Open Access

Stechnolock: Cardiology and Cardiovascular Medicine

CASE REPORT

Concomitant Pulmonary Embolism and Aortic Dissection: Diagnostic and Therapeutic Difficulties

Soya E[1,2], Boka KJM[1], Vy L[1], N'cho-Mottoh MPB[1,2], Daniogo M[1], Ekou A[1,2], Yao H[1,2], Kra L[1], Ghassani A[3], Kotchi Joël Michée BOKA[1,2]

[1]Medical Intensive Care Service, Abidjan Heart Institute, BPV 206 Abidjan, Ivory Coast

[2]Training and Research Unit in Medical Sciences, Félix Houphouët-Boigny University, 01 BPV 34 Abidjan 01, Ivory Coast

[3]Department of Vascular Surgery, Abidjan Heart Institute, BPV 206 Abidjan, Ivory Coast

[*]**Corresponding Author:** Kotchi Joël Michée BOKA, Medical Intensive Care Service, Abidjan Heart Institute, University Hospital of Treichville, 01 BPV 03 Abidjan 01, Ivory Coast. Tel.: 00 225 07 08 71 32 81, E-mail: joelmicheeboka@gmail.com

Citation: Soya E, Boka KJM, Vy L, N'cho-Mottoh MPB, Daniogo M et.al (2023). Concomitant Pulmonary Embolism and Aortic Dissection: Diagnostic and Therapeutic Difficulties. Stechnolock: Cardiol and Cardiovasc Med. 1-10

Abstract

Background: Pulmonary embolism and aortic dissection are two life-threatening cardiovascular emergencies that are difficult to distinguish clinically, especially when they coexist, constituting atherapeutic dilemma in the absence of a clearly defined protocol in the literature. We report a similar case diagnosed and successfully managed at the Abidjan Heart Institute.

Case report: This was a 55-year-old patient who presented with infarct-like pain associated with dyspnea, which motivated his admission to the cardiology emergency room. The diagnosis of acute coronary syndrome without permanent ST-segment elevation with positive troponin was retained on the basis of the electrical changes and the rise of the myocardial necrosis markers, but without any significant substrate at coronary angiography the day after his admission. The evolution undertreatment marked by the persistence of the symptoms led to the realization of a complementary assessment in particular echography making suspect a pulmonary embolism, confirmed byangioscanner with in bonus, the coexistence of a dissection of the aorta of type B of Stanford, successfully managed by the thoracic endovascular aortic repair (TEVAR) on the basis of the refractory pain, leaving free course to the optimal treatment of the pulmonary embolism by effective anticoagulation

Conclusion: Pulmonary embolism and simultaneous type B aortic dissection remains a diagnostic and therapeutic dilemma whose management has been revolutionized by recent advances in TEVAR that seem to improve hospital mortality.

Keywords: Pulmonary; embolism-Aortic dissection-anticoagulation-TEVAR.

Background

Diseases of the heart, aorta, lungs, esophagus, stomach, mediastinum, pleura, and abdominal viscera can all cause chest discomfort [1]. Recognition and exclusion of life-threatening etiologies of chest pain such as acute coronary syndrome, acute aortic dissection, and pulmonary embolism, based on a thorough analysis of the medical history, with particular emphasis on the onset and quality of pain and associated symptoms, followed by a detailed physical examination are of paramount importance for appropriate and early management [1]. However, these pathologies can be difficult to distinguish clinically, especially when they coexist. Stanford type B aortic dissection, which is less frequent than type A [2,3] and pulmonary embolism, although very rare, can present simultaneously and constitute a real therapeutic dilemma, being pathologies with incompatible treatments. Indeed, the anticoagulation indicated in case of pulmonary embolism may increase the hemorrhagic risk in case of aortic dissection, and the harmful therapeutic abstention related to embolism [1,4,5]. We report a similar case diagnosed and successfully managed by Thoracic Endovascular Aortic Repair (TEVAR) at the Abidjan Heart Institute. To our knowledge, very few authors have previously describedthis exceptional association in the literature [1,4-10].

Case Report

This was a 55-year-old patient whose history included known arterial hypertension for more than 10 years on fixed triple therapy, namely perindopril arginine, indapamide, and amlodipine, and knee arthritis on a combination of crystallized colchicine, tiemonium methylsulfate, and opium.

