



Case Report

Cardiac tamponade: Better prognosis in association with pulmonary embolism: Case report

A. Barag ^{b,*}, S. Harouna Idrissa^b, N. Mahoungou-Mackonia^b, S. Arous^{a,b}, G. Bennouna^{a,b},
L. Azzouzi^{a,b}, R. Habbal^{a,b}

^a Faculty of Medicine and Pharmacy, Hassan II University of Casablanca, B.P. 5696, Casablanca, Morocco

^b Department of Cardiology, IBN ROCHD University Hospital, Casablanca, B.P. 5696, Casablanca, Morocco

ARTICLE INFO

Keywords:

Tamponade
Tuberculosis
Pulmonary embolism
Pulmonary arterial hypertension
Prognosis

ABSTRACT

The tamponade leads to an increase in intrapericardial pressure, which impairs the diastolic filling of the ventricles and reduces ejection. However, the association with pulmonary arterial hypertension, which in turn leads to an intracardiac hyper-pressure, constitutes a compensatory mechanism.

We report the case of a 23 year old patient followed for anemia due to martial deficiency for 2 years, who consulted us with right heart failure evolving for 5 months, a hemoptoic cough and chest tightness. The entire workup revealed a pericardial and pleural tamponade of tuberculous origin associated with a pulmonary embolism evaluated at 15% according to the Qanadli score. After stabilization of her hemodynamic state, the patient was put on anti-bacillary and anticoagulant treatment with a good improvement of her cardiopulmonary state. She was discharged after 1 month (satisfactory check-up), regularly followed up in outpatient clinic with check-up of hemostasis and cardiac echography every 2 weeks. She was declared cured of her tuberculosis after 6 months of treatment. There was no recurrence after two years of follow-up.

The combination of tamponade and pulmonary hypertension is synergistic in that it improves the patient's prognosis.

1. Introduction

Pericardial tamponade corresponds to the acute or subacute compression of the cardiac cavities by a rapid build-up of pericardial effusion, resulting in an abrupt increase in intrapericardial pressure, which impairs the diastolic filling of the ventricles and reduces ejection. Its etiologies are multiple, dominated by neoplasia in developed countries and by tuberculosis in the African context [1]. The positive predictive value of its pathophysiology is questioned when tamponade is associated with pulmonary arterial hypertension (PAH). Indeed, pulmonary embolism leads to pulmonary arterial hypertension with a consequent right heart intra-cavity hyper-pressure. PAH then acts as a protective mechanism against right-sided cavitory collapse due to tamponade. This association remains rare and was hardly described before 2002, as reported by V. Nguyen Ba et al. [2].

We report a case in which the etiology of tamponade was multifocal tuberculosis and review the different mechanisms intrigued in this

pathophysiology, making the prognosis of tamponade well improved in our patient.

2. Case presentation

Our work consists of a single case report and has been reported in accordance with SCARE 2020 criteria [3].

The patient was 23 years old and had a history of iron deficiency anemia. She consulted the cardiological emergency room for an aggravating dyspnea evolving for 5 months, relieved by anteflexion of the trunk, associated with a hemoptoic cough of small abundance (30 cc) and thoracic oppression, of recent appearance for 2 days, the whole evolving in a context of weight loss and feverish sensation.

On clinical examination, the patient was conscious, Glasgow Score Coma 15/15, good general condition. She had edema of the lower limbs up to mid-leg, turgidity of the jugular veins and hepato-jugular reflux, dyspnea stage 4 of the New York Heart Association (NYHA), blood

* Corresponding author. Department of Cardiology, IBN ROCHD University Hospital, Casablanca, Morocco.

E-mail address: barageamadou80@gmail.com (A. Barag ).

¹ Personal address: lot du parc Avenue 2 Mars, blvd anoual N 31 Bis Casablanca.

