troponin test was normal but her BNP and d dimer were both elevated (Table 1). A CT pulmonary angiogram was arranged given the history and positive d dimer test, which was negative for pulmonary embolism but showed a small pericardial effusion and bilateral pleural effusions in addition to left basal atelectasis (Fig. 2).

In light of these findings, urgent formal echocardiogram was arranged, which demonstrated a structurally normal heart, an ejection fracture of 60–65%, tricuspid regurgitation, mild pulmonary

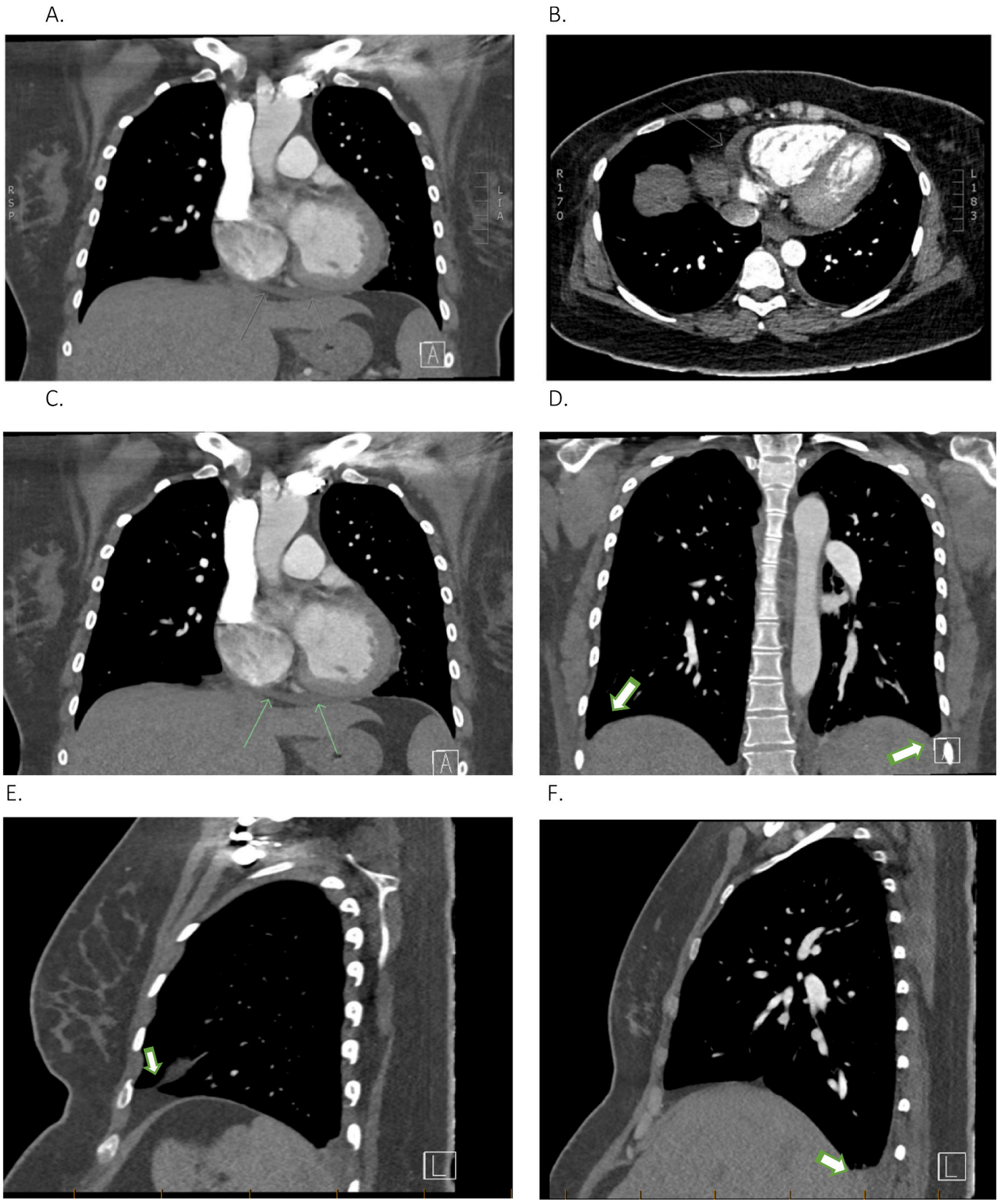

Fig. 2. A. and B. CTPA showing pericardial effusion with thin arrows, no features of right heart strain are observed. C. and D. Bilateral small right and left pleural effusions marked with thick arrows. E. Right lung with a small pleural effusion marked with thick arrow. F. Left lung showing basal subsegmental atelectasis with small ground-glass opacities adjacent to the left basal pleural effusion.

hypertension and a new pericardial effusion. The patient had presented with pathognomonic chest pain, and the combination of pathognomonic positional chest pain, with pericardial effusion, met 2 of the 4 diagnostic criteria for pericarditis [1–5]. Urgent referral to maternal heart specialists was sought and the diagnosis of preeclampsia and superimposed pericarditis was formalised.