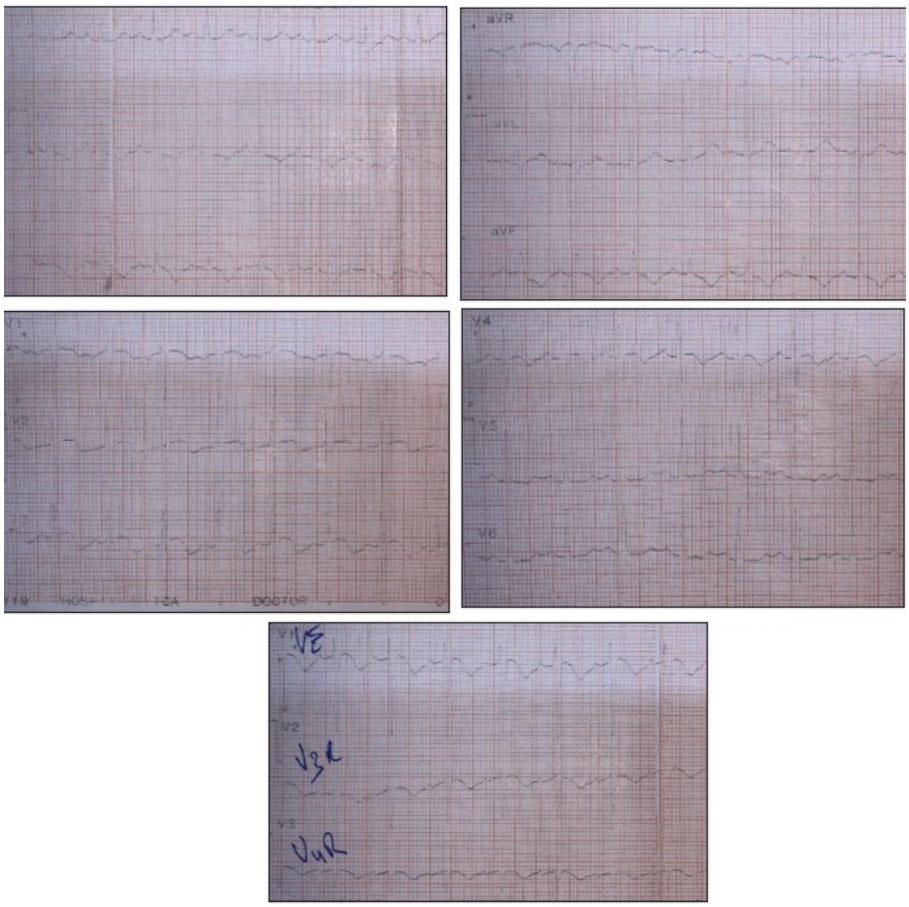

Figure 1: Sinus tachycardia and negative T waves in the anteroseptal, apical and inferior territories with electrical extension to right ventricle

He was admitted to the emergency department of the Abidjan Heart Institute for investigation of constrictive, retrosternal, prolonged chest pain radiating to the left arm, associated with dyspnea. The clinical and paraclinical investigations had revealed : infarct chest pain, elevated blood pressure figures at 149/107 mmHg, fine crepitus rales at the pulmonary bases, electrocardiographic abnormalities of sinus tachycardia at 115 bpm and negative T waves in the anteroseptal, apical and inferior territories

with electrical extension to the right ventricle (Figure 1), and radiological abnormalities in particular unwinding of the aortic knob and blunting of the right costophrenic angle (Figure 2).