<https://doi.org/10.1016/j.amsu.2021.102410>

Received 18 April 2021; Received in revised form 15 May 2021; Accepted 16 May 2021

Available online 19 May 2021

2049-0801/  2021 The Authors. Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license

(<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

pressure at 80/60 mmHg, tachycardia at 135 beats/min, saturation at 86% in room air and 98% under 2L of O₂, respiratory rate at 35 cycles/min Temperature 38.5 °C and cardiopulmonary auscultation found muffled heart sounds and right pleural fluid effusion syndrome (abolition of vocal vibrations and vesicular murmur with dullness on percussion). ECG: sinus tachycardia at 142 beats/min, diffuse microvoltages and electrical alternation [Fig. 1]. Cardiac ultrasound: large pericardial effusion 23 mm opposite the right ventricle, 34 mm opposite the right atrium and 50 mm posteriorly, significant flow variation. The right ventricle was dilated to 48 mm basal diameter with moderate longitudinal systolic dysfunction without paradoxical septum, dilated vena cava to 26 mm noncompliant in pulmonary hypertension to 58 mmHg. Left ventricular systolic function was normal with LVEF = 55%. [Fig. 2].

Pericardiocentesis was performed after conditioning of the patient, with collection of 3100 ml of a citrine yellow fluid (exudate with 46 mg of protein with lymphocyte predominance 83% without objectified germ with a positive expert gene) coupled with pleural drainage of 1500 ml (exudate with 39 mg of protein, sterile), compensated by albumin infusion. A pleural puncture-biopsy was performed with anatomopathological study finding an epithelio-giganto-cellular granuloma without caseous necrosis.

Biological workup: Hb: 12 g/dl; WBC: 12,000 μ 3; Plq: 394,000 μ 3; CRP: 310 mg/L, D-Dimer: 5600 μ g/L, PT 87%; APTT: 23 sec, fibrinogen: 6.5 g/L. Urea: 0.46 g/L, creatinine 9.6 mg/L, Troponins: 37 ng/L.

In view of the persistence of dyspnea despite pericardiocentesis associated with signs of acute pulmonary cardiopathy, a thoracic angioscanner was performed, objectifying a left inferior segmental lobar pulmonary embolism and the right intermediate branch evaluated at 15% according to the Qanadli score [Fig. 3]. The echodoppler of the lower limbs came back normal, without peripheral abnormality. The complement by ADA assay in the pericardial puncture fluid was positive at 96 IU/L.

In view of this picture, the diagnosis of pericardial and pleural tamponade of tuberculous origin associated with pulmonary embolism was made.

After a specialist opinion in pneumology and physiology, the patient was put on antibacillary treatment for 6 months, i.e. 2 months of EHRZ and 4 months of RH associated with anticoagulant treatment with unfractionated heparin by weighted dose, then on antivitamin K with close monitoring of the haemostatic balance. Clinically, the evolution was marked by the total regression of dyspnea, the absence of recurrence of cough, and the improvement of blood pressure figures. Daily cardiac

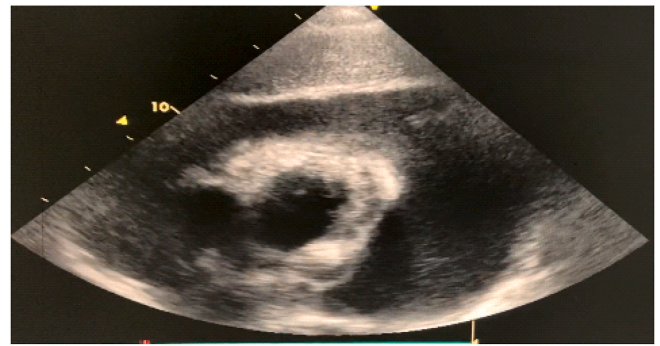


Fig. 2. Echocardiographic image showing an important pericardial effusion with swinging heart.

ultrasound monitoring confirmed the progressive regression of the effusion and the thoracic angioscanner (after 3 months) showed that the pleural effusion had subsided and that there was no sign of pulmonary embolism.

