INTRODUCTION

The concurrence of pulmonary embolism (PE) and aortic dissection is rare and therefore creates therapeutic dilemmas [1]. Anticoagulation is the cornerstone therapy for PE and is upgraded to systemic thrombolysis in patients with hemodynamic instability [2]. However, acute aortic dissection is commonly deemed a contraindication to such therapies, and several investigators have reported aortic leakage during anticoagulation therapy in patients with aortic dissection [3], although this concern has never been proven. Conversely, recent reports showed the safety of anticoagulation in intramural hematoma and acute type B aortic dissection (TBAD) [1,4,5]. Moreover, contradictory evidence exists about the role of anticoagulation therapy on the false lumen patency [6,7].

We herein describe a patient with TBAD complicated by PE. This report will outline the safety of anticoagulation therapy in a patient with aortic dissection, as well as the role of early thoracic endovascular aortic repair (TEVAR), the possible interaction with anticoagulation therapy, and finally the effect of anticoagulation therapy on the false lumen patency.

CASE

A 60-year-old man with a history of chronic obstructive pulmonary disease was admitted to a hospital because of the sudden onset of abdominal pain. No history of previously known cardiovascular risk factors or cardiovascular diseases was reported by the patient. Initially, he was treated as an outpatient with the impression of gastric ulcer. However, his symptoms aggravated and he returned to the index hospital 12 hours after his outpatient visit owing to intractable abdominal pain, episodes of syncope, and respiratory distress necessitating intubation. A bedside
ultrasound examination revealed massive pleural effusion and abdominal aortic dissection. A computed tomography angiography (CTA) scan revealed a TBAD extending to the abdominal aorta (Fig. 1). The patient's syncope and respiratory distress were attributed to the TBAD and the concomitant pleural effusion. The TBAD was classified as impending to rupture, and the patient was referred to our center for TEVAR.

Our rapid initial evaluation via transthoracic echocardiography showed severe right ventricular (RV) enlargement along with moderate RV systolic dysfunction, mid-RV free-wall akinesia, and normal motion at the apex (McConnell sign). Left ventricular hypertrophy was also apparent, probably indicative of long-standing uncontrolled hypertension. A pulmonary CTA confirmed intraluminal filling defects in both the right upper and left lower pulmonary lobar branches in addition to the left and right upper and lower pulmonary segmental and subsegmental branches, suggestive of acute PE (Fig. 2). A bedside Doppler venous ultrasound also revealed left popliteal acute deep venous thrombosis.

Two options were proposed for the treatment of the concomitant PE: deployment of an inferior vena cava filter or emergent TEVAR alongside anticoagulation therapy. Because the radiological findings were suggestive of impending rupture, the decision was made to proceed with TEVAR (Fig. 3) [8]. Unfractionated heparin was immediately started, and vascular accesses (via both common femoral arteries) were obtained under ultrasound guidance. TEVAR was percutaneously commenced using vascular closure devices (Perclose ProGlide; Abbott Vascular Devices, Redwood City, CA, USA). The right radial access was obtained for aortic root injection and device positioning, and the left radial access was used for coil delivering. For the prevention of spinal cord ischemia, a single 36×40×217 mm Zenith Alpha Thoracic Endovascular Graft (Cook Medical, Bloomington, IN, USA) was implanted just after the left carotid artery and above the celiac artery, covering the entry site. Rapid RV pacing was applied for correct endograft deployment.

In our patient, for the prevention of future type II endoleak, 3 Nester coils (Cook Medical) were deployed via the left radial access into the insertion of the subclavian artery. After the termination of TEVAR, a pigtail catheter was inserted for the drainage of the patient’s massive hemothorax.

Given the patient’s concomitant RV dysfunction and history of chronic obstructive pulmonary disease, weaning from the ventilator was delicately performed in a stepwise manner. The control CTA was satisfactory in terms of the endovascular procedure and proper device positioning (Fig. 4). However, a small type II endoleak from the left subclavian artery was detected, for which a conservative approach was decided. After 15 days, the patient was discharged from the hospital in good condition on warfarin with an inter-

Fig. 1. Three-dimensional reconstructed computed tomography images showing a type B aortic dissection, starting just at the origin of the left subclavian artery (A) and extending down to the left common iliac artery (B). All visceral vessels are separated from the true lumen (B).
At the 6-month follow-up, a CTA scan showed reduced size of the false lumen with nearly complete thrombosis (Fig. 5). Apart from the index event, our patient did not mention any history of previous venous thromboembolism. Consequently, we stopped anticoagulation after the 6-month therapy. The RV function was preserved in the follow-up echocardiography.