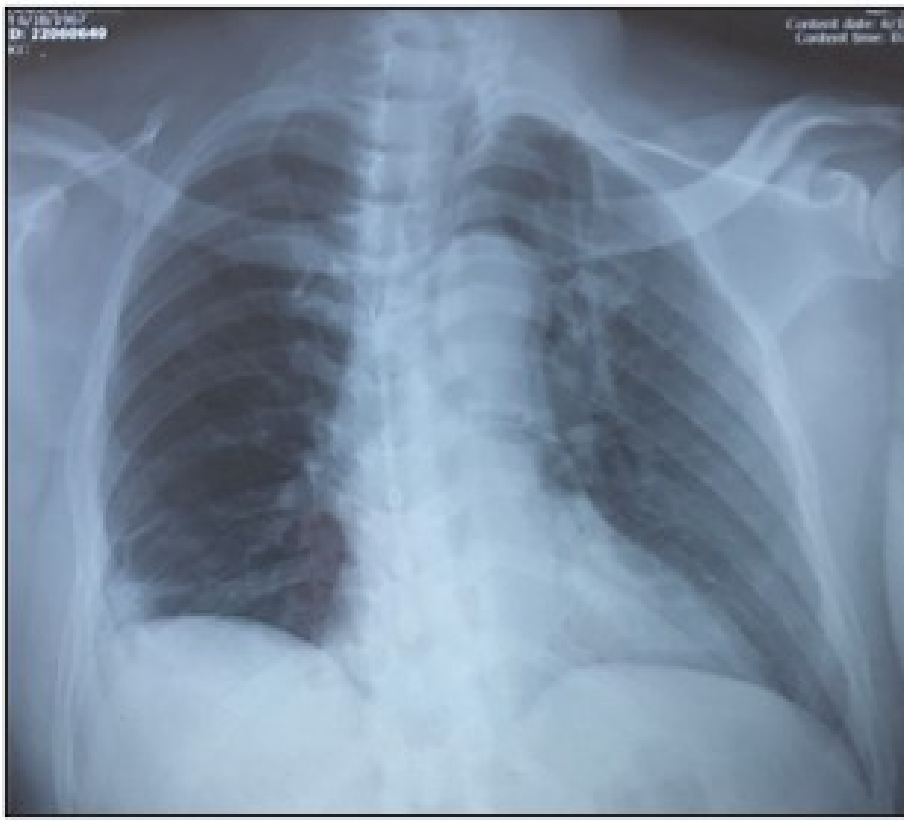

Figure 2: Frontal chest radiograph showing unwinding of the aortic button with blunting of the right costophrenic angle.

The biological workup showed an ultrasensitive troponin I level elevated to 94.3 ng/L at first contact (H0), then to 122 ng/L at the third hour (Normal Value: < 2 ng/L), with creatinine at 15.8 mg/L (Normal Value: 6-14 mg/L), either MDRD clearance at 59.02 mL/min, a hemoglobin level of 16 g/dL (Normal Value: 12-18g/dL), platelets at 161.10^3 elements/mm^3 (Normal Value: $150\text{-}400.10^3$) and a spontaneous prothrombin level of 64 % (Normal Value: 65-100 %).The diagnosis of troponin-positive non-ST-segment elevation acute coronary syndrome (NSTEMI) complicated with Killip stage II and at high ischemic risk was made. The patient was hospitalized in a cardiac intensive care unit with multiparametric monitoring and drug therapy consisting of bisoprolol, acetylsalicylic acid, Atorvastatin, Ramipril, Enoxaparin sodium, Furosemide and Tramadol. In addition, the myocardial fraction of creatine kinase (CK-MB) was 17.7 IU/L (Normal Value: 25 < IU/L) and the coronary angiography performed the day after his admission highlighted a coronary network free of lesions (Figure 3). Re-evaluation transthoracic echocardiography revealed dilated right heart chambers with a hypokinetic ventricle (TAPSE = 16 mm), dilatation of the pulmonary artery trunk and infundibulum with thrombus at the trunk, elevated pulmonary arterial pressures and resistances (SPAP = 71 mmHg, Vmax IT/ITV s/pulm = 0.37) (Figure 4), raising suspicion of pulmonary embolism.

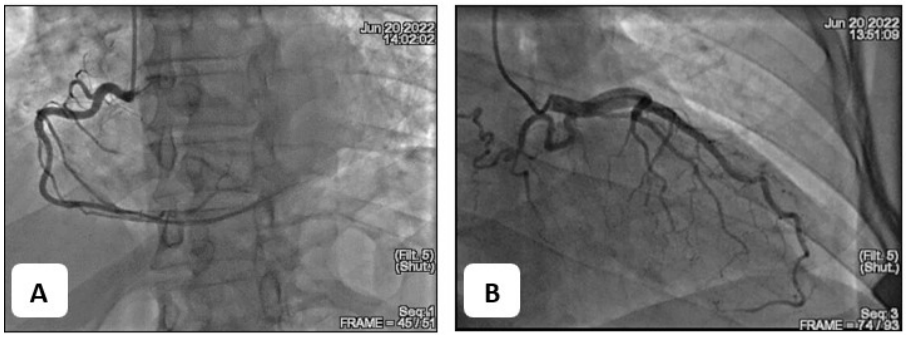

Figure 3: Coronary angiography demonstrating a dominant right (A) and left (B) coronary network free of lesions