The patient was declared discharged after 1 month of treatment with regular follow-up in an outpatient cardiology clinic. After 2 years, there was no recurrence.

3. Clinical discussion

Tamponade is an acute and subacute compression of the cardiac chambers by the rapid appearance of a pericardial effusion, usually of great abundance with an increase in intrapericardial pressure, which impedes the filling of the right ventricle (RV), leading to an increase in the diastolic and mean pressure of the right ventricle-right auricle (RA) [3,4]. This is an absolute medical-surgical emergency with a life-threatening prognosis and the morbi-mortality of this condition is highly dependent on the speed of diagnosis.

Clinically, it appears as cardiogenic shock related to adiastrale and low flow, of which the paradoxical pulse is the pathognomonic sign [4, 5] associated with signs of right ventricular failure (RVF). Ultrasound signs are essentially two-dimensional, a "beating heart" appearance associated with collapse of the RV in protomesodiastole and the RA in telediastole with a plethoric inferior vena cava (IVC). Pulsed Doppler shows flow variation with an increase (>40%) in tricuspid and pulmonary velocities, an inspiratory decrease (>20%) in transmitral and subaortic Doppler velocities, and an inspiratory inversion of the E/A

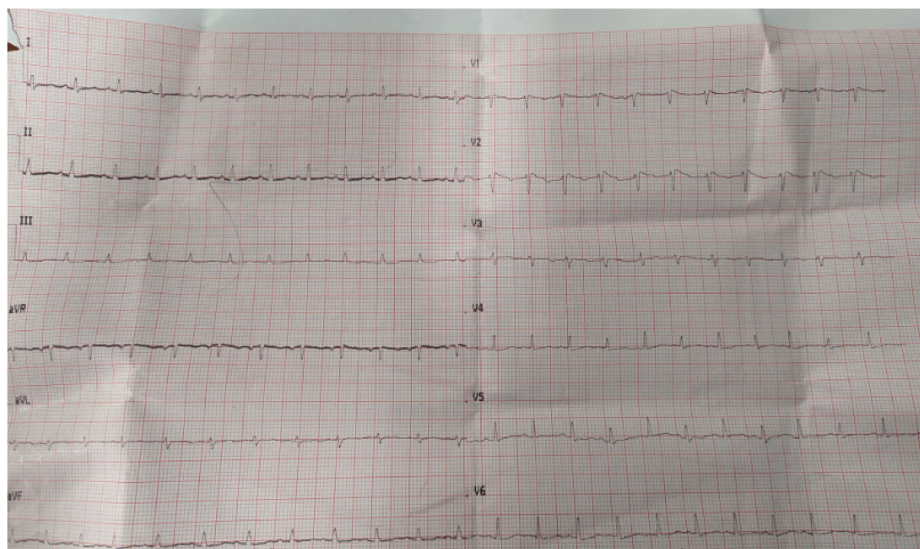


Fig. 1. ECG 12 derivations notifying sinus tachycardia at 137 beats per minute and diffuse microvoltage