The patient was immediately started on labetalol 200 mg three times daily and slow-release nifedipine 30 mg to stabilise her blood pressure. Labetalol was gradually tapered off during her hospital admission and enalapril 5 mg daily was commenced prior to discharge. Frusemide 40 mg was initially administered intravenously for fluid overload. She responded well, achieving a good diuresis, allowing the medication to be tapered off after 48 h. Ibuprofen and colchicine 500microg were commenced twice daily for management of pericarditis and continued for 6 weeks, at which time it was ceased at outpatient follow-up in the maternal heart clinic.

On review 1 month after discharge, the patient showed significant improvement in symptoms and blood pressure control. The autoimmune screen, including anti-nuclear antibodies, anti-cyclic citrullinated peptide, and rheumatoid factor, returned negative. Thyroid function tests remained within normal limits, with thyroxine doses unchanged postnatally. Having made a full recovery at this time she was discharged from clinic to the care of her general practitioner, with the recommendation to repeat her echocardiogram in 5 years to assess for progression of the tricuspid regurgitation.

3. Discussion

It is well established that women in the postpartum period are subject to increased morbidity from a variety of causes. The majority of hospital readmissions in this period are due to common events, uterine and wound infections, hypertension and preeclampsia [12]. Cardiomyopathy and exacerbations of chronic cardiac disease represent a smaller proportion of women hospitalised in this period [13]. Events such as pericarditis, especially superimposed on preeclampsia, remain very rare [5–8,12]. Despite a paucity of such events within the literature, new literature on cardiac complications of preeclampsia may shed light on this case.

For women with preeclampsia, new research reports increased rates of cardiovascular disease, from cardiac remodelling to myocardial infarction [10]. In the setting of severe and early-onset preeclampsia, left ventricular remodelling occurred in as many as 81% of patients [10,11] whilst segmental impaired relaxation occurred in 85%. Rates of hypertrophy are reportedly lower for both right and left ventricles, at ~50%, and impaired contractility occurs in 60% of patients [10,11]. Rates of myocardial infarction were also found to be increased 13-fold, and heart failure 8-fold [10,11]. Individual case reports of pericardial disease, specifically cardiac tamponade secondary to significant pericardial effusion, appear to be rising [13–16]. Whilst there are a handful of studies documenting the occurrence of idiopathic pericarditis in pregnancy, no association with preeclampsia has been documented. Of the individual cases reported in the literature, the majority have an autoimmune aetiology with underlying SLE [13–17]. In the present case, the patient was tested and found not to have any positive biomarkers for autoimmune conditions outside of hypothyroidism.

Limited numbers of case reports make it difficult to ascertain appropriate treatment and thus prognosis of pericarditis in both acute and recurrent forms. Treatment within the general population requires ibuprofen, aspirin or indomethacin in high doses administered either PO or IV [17]. Colchicine, for up to 6 months after diagnosis, is included in combination with a non-steroidal anti- inflammatory drug (NSAID) [15]. In instances of failure to respond to this combination, prednisolone is added as a third–line treatment at the lowest effective dose [17]. To counter the side-effects of long-term steroids, vitamin D and bisphosphates are highly recommended. In instances where all the above have failed, and use of IL1 receptor antagonists have been found to be effective [17]. Pending on the developmental timing and the aetiology of the pericarditis, differing treatment regimens are recommended.

For those in early stages of pregnancy, standard-dose aspirin and NSAIDS can be utilised safely up to 20 weeks. Thereafter, the use of NSAIDS is not recommended in order to avoid the risk of early ductus venosus closer and renal atresia [17]. Low-dose aspirin (100 mg per day) can be continued up to 36 weeks [17]. Prednisolone and colchicine can be utilised throughout pregnancy and breastfeeding safely, according to current evidence [17]. NSAIDS can be resumed after delivery. However, aspirin is preferably avoided [17]. A handful of studies document the use of anti-IL1 agents in pregnancy; two report on canakinumab and seven the use of anakinra [17]. All studies suggest outcomes that do not include risk of major malformation. Furthermore, use of anakinra in breastfeeding showed no obvious adverse effects [17]. Given that this evidence remains limited, current recommendations are based on clinical expertise and cost-benefit ratio of use.