Thoracic angioscan performed confirmed massive bilateral proximal pulmonary embolism with pulmonary hypertension, but as an added bonus, coexisting dissection of the descending and abdominal aorta, either a Stanford type B, with thrombosis of the false channel (Figure 5).

The progession on drug therapy marked by persistent dyspnea with a percutaneous saturation of 85% under 3l/min of pure oxygen, tachycardia at 110 bpm, and refractory basithoracic pain prompted placement of a covered stent graft in the descending aorta via the femoral approach with exclusion of the aortic dissection portal of entry at the foot of the left subclavian (figure 6)

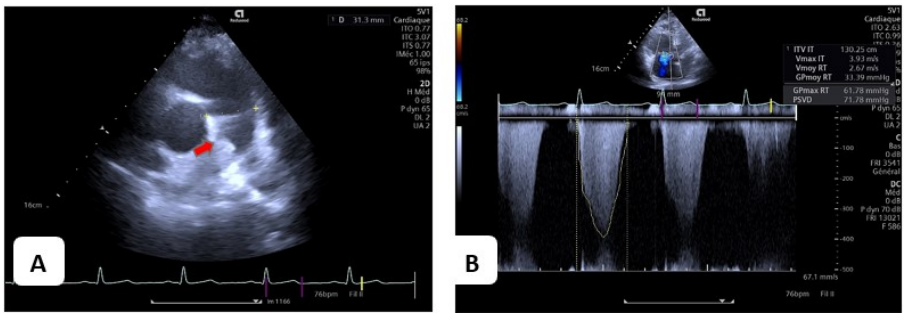

Figure 4: Transthoracic ultrasound showing a clot in the trunk (red arrow) with dilatation of the pulmonary artery trunk and infundibulum (A), elevated arterial pressures and pulmonary resistances (B).

Additional drug therapy included Rivaroxaban substituting for Enoxaparin sodium. The evolution under effective anticoagulation after stenting was normalization of percutaneous saturation to 97% on room air and regression of signs of call, notably dyspnea and basithoracic pain, with blood pressure at 107/69 mmHg, heart rate at 85 bpm and hemoglobin at 10.8 g/dl.

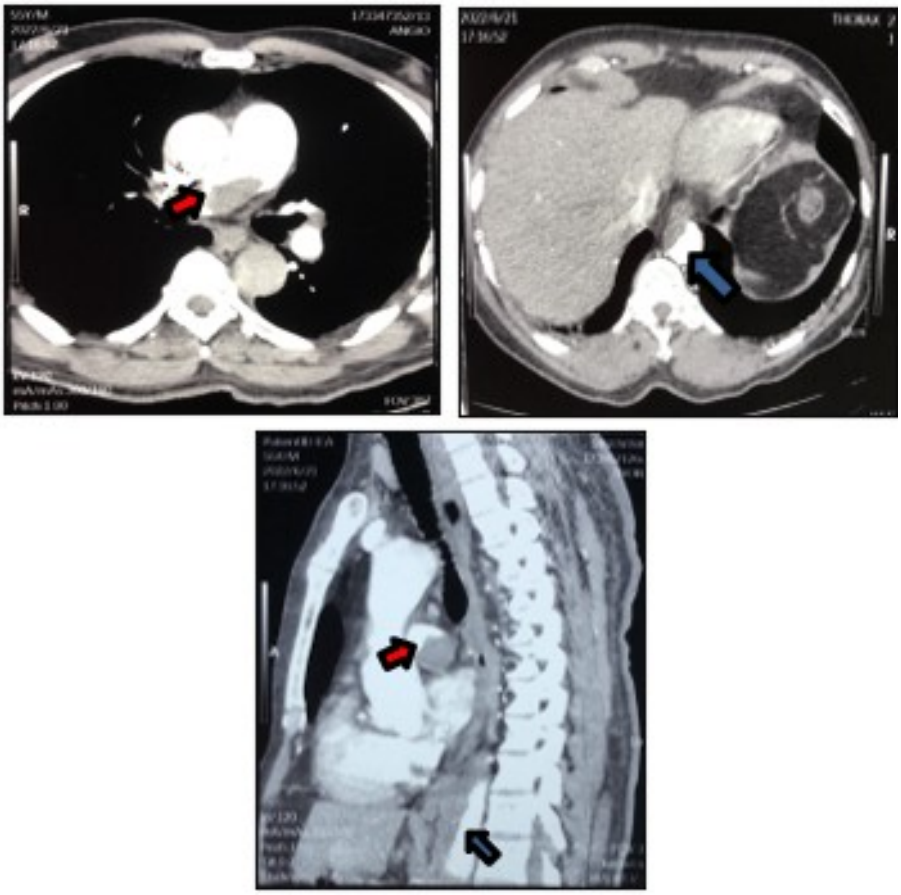

Figure 5: Angioscan confirming massive bilateral proximal pulmonary embolism (red arrow) and coexisting dissection of the descending and abdominal aorta (Stanford type B), with thrombosis of the false duct (blue arrow)

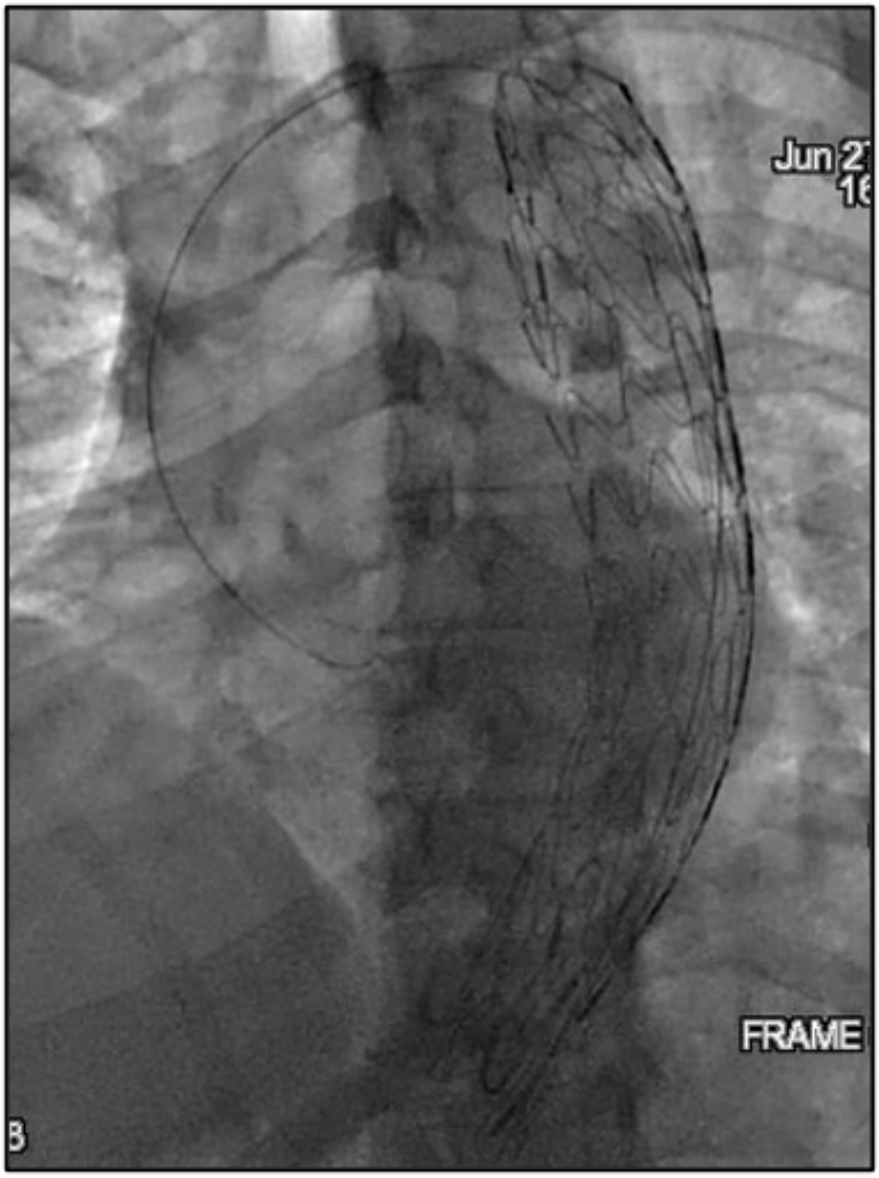

Figure 6: Angiography showing the covered stent in the descending aorta.

Discussion

Pulmonary embolism (PE) results from partial or total obliteration of the pulmonary artery or one of its branches by a circulating foreign body, most often of fibrino-cruciate origin [11]. PE results in a high percentage of underdiagnosed deaths each year because its positive diagnosis is elusive and the differential is very wide [12]. As observed in our case report, this disease can simulate a coronary syndrome with electrical changes and elevation of myocardial necrosis markers without significant substrate on coronary angiography, causing misdiagnosis and mismanagement. Fadoua Mouedder and al, observed a similar case in 2019 in Morocco [13]. This would testify to the diagnostic difficulty of pulmonary embolism, its clinical presentation not being pathognomonic and being able to simulate other medical-surgical emergencies.

Aortic dissection corresponds to a longitudinal cleavage of the aortic wall at the level of the media from an intimal breach which constitutes a portal of entry [14]. A false channel is then formed as opposed to the true channel or lumen, where blood under pressure enters the neoformed cavity, thus favoring the extension of the dissection. The influx of blood is limited by thrombosis of the portal of entry, which is beneficial to the patient [14]. There are several classifications, but the most widely used are those of Stanford and DeBakey because of their therapeutic application. The Stanford classification distinguishes two types according to the location of the entry point: type A, when the entry point is located on the ascending aorta; and type B,

when the entry point does not involve the ascending aorta.

DeBakey's classification subdivides the dissection process, with type I dissection involving the entire aorta, type II dissection involving only the ascending aorta, and type III dissection sparing the ascending aorta and arch. DeBakey types I and II correspond to Stanford type A, and type III to Stanford type B [15,16]. Aortic dissection and pulmonary embolism are both life-threatening cardiovascular emergencies. Although both cases may present with atypical chest pain, aortic dissection or pulmonary embolism are the chief presenting complaints and the other is an incidental finding in most cases. Symptoms of pulmonary embolism were more prominent and aortic dissection was an incidental finding, in our study.

This exceptional coexistence is rare and hardly discussed in the literature. Indeed, since 2005 when the first observation was reported by Hsin-Bang Leu and Wen-Chung Yu, in Taiwan, we present to our knowledge the ninth case in the literature in a 55-year-old man from the Ivory Coast [1,4-10]. Although aortic dissection is a rare condition, Stanford type A is predominant [2,3].

Indeed, five of the eight cases described in the literature were type A dissections associated with pulmonary embolism and only three were type B [4,5,10,15,16]. Our patient is the fourth type B reported and the only one that has benefited from thoracic endovascular aortic repair to our knowledge [1,4-10]. The typical patient profile appears to be elderly male and the most common risk factor is hypertension. In fact, like our patient, seven of the eight reported cases had hypertension [10,15]. Aortic dissection is usually observed in hypertensive patients [17].

The management of aortic dissection depends on the type. Stanford type A constituting a cardiovascular surgical emergency while type B, uncomplicated, with pain as the only sign, is usually managed medically [17]. However, type B aortic dissection can be complicated, defined by the occurrence of aortic rupture, target organ ischemia (stroke, limb ischemia, spinal cord ischemia, digestive ischemia, renal ischemia), poorly controlled hypertension, rapid aneurysmal transformation, or persistent or refractory pain, requiring surgical management [18, 19]. The mainstay of pulmonary embolism management is anticoagulation, and thrombolytic therapy may be necessary in cases of poorly tolerated embolism [20]. However, when pulmonary embolismis accompanied by aortic dissection, anticoagulation cannot be administered because of the risk of recanalization or re-dissection of the thrombosed false lumen [21]. Indeed, a thrombolytic could lyse the thrombus obstructing the false lumen or an anticoagulant could maintain the bleeding and prevent the thrombosis of the portal of entry, which constitutes a real therapeutic dilemma. In the absence of a clear protocol in the face of this dilemma, the management of this patient was initially antihypertensive medical treatment, initiated in accordance with the recommendations, which was aimed at reducing the ejection force of the left ventricle and the level of arterial pressure [22] thereby reducing the deleterious effect of arterial pressure and tachycardia on the vascular wall, and heparino-phylaxis to prevent subsequent thrombosis formation.

As a matter of fact, in aortic dissections, the formation of a thrombosis of the portal of entry can be considered as a beginning of healing [23]. It would therefore have been dangerous to jeopardize this early healing by effective anticoagulation outside of surgical management.

Moreover, the angioscanner having revealed a massive bilateral proximal pulmonary embolism, inview of the need for effective anticoagulation, desaturation and persistent thoracic pain under medical treatment, we opted in this patient in the acute phase for a thoracic endovascular aortic repair (TEVAR).

The purpose of this endovascular repair was to stabilize the dissection, limit circulation in the false channel, and promote the evolution of its thrombosis, leaving room for optimal treatment of the pulmonary embolism with effective anticoagulation. Fattori and coll, in a study of International Registry of Acute Aortic Dissection (IRAD) comparing medical treatment versus endovascular treatment, on 1129 patients with type B aortic dissection, evaluated between 1995 and 2012, showed a lower mortality rate in the TEVAR group (n= 276 or 24.4 %) versus medical treatment alone (n= 853 or 75.6 %) going from an estimated mortality of 15.5% in the TEVAR group versus 29.0 % in the medical group (p= 0.018) [24].

Even if medical management remains the treatment of choice for type B aortic dissections, known as uncomplicated aortic dissections, TEVAR is tending to be used more and more in uncomplicated forms. Recent progress in TEVAR seems to improve in-hospital mortality and prevent the occurrence of long-term complications (aneurysm, extension of the dissection) [24].

Conclusion

The coexistence of pulmonary embolism and Stanford type B aortic dissection remains a morbid and rare association, of difficult diagnosis, as the clinical and paraclinical manifestations may simulate several other medical-surgical emergencies and and delay diagnosis and optimal management, which however remains a real therapeutic dilemma, little discussed in the literature and without a clearly defined protocol. In our study, the initiation of curative anticoagulation after TEVAR could be proposed as a therapeutic course of action, subject to hemodynamically stable pulmonary embolism. Although TEVAR remains the treatment of choice for complicated type B aortic dissections, as in this case, it is increasingly used in uncomplicated forms.

Conflict of Interest

None

Author Contribution

Soya Esaïe: critical review of the article, approval of the final version of the article, responsible for all aspects of the work

Boka Kotchi Joël Michée: conception of the work, writing of the article, responsible for all aspects of the work.

Vy-Légré: data acquisition, responsible for all aspects of the work.

N'cho-Mottoh Marie-Paule: critical revision of the article, approval of the final version of the article, responsible for all aspects of the work.

Daniogo Mbe: data acquisition, responsible for all aspects of the work.

Ekou Arnaud: critical review of the article, approval of the final version of the article, responsible for all aspects of the work.

Yao Herman: critical review of the article, approval of the final version of the article, responsible for all aspects of the work.

Kra Lossan: data acquisition, responsible for all aspects of the work.

Ghassani Afif: critical review of the article, responsible for all aspects of the work

KONIN Christophe: critical revision of the article, approval of the final version of the article, responsible for all aspects of the work.

References

1. Ramponi F, Papps T, Edwards J (2018). Successful Repair of Concomitant Acute Type A Aortic Dissection and Saddle Pulmonary Embolism. Aorta (Stamford) 6: 34-6.

2. HowardDPJ, Banerjee A, FairheadJF, Perkins J, Silver LE et.al (2013). Population-Based Study of Incidence and Outcome of Acute Aortic Dissection and Premorbid RiskFactorControl: 10-Year Results From the Oxford Vascular Study. Circulation 127: 2031-7.