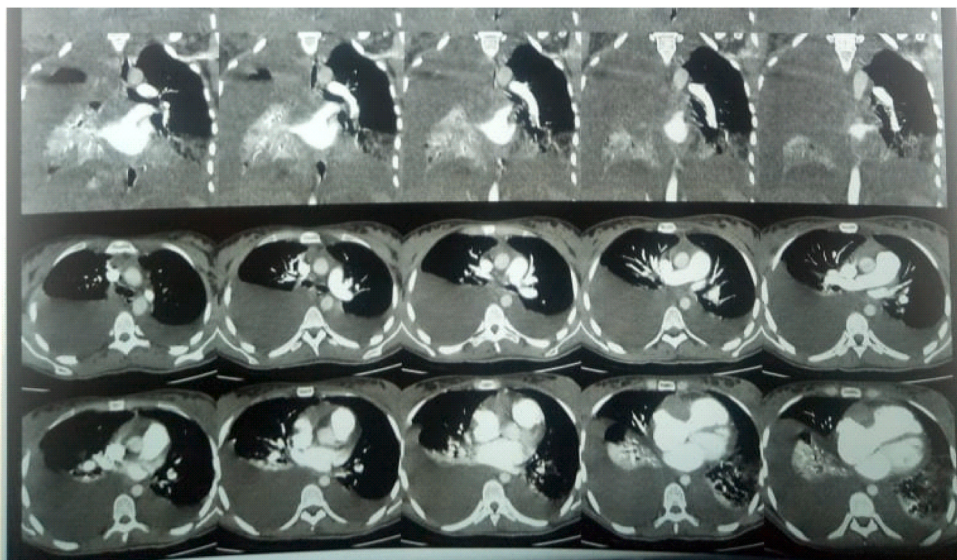


Fig. 3. Thoracic angioscanner with parenchymal window showing a left lower segmental lobar pulmonary embolism and the right intermediate branch.

ratio on the mitral and tricuspid Doppler profiles. In practice, these Doppler signs are most often difficult to objectify when the effusion is abundant, given the mobile nature of the heart within the pericardial sac [3,4]. In our patient, we objectified cardiogenic shock with signs of DRV. Transthoracic echocardiography showed a very abundant effusion at 50 mm with a dancing heart, left ventricular collapse and flow variation, all over a plethoric vena cava. Such an abundance of effusion suggests that it was built up very gradually over the 5 months following the onset of the symptomatology, which would have allowed the pericardium to adapt to avoid the hemodynamic repercussions of the major compression of the right chambers by the effusion.

The etiologies of tamponade are dominated by neoplasia and tuberculosis. The latter remains dominant in the African context (60%) [1], and must be sought especially in case of insidious evolution in young patients in endemic areas. In our patient, the medical etiological diagnosis of pleural and pericardial tuberculosis was confirmed; the evolution being according to a progressive installation mode. On the other hand, we noted the presence of contradictory echographic signs, namely: a dilated RV at 48 mm in moderate longitudinal systolic dysfunction in strong PAH with absence of paradoxical septum and collapse of the right cavities. They can have only one explanation: an increase in resistance in the pulmonary arteries increasing the pressure in the RV, possibly related to left heart valve disease resonating in the right heart, chronic obstructive pulmonary disease (COPD), PAH-related systemic diseases, and pulmonary embolism. Our patient had no notion of systemic disease or COPD, no valvulopathy objectified on transthoracic echography, only pulmonary embolism could have this explanation. Indeed, pulmonary arterial hypertension (PAH) raises the pressures of the right ventricle and the right atrium. These pressures are then sufficient to resist the collapse of the right cavities by the pericardial effusion. Transmural pressure in the right cavities is temporarily maintained (between high intraventricular pressure due to PAH and high intrapericardial pressure due to effusion). This prevents restrictive shock leading eventually to asystole by acting as a protective effect [3, 6]. Our results corroborate with data from the literature which report that the presence of PAH reduces the echographic diagnostic value of pericardial tamponade by mask effect.

AKINCI et al., in 2002 [5], reported the case of a patient who developed porto-pulmonary hypertension after liver transplantation complicated by cardiogenic shock. On transthoracic echocardiography, a pericardial effusion was objectified without RV collapse without further echographic evidence of tamponade. Pericardiocentesis

improved the clinical picture. Pulmonary hypertension was a protective factor of the right cavities to the external pressures exerted by the pericardium as in our patient where there was an absence of collapse of the right cavity. On the other hand, Frey et al., in 1989, reported a case of primary PAH and dyspnea in a patient for whom tamponade was suspected because of the evidence of LV collapse while the right cavities were dilated as in our patient. This could be explained by the fact that the intrapericardial pressures were lower than the pressures in the right cavities and higher than those of the LV. Finally, V. Nguyen Ba et al reported in 2003 the case of early contradictory signs associated with tamponade, i.e. moderate LV collapse without paradoxical septum as in our patient, related to PAH caused by pulmonary embolism. The etiological diagnosis of this association between pulmonary embolism and tamponade was pulmonary neoplasia, reported by several studies [3], in contrast to our study, where tuberculosis was the etiology of this association due to its thromboembolic nature [7–9]. Our observation is therefore a relationship between tamponade and pulmonary embolism, with multifocal tuberculosis as the etiological diagnosis. Pulmonary embolism was a protective factor for the patient against the severity of tamponade.

4. Conclusion

Pericardial tamponade and pulmonary embolism are two serious pathologies. The association between the two is rare and of various etiologies. Pulmonary embolism is therefore a survival factor for the patient because of the PAH it provides. In all cases, management must be done as soon as possible because the heart is subjected to a double pressure: intrapericardial and intracardiac.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this

journal on request.

Sources of funding

The authors declared that this study has received no financial support.

Author statement

Barage Amadou: Corresponding author writing the paper.
 Harouna Idrissa. Seydou: writing the paper.
 Mahoungou-Mackonia Noel: writing the paper.
 Arous Salim: writing the paper.
 Bennouna Ghali: writing the paper.
 Azzouzi Leila: writing the paper.
 Habbal Rachida: Correction of the paper.

Registration of research studies

1. Name of the registry: researchregistry
2. Unique Identifying number or registration ID: 6756
3. Hyperlink to your specific registration (must be publicly accessible and will be checked):

Guarantor

BARAGE AMADOU.

Declaration of competing interest

Authors of this article have no conflict or competing interests. All of the authors approved the final version of the manuscript.

References

- [1] A. Kinga, E. Ayo Bivigou, C. Allognon, J.R. Mackanga, J.B. Mipinda, J.E. Eke Nzengue, *Epid miologie des  tiologies des p ricardites liquidiennes   Libreville*, *Health Sci. Dis.* 21 (9) (Septembre 2020) 35–38.
- [2] Ba V. Nguyen, B. Debien, T. Villevieille, G. Quiniou, L. Pollet, B. Lenoir, et al., *L'embolie pulmonaire au secours de la tamponnade cardiaque*, *Ann Fr Anesth R animation*. d c 22 (10) (2003) 896–899.
- [3] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, for the SCARE Group, *The SCARE 2020 guideline: updating consensus surgical Case REport (SCARE) guidelines*, *Int. J. Surg.* 84 (2020) 226–230.
- [4] S. Akhbour, B.A. Khennine, L. Oukerraj, J. Zarzur, M. cherti, *Pericardial Tamponade and Coexisting Pulmonary Embolism as First Manifestation of Non-advanced Lung Adenocarcinoma*, *Internet, Pan Afr Med J* (2014) 18. cit  18 d c 2020].
- [5] S.B. Akinci, S.P. Gaine, W. Post, W.T. Merrit, H.P. Tan, B. Winters, *Cardiac tamponade in an orthotopic liver recipient with pulmonary hypertension**, *Crit Care Med.* mars 30 (3) (2002) 699–701.
- [6] M.U. Khan, R.N. Khouzam, *Protective effect of pulmonary hypertension against right-sided tamponade in pericardial effusion*, *South Med J.* janv 108 (1) (2015) 46–48.
- [7] N. Dermeh, N. Djami, R. Amrane, A. Fissah, *Tuberculose pulmonaire et maladie thromboembolique une association non fortuite*, *Rev Mal Respir.* janv 35 (2018) A263.
- [8] J. Ben Amar, B. Dahri, H. Aouina, H. Bouacha, *Maladie veineuse thromboembolique au cours de la tuberculose*, *Rev Pneumol Clin.* d c 71 (6) (2015) 327–334.
- [9] C. Dentan, O. Epaulard, D. Seynaeve, C. Genty, J.L. Bosson, * tude r trospective de l'association entre tuberculose et maladie thromboembolique veineuse selon la classification internationale des maladies, neuvi me r vision*, *Rev M decine Interne.* d c 35 (2014) A54–A55.