4. Conclusion

This case describes the rare occurrence of pericarditis in association with preeclampsia in the postpartum state. It highlights that pericardial disease should not be dismissed as a differential diagnosis in those presenting with chest pain in the postpartum period. Early multidisciplinary team involvement resulted in urgent diagnosis and treatment, and eventual complete resolution of the disease without recurrence. Further investigation into the cause of postpartum pericarditis and the association with preeclampsia is recommended. Finally, studies on the use of steroids, colchicine and the more novel IL1 antagonists are required to establish safer dosing regimens and to improve long-term outcomes for mothers and their children alike.

Contributors

Lauren Fisher was involved in patient care, conception of the case, literature review, drafting of the manuscript and the editing process.

Eman Alnaggar was involved in patient care, the conception of the case report, and manuscript editing and revision.

Both authors approved the final submitted manuscript.

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Patient consent

Obtained.

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Conflict of interest statement

The authors declare that they have no conflict of interest regarding the publication of this case report.

References

[1] R.A. Lange, L.D. Hillis, Acute Pericarditis, N. Engl. J. Med. 351 (21) (2004) 2195–2202.

[2] M.M. LeWinter, Clinical practice. Acute pericarditis, N Engl J Med. 371 (25) (2014) 2410–2416.
[3] E. Bizzi, C. Picchi, G. Mastrangelo, M. Imazio, A. Brucato, Recent advances in pericarditis, Eur. J. Intern. Med. 95 (2022) 24–31.
[4] M.A. Marinella, Electrocardiographic manifestations and differential diagnosis of acute pericarditis, Am. Fam. Physician 57 (4) (1998) 699–704.
[5] J. Robinson, W. Brigden, Recurrent pericarditis, Br. Med. J. 2 (5600) (1968) 272–275.
[6] M. Imazio, A. Brucato, Management of pericarditis in women, Womens Health (Lond). 8 (3) (2012) 341–348.
[7] A. Ramdass, T. Qureshi, J. Matharu, J. Cury, V. Seeram, An unusual case of postpartum dyspnea, Chest 147 (2) (2015) e38–e43.
[8] Z.P. Arany, C.M. Walker, L. Wang, Case 22-2014, N. Engl. J. Med. 371 (3) (2014) 261–269.
[9] P. Gathiram, J. Moodley, Pre-eclampsia: its pathogenesis and pathophysiolgy, Cardiovasc. J. Afr. 27 (2) (2016) 71–78, https://doi.org/10.5830/CVJA-2016-009.
[10] K. Melchiorre, R. Sharma, B. Thilaganathan, Cardiovascular implications in preeclampsia: an overview, Circulation 130 (8) (2014) 703–714, https://doi.org/10.1161/CIRCULATIONAHA.113.003664 (PMID: 25135127).
[11] A. Vaught, L. Kovell, L. Szymanski, et al., Acute cardiac effects of severe pre-eclampsia, J. Am. Coll. Cardiol. 72 (1) (2018) 1–11.
[12] W.M. Callaghan, A.A. Creanga, E.V. Kuklina, Severe maternal morbidity among delivery and postpartum hospitalizations in the United States, Obstet. Gynecol. 120 (5) (2012) 1029–1036.
[13] Astrit Gashi, Case report of severe preeclampsia and associated postpartum complications, J. Case Rep. Stud. ISSN (2016) 2348–9820, https://doi.org/10.15744/2348-9820.4.408.
[14] R.F. Awalia, An Indonesian pregnant woman with systemic lupus erythematosus and cardiac tamponade: a case report, IJSCR (2022), https://doi.org/10.1016/j.ijscr.2022.107159. ISSN 2210-2612,.
[15] P. Hildbrand, C. Eigenmann, M. Gugger, H.P. Marti, Hullin, Pre-eclampsia with acute heart failure postpartum as primary manifestation of systemic lupus erythematosus, Eur. J. Heart Fail. 7 (2005) 1057–1058, https://doi.org/10.1016/j.ejheart.2004.11.012.
[16] D. Corradi, R. Maestri, F. Facchetti, Postpartum Churg-Strauss syndrome with severe cardiac involvement: description of a case and review of the literature, Clin. Rheumatol. 28 (6) (2009) 739–743.
[17] A. Brucato, M. Imazio, S. Curri, G. Palmieri, R. Trinchero, Medical treatment of pericarditis during pregnancy, Int. J. Cardiol. 144 (3) (2009) 413–414.