**DISCUSSION**

Traditionally, conservative management has been offered as the main therapeutic strategy for TBAD, with TEVAR being reserved for complicated cases such as hemodynamic instability, malperfusion, uncontrolled hypertension, and intractable pain [9]. Nevertheless, several reports have shown that >40% of initially uncomplicated TBAD cases will be complicated during their courses and, consequently, early TEVAR may be a rational solution in high-risk patients [10]. Several indices have been suggested as predictors of late complications, the most significant of which are entry tear >10 mm, total aortic diameter >40 mm, and false lumen diameter >20 mm [8]. All these radiological indices were positively identified in our patient, prompting us to opt for a more invasive management (Fig. 3).

In addition to TBAD, our patient was complicated by another critical condition: PE. Classically, acute aortic dis-
section has been considered a contraindication to thrombolytic therapy, and anticoagulation has been judged too risky based on the notion that it increases the risk of aortic rupture or massive bleeding. Hence, the combination of acute aortic rupture and PE, albeit rare, presents a daunting challenge [1]. Our patient’s PE may have played a role in aggravating his respiratory symptoms at the initial hospitalization. Nonetheless, although he was intubated upon admission to our hospital, the fact that he had a stable blood pressure precluded prioritizing thrombolytic therapy. In any case, all available guidelines stipulate that immediate anticoagulation is of utmost importance in similar circumstances [2]. Al-Zuabi et al. [3] reported an aggravating aortic leak after the start of anticoagulation therapy in a patient with concomitant aortic dissection and PE. However, the association between anticoagulation therapy and bleeding risk has never been proven. Cañadas et al. [11] reported no increase in bleeding complications in 3 patients with acute intramural hematoma, in whom anticoagulation was indicated for PE or atrial fibrillation. Importantly, anticoagulation therapy does not seem to influence the morphological course of intramural hematoma. Apart from the safety of anticoagulation in intramural hematoma, several reports have demonstrated its safety in patients complicated by aortic dissection. Mitu et al. [1] reported no bleeding events after the initiation of anticoagulation in a patient with TBAD complicated by PE. Similar results on the safety of anticoagulation in TBAD were reported by Bocchino et al. [4], who described a patient with TBAD complicated by acute left ventricular thrombosis necessitating anticoagulation therapy. The existing literature contains no reports of bleeding events or dissection-related complications [4]. Further in this regard, von Kodolitsch et al. [5] confirmed the safety of anticoagulation in repaired acute type A aortic dissection.

Another important concern with respect to anticoagulation therapy in acute dissection is its possible influence on the false lumen patency [12]. A patent false lumen has been related to a poor long-term prognosis [12]. However, recent reports have shown no impact or beneficial effects of anticoagulation therapy on false lumen remodeling [6,7]. Song et al. [6], in their series of 136 patients with acute DeBakey type I aortic dissection, showed that the mean segmental aortic growth rate was less in the anticoagulation group than in the no-anticoagulation group (2.9±1.3 vs. 4.5±2.8 mm/y; P=0.0184), and concluded that early anticoagulation therapy may have a beneficial impact on the thrombosis extension, rate of aortic growth, and long-term survival.

It should be noted that aortic pulsation may result in device displacement during deployment and the induction of hypotension may not only be ineffective but may also have deleterious effects on the patient’s condition. However, rapid RV pacing may prove to be effective with the fewest complications [13]. Careful positioning of the temporary RV pacing lead, especially in patients with RV dysfunction, is mandatory. Moreover, the RV function should be protected against further compromise by ensuring that the pacing is brief and the RV pacing lead is immediately removed after device positioning [13].

One of the therapeutic options that we considered for our patient was the implantation of an inferior vena cava filter. In the absence of any conclusive evidence in the literature about a superior strategy, we ultimately opted for TEVAR considering that it was indicated by the presence of the radiological predictors of late complications, with a view to preventing possible bleeding events such as rupture and leakage.

As mentioned above, our patient had all the radiological predictors indicating a late TBAD complication. Nonetheless, in patients without these predictors, the safe option is again anticoagulation therapy because it is associated with a lower risk of aortic rupture/leakage. To our knowledge, all of the above-mentioned studies on the safety of anticoagulation therapy in TBAD were conducted in patients who were medically managed, which again underscores the safety of the mentioned strategy [2,4].

In conclusion, a patient with concomitant TBAD and PE was successfully treated with early TEVAR and subsequent anticoagulation. Anticoagulation in acute aortic dissection was well tolerated in this case, although TEVAR contributed to a decrease in the false lumen pressure. Further investigations on the safety of anticoagulation therapy in patients with acute dissection are required to find more evidence-based solutions.

**CONFLICTS OF INTEREST**

The authors have nothing to disclose.

**ORCID**

Amirkhosro Gouran  
https://orcid.org/0000-0002-2715-7708

Hassan Safdari  
https://orcid.org/0000-0002-8209-0091

Hamidreza Pouraliakbar  
https://orcid.org/0000-0002-2557-4030

Omid Shafe  
https://orcid.org/0000-0001-7927-4562

Jamal Moosavi  
https://orcid.org/0000-0001-5964-1724

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AUTHOR CONTRIBUTIONS

Concept and design: OS, PS. Data collection: AG, HP, HS, JM, BM. Writing the article: AG, PS. Critical revision of the article: JM, BM. Final approval of the article: OS, PS.

REFERENCES

1) Mitu O, Miftode R, Pintilie A, Petris AO. Rare association of acute pulmonary embolism and type B aortic dissection: anticoagulant therapeutic dilemma. Romanian J Cardiol 2019;29:47-50.
2) Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjula VP, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS): the task force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). Eur Heart J 2020;41:543-603.
3) Al-Zuabi S, Varkey AP, Abdalmaksoud SF, Alothman HS, Alrashdan I. A rare case of leaking thoracic aortic aneurysm in a patient with massive pulmonary embolism. Egypt J Intern Med 2013;25:47-50.
4) Bocchino PP, De Filippo O, Piroli F, Scacciatella P, Imaizo M, D’Ascenzo F, et al. Anticoagulant and anti-thrombotic therapy in acute type B aortic dissection: when real-life scenarios face the shadows of the evidence-based medicine. BMC Cardiovasc Disord 2020;20:29.
5) von Kodolitsch Y, Wilson O, Schüler H, Larena-Avellaneda A, Köbel T, Wipper S, et al. Warfarin anticoagulation in acute type A aortic dissection survivors (WATAS). Cardiovasc Diagn Ther 2017;7:559-571.
6) Song SW, Yoo KJ, Kim DK, Cho BK, Yi G, Chang BC. Effects of early anticoagulation on the degree of thrombosis after repair of acute DeBakey type I aortic dissection. Ann Thorac Surg 2011;92:1367-1374; discussion 1374-1375.
7) Öztürk P, Apaydin AZ, Karakuş E, Kılıç AÜ, Özbaran M. The impact of oral anticoagulation on false lumen patency in acute type A aortic dissections. Turk Gogus Kalp Damar Cerrahisi Derg 2018;26:345-350.
8) Schwartz SI, Durham C, Clouse WD, Patel VI, Lancaster RT, Cambria RP, et al. Predictors of late aortic intervention in patients with medically treated type B aortic dissection. J Vasc Surg 2018;67:78-84.
9) Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE Jr, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SIR/STS/SVM Guidelines for the diagnosis and management of patients with thoracic aortic disease. A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. J Am Coll Cardiol 2010;55:e27-e129.
10) Reutersberg B, Trenner M, Haller B, Geibbüsch S, Reeps C, Eckstein HH. The incidence of delayed complications in acute type B aortic dissections is underestimated. J Vasc Surg 2018;68:356-363.
11) Cañadas MV, Vilacosta I, Ferreirós J, Bustos A, Díaz-Mediavilla J, Rodríguez E. Intramural aortic hematoma and anticoagulation. Rev Esp Cardiol 2007;60:201-204. Spanish.
12) Evangelista A, Salas A, Ribera A, Ferreira-González I, Cuellar H, Pineda V, et al. Long-term outcome of aortic dissection with patent false lumen: predictive role of entry tear size and location. Circulation 2012;125:3133-3141.
13) Nienaber CA, Kische S, Rehders TC, Schneider H, Chatterjee T, Bünger CM, et al. Rapid pacing for better placing: comparison of techniques for precise deployment of endografts in the thoracic aorta. J Endovasc Ther 2007;14:506-512.