3. Soya E, Tro G, N'Djessan JJ, Boka B, Dabiogo M et al (2021). Epidemioclinical characteristics and therapeutic management of aortic dissection at the Abidjan Heart Institute. Cah Santé Publique 20: 62-9.

4. Chaulagai B, Acharya D, Poudel S, Puri P (2021). Simultaneous aortic dissection and pulmonary embolism: a therapeutic dilemma. Cureus 13: e12952.

5. Ng E, Ekladious A, Wheeler LP (2019). Thrombus risk versus bleeding risk: a clinical conundrum. BMJ Case Rep 12: e228344.

6. Tudoran M, Tudoran C (2016). High-risk pulmonary embolism in a patient with acute dissecting aortic aneurysm. Niger J Clin Pract 19: 831-3.

7. Herrera RN, Miotti JA, Pereyra AS, Lobo MV, Ibarra MT, Tomé Guzmán AF (2012). Marfan syndrome associated with aortic dissection, venous thromboembolism and hyperhomocysteinemia. Medicina (BuenosAires) 72: 478-80.

8. Masinarivo DR, Rakotomanana JL (2017). Clinico-electrocardiographic appearance of pulmonary embolism masking aortic dissection revealed by chest angioscan. Pan Afr Med J 28: 3.

9. Leu HB, Yu WC (2005). Images in cardiology: massive pulmonary embolism in a patient with type A aortic dissection. Clin Cardiol 28: 53-7.

10. Tiemtoré-Kambou BMA, Koama A, Kontogom S, Tiendrébéogo JZ, Bayala D et al. (2022). Aortic dissection-Pulmonary embolism association: A therapeutic dilemma.Radiol Case Rep 17: 2779-83.

11. Kamp TJ, Goldschmidt-Clermont PJ, Brinker JA, Resar JR (1994). Myocardial Infarction, aorticdissection and thrombolytic therapy. Am Heart J 128: 1234-7.

12. Blanke P, Apfaltrer P, Ebersberger U, Schindler A, Langer M, Schoepf UJ (2012). CT detectionof pulmonary embolism and aortic dissection. Cardiol Clin 30: 10316.

13. Mouedder F, Laachach H, Elyandouzi A, Fliti A, Toutai C et al. (2019). A pulmonaryembolism simulating acute coronary syndrome. Pan Afr Med J 33: 75.

14. Pierre HOUTIN (2021). Type B aortic dissection, descriptive and analytical study according togender [Thesis Med]. Marseille: Faculté des sciences médicales et paramédicales: AixMarseille université; 31.

15. Nienaber CA, Eagle KA (2003). Aortic dissection: new frontiers in diagnosisand management:Part I: from etiology to diagnostic strategies. Circulation 108: 628-35.

16. Badidi M, Benyass A, Lakhal Z, Chaib A, Raissouni M, et al (2009). Management of acute aorticdissection. Maroc Méd 31: 213-18.

17. Tsai TT, Nienaber CA, Eagle KA (2005). Acute aortic syndromes. Circulation 112: 3802-13.

18. AhmadF, CheshireN, HamadyM (2006). Acute aortic syndrome: pathology and therapeuticstrategies. Postgrad Med J 82: 30512.

19. DakeMD, et al. (1999). Endovascular stent-graft placement for the treatment of acute aorticdissection. N Engl J Med 340: 154652.

20. Doherty S (2017). Pulmonary embolism: an update. Aust Fam Physician 46: 816-20.

21. Morimoto S, Izumi T, Sakurai T, et al. (2007). Pulmonary embolism and deep vein thrombosiscomplicating acute aortic dissection during medical treatment. Intern Med 46: 477-80.

22. Erbel R, Alfonso F, Boileau C, et al (2001). Diagnosis and management of aortic dissection. EurHeart J 22: 1642-81.

23. BarronDJ, Livesey SA, Brown IW, et al. (1997). Twenty-year follow-up of acute type A dissection:the incidence and extent of distal aortic disease using magnetic resonance imaging. J CardSurg 12: 147-59.

24. ErbelRet al. (2014). ESC Guidelines on the diagnosis and treatment of aortic diseases:document covering acute and chronic aortic diseases of the thoracic and abdominal aorta ofthe adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of theEuropean Society of Cardiology (ESC). Eur Heart J 35: 2873926.