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HYDATID PULMONARY EMBOLISM A RARE CAUSE OF NON-THROMBOTIC OF PULMONARY EMBOLISM: REPORT OF 3 CASES AND REVIEW OF PUBLISHED REPORTS

María José Cristo Ropero[1]*, Carolina España Dominguez[2], Sergio Alonso Charterina[3], Teresa Segura De La Cal[1], Fernando Arribas[4], María Jesús López Gude[5], Pilar Escribano Subías[1]

[1]Pulmonary Hypertension Section, Cardiology Department of Hospital Universitario 12 de Octubre. Madrid, Spain.
[2]Pneumology Department of Hospital Universitario Puerta del Mar. Cádiz, Spain.
[3]Radiology Department of Hospital Universitario 12 de Octubre. Madrid, Spain.
[4]Cardiology Department of Hospital Universitario 12 de Octubre. Madrid, Spain.
[5]Cardiovascular Surgery Department of Hospital Universitario 12 de Octubre. Madrid, Spain.

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***Corresponding author: María José Cristo Ropero**

Pulmonary Hypertension Section, Cardiology Department of Hospital Universitario 12 de Octubre. Madrid, Spain.

ABSTRACT

Hydatid pulmonary embolism a rare cause of non-thrombotic of pulmonary embolism. Report of 3 Cases and Review of Published Reports **Objectives:** Cystic echinocccosis is a rare disease that can still be found in some rural areas. Pulmonary embolism due to hydatid cysts, although rare could be its clinical presentation. **Methods:** We report three clinical cases of hydatid pulmonary embolism (HPE), each one illustrating a particular presentation of this condition. We have carried out an exhaustive review of the literature, comparing our findings with previous ones. **Results:** HPE is an uncommon condition and only a few case-reports can be found in the literature. We report three clinical cases each one illustrating a particular presentation of this condition. The first one constitutes a chronic active form, the second represents a chronic stable isolated pulmonary affection, while the third case describes the clinical course of an acute HPE. This report is also the first to describe an exceptional case of chronic stable hydatidosis with isolated involvement of the pulmonary artery. HPE clinical presentation can be very varied because it is related to the size and location of the cysts. Surgery is the treatment of choice, but it is not always feasible. Hence, the diagnostic and therapeutic approaches as well as prognosis vary depending on HPE presentation. **Conclusion:** Hydatid pulmonary embolisms are rare but should be taken into account in the differential diagnosis of non-thrombotic PE. Diagnostic and therapeutic approaches need to take into account the possible clinical presentations and importantly, the feasibility of a surgical approach, which is to date the only definitive treatment.

KEYWORDS: Hydatid disease; non-thrombotic of pulmonary embolism; surgery.

INTRODUCTION

Hydatid disease is caused by the infection with the larval stage of Echinococcus granulosuss, and it can still be found in some Spanish rural areas. Domestic animals like dogs are the primary carriers and humans can be intermediary carriers.[1] Symptoms depend on the location, size, and integrity of the cyst.[2–4] Although pulmonary embolism due to hydatid cysts (HC) is an infrequent clinical entity, it should be taken into account in the differential diagnosis of non-thrombotic causes of pulmonary embolism.

We report three clinical cases of hydatid pulmonary embolism (HPE), each one illustrating a particular presentation of this condition. The first one constitutes a chronic active form, the second represents a chronic stable isolated pulmonary affection, while the third case describes the clinical course of an acute HPE.

METHODS

Patients were included after referral by their physicians to our centre. All patients were evaluated by routine medical history, clinical examination, and different imaging techniques such as transthoracic echocardiography (TTE), high-resolution computed tomography (CT), ventilation/perfusion lung scan, abdominal ultrasound, or cardiac magnetic resonance imaging (MRI). Pulmonary hypertension (PH) diagnosis

was established by right heart catheterization according to the last Nice PH definition (2018 group). If catheterization was not available, we considered PH diagnosed when the TTE probability was high according to the European guidelines.[5]

RESULTS

The first patient is a 53-year-old female and constitutes a chronic active clinical presentation. She was diagnosed with hydatidosis at the age of 26, after admission for constitutional syndrome and fever. At that time, serology for *Echinococcus* was positive, but no HC were detected. At age of 32, immediately after pregnancy, she suffered an acute PE. At that point, CT scan showed multiple bilateral pulmonary nodular lesions and filling defects in pulmonary arteries compatible with hydatid embolisms *(figure 1)*. Cystectomy was not considered technically feasible and lung transplant was dismissed due to the active infectious process. She was started on treatment with albendazole and praziquantel. During follow-up, the patient presented recurrent episodes of pleuritic pain, fever, haemoptysis and expectoration of cyst fluid, membranes, and scolices and her clinical course further worsened with the development of chronic PH. She was referred to our PH unit in 2019. New available examinations such as cardiac MRI, permitted us to identify the presence of a HC in the apical region of the interventricular septum that fortunately did not affect cardiac rhythm or function *(figure 1)*. Therapeutic options were extensively discussed. Myocardial cystectomy was ruled out due to surgical complexity and pulmonary endarterectomy was also discouraged taking into account the distal embolic involvement on top of the diffuse parenchymal affection. She was hence started on medical PH-specific treatment (bosentan 125mg twice daily and riociguat 2.5mg three times a day) with subsequent improvement in her functional class. No anticoagulation was prescribed because of the recurrent haemoptysis episodes.

The second clinical case is a 54-year-old male patient and represents a chronic stable clinical presentation. He was incidental diagnosed with chronic pulmonary thromboembolic disease without PH during an admission for pneumonia in 2016 *(figure 2)*. The patient was asymptomatic with normal functional class over follow-up. A CT scan requested two years later exhibited growth of the intraluminal lesion. A positron emission tomography scan was then performed, showing no signs of malignancy. During the diagnostic work-up process, the patient reported a family history of hepatic and pulmonary HC. Hence, the requested equinococus serology confirmed the diagnosis and treatment with albendazole and praziquantel was started. Abdominal ultrasound and cardiac MRI excluded the involvement of other organs. On the contrary, pulmonary MRI showed several daughter cysts into the left main pulmonary artery's lumen, extending to the left upper and lower lobar arteries *(figure 2)*. After a thorough multidisciplinary discussion, pulmonary endarterectomy and cyst excision were indicated in order to prevent further spread of the cystic material, to minimize the risk of cyst fracture and even to remove the entire additional thrombotic component. Six months after surgery, a CT showed complete resolution of the HC membranes and thrombotic lesions in the left main pulmonary artery; however, the defects in the left upper and lower subsegmental arteries persisted *(figure 3)*. Importantly, no new embolized cysts were found in other organs. These residual distal lesions were not suitable for surgical access, so a conservative attitude was adopted.

Finally, the third case describes the clinical course of an acute HPE in a 40-year-old female patient. She admitted to the emergency room due to generalized urticaria, dyspnoea, and chest tightness. Bedside-TTE showed signs of severe PH. Chest-CT scan demonstrated multiple bilateral filling defects suggesting acute PE. An abdominal CT scan was performed which clearly revealed various hepatic HC, the biggest one showing a broken wall draining directly to the inferior vena cava *(figure 4)*. Treatment with albendazole and praziquantel was started. The multidisciplinary team meeting agreed to proceed with a two-step surgical approach. Partial hepatectomy and extraction of the free cysts in the inferior vena cava would be the first step. The second step would consist on a thromboendarterectomy aiming to remove the intravascular cysts from the pulmonary arteries. Without warning, while waiting for intervention, the patient presented an anaphylactic shock. She received treatment with adrenaline perfusion and non-invasive mechanical ventilation. In addition, TTE showed severe left ventricular dysfunction and dynamic electrocardiogram changes highly suggestive of hypersensitivity coronary spasm (Kounis syndrome). Finally, the patient died due to mixed cardiogenic and anaphylactic shock.

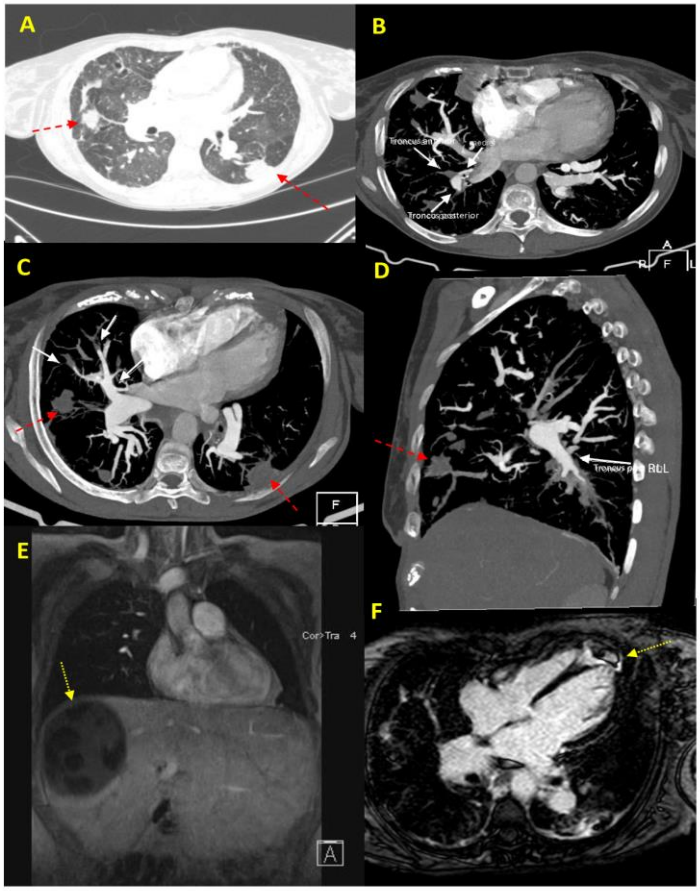

Figure 1: Red arrows show the hydatid cysts in the pulmonary parenchyma (1A, C, D) on computed tomography. White arrows show the filling defects in pulmonary arteries compatible with hydatid embolisms (1B, C, D). Yellow arrows show a big hepatic hydatid cyst (1E) and a hydatid cyst in the apical region of the interventricular septum (1F) on MRI.

Figure 1: Finding on CT and MRI in clinical case number 1.

Red arrows show the hydatid cysts in the pulmonary parenchyma (1A, C, D) on computed tomography. White arrows show the filling defects in pulmonary arteries compatible with hydatid embolisms (1B, C, D). Yellow arrows show a big hepatic hydatid cyst (1E) and a hydatid cyst in the apical region of the interventricular septum (1F) on MRI.

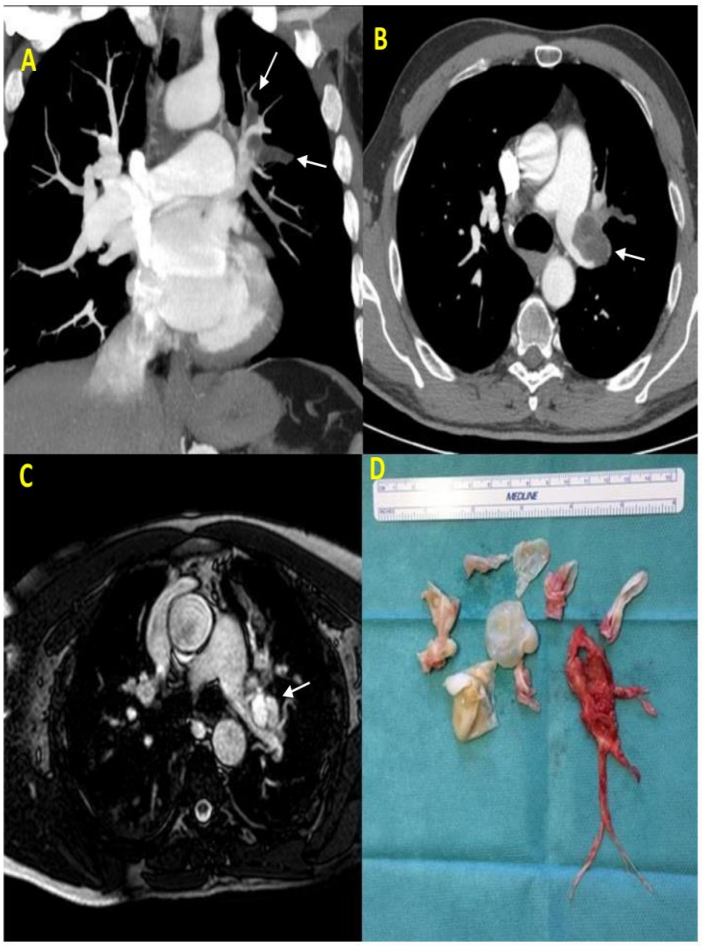

Figure 2: CT shows an intravascular filling defect in the main left pulmonary artery (2B) and in two left upper segmental arteries (2A). Pulmonary MRI confirms the diagnosis of HPE showing several daughter cysts into the left main pulmonary artery's lumen (2C). Intraoperative photograph shows all extracted hydatid cysts and the thrombotic component that surrounded them (2D)

Figure 2: Finding on CT and MRI in clinical case number 2 and correlation of intraoperative findings.

CT shows an intravascular filling defect in the main left pulmonary artery (2B) and in two left upper segmental arteries (2A). Pulmonary MRI confirms the diagnosis of HPE showing several daughter cysts into the left main pulmonary artery's lumen (2C). Intraoperative photograph shows all extracted hydatid cysts and the thrombotic component that surrounded them (2D)

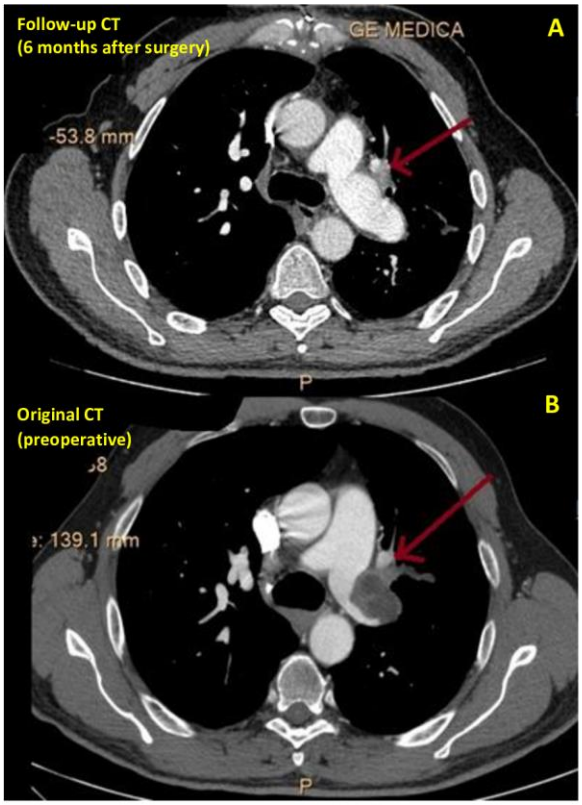

Figure 3: Six months after surgery, a CT showed complete resolution of the hydatid cyst membranes and thrombotic lesions in the left main pulmonary artery. In 3A, the arrow shows the intravascular filling defect in left upper segmental artery.

Figure 3: Finding on CT after surgery.

Six months after surgery, a CT showed complete resolution of the hydatid cyst membranes and thrombotic lesions in the left main pulmonary artery. In 3A, the arrow shows the intravascular filling defect in left upper segmental artery.

Figure 4: Finding on CT in clinical case number 3

Chest-CT scan shows multiple bilateral filling defects suggesting acute PE (4A, B, C). The picture 4A shows an upper abdominal cross sectional which reveal a big hepatic hydatid cyst (discontinuous red arrows). The pictures 4D and E show a cyst which present a broken wall draining directly to the inferior vena cava (discontinuous red arrows)

Table 1:	**Case 1**	**Case 2**	**Case 3**
Sex (age)	Female (53)	Male (54)	Female (40)
Contact with animals	No	Yes	No
Previously diagnosed with hydatidosis	Yes	No	No
Clinical presentation			
Acute pulmonary embolism	Yes (after pregnancy)	Yes	Yes
Anaphylactic reaction	No	No	Yes
Hepatic cyst	Yes (one)	No	Yes (multiple, one broken draining into the IVC)
Pulmonary cysts	Yes (multiple)	No	Yes (multiple)
Pulmonary hypertension	Yes	No	Yes
mPAP	*Estimated sPAP based on TRV*	*23 mmHg*	*43 mmHg*
CI	*was 65 mmHg*	*3,1 l/min/m2*	*3.3 l/min/m2*

PVR		*2.1 Wood units*	*6,5 Wood units*
Other affected organs	Yes (heart)	No	Yes (peritoneal cavity)
Treatment			
Surgery	No (not feasible)	Yes	No (early death)
Vasodilator therapy	Yes	No	No
mPAP: mean pulmonary artery pressure. CI: Cardiac index. PVR: pulmonary vascular resistance. IVC: inferior vena cava. sPAP: systolic pulmonary artery pressure. TRV: tricuspid regurgitation velocity			

Table 2: Clinical cases of pulmonary arterial hydatid cysts.

Article. Year. Geography	N	Age. (Sex) History for HC	Clinical presentation	Pulmonary hypertension	Other organs affected	Surgical treatment of PE	Follow-up
Özer. 2001(10) Turkey	5	61-year-old (female). No	Chronic (1 year)	Yes	Right atrium	No	Discharged alive
		24-year-old (female). Yes (liver surgery)	Acute pulmonary embolism	Yes	Right ventricular apex	Yes	
		17-year-old (male). No	Acute pulmonary embolism	Yes	Both lungs. Interventricular septum.	Yes.	Discharged alive. Persistent PH after 2 years
		For the other 3 patients, cysts presented with peripheral arterial embolization.					
Odev.2002(22) Turkey	2	14 year-old (male). No	Chronic (3 acute pulmonary embolism)	Yes (after 3rd episode)	Lung and right ventricle	Yes. Embolectomy could not be performed in the 3rd surgery	Discharged alive
		39 year-old (female). No	Acute pulmonary embolism	Unknow	Lung, right atrium and superior vena cava.	Yes (+ cardiac). Embolectomy could not be perfomed	Discharged alive
Koksal. 2006(23) Turkey	1	24-year-old (male) Yes (liver)	Acute pulmonary embolism	Unknow	Lung.	Yes (in two stages. + pneumonectomy)	No relapse after 14 months
Gecmen 2011(24) Turkey	1	24-year-old (male) Yes (lung surgery)	Acute pulmonary embolism	Yes	Lung and right ventricle	Yes	Discharged alive
Akgun 2011(4) Turkey	1	43-year-old (male) No.	Subacute (3 weeks)	Yes	Lung, right atrium, liver IVC	No (patient refused liver surgery). Embolectomy was not performed due to high risk of anaphylaxis)	Alive at 2 months of follow-up. Continued on medical therapy
Poyraz. 2016(12) Turkey	1	45-year-old (male) Yes (liver)	Acute pulmonary embolism	Unknow	Lung. Liver. IVC	No (patient refused)	Discharged alive
Savaş 2017(11) Turkey	1	48-year-old (female) Yes	Subacute (1 month)	Yes	No	Yes	Death (on the second day after surgery

		(heart)					or two days after surgery)
Şahpaz 2017(25) Turkey	1	15-year-old (male) No.	Sudden death	Unknown	Liver. Lung.	No	Death
Orhana.2018(26) Turkey	1	26-year-old (male). Yes (lung and liver surgery)	Acute pulmonary embolism	Unknow	Lung and right ventricle	Yes (+ cardiac)	Discharged alive
HC: hydatid cyst. PH: pulmonary hypertension. IVC: inferior vena cava							

DISCUSSION

HPE is an uncommon condition and only a few case-reports can be found in the literature *(table 2)*. This case-series is of particular interest as it includes three possible clinical forms of HPE presentation. Moreover, to our knowledge, this report is the first to describe an exceptional case of chronic stable hydatidosis with isolated involvement of the pulmonary artery (nº 2). It also highlights how diagnostic and therapeutic approaches as well as prognosis vary depending on HPE presentation *(table 1)*

Baseline Characteristics

Hydatid disease or echinococcosis still represents a significant health problem, especially in certain areas in South America, the Mediterranean countries, east Africa, and Australia (6). The disease is most frequently encountered in regions with extensive rural areas and close contact between humans and animals. In a Turkish study, Arinc et al.[7] reported that 54,1% of pulmonary HC cases came from these rural areas. (8) However, HPE was not reported in the largest Turkish study.[8] Thus, HPE are generally published as independent case reports *(table 2)*.

According to the literature,[9] hydatid disease is not common in Spain. However, we do occasionally face hydatidosis and HPE cases in our clinical practice and we need to bear it in mind in the differential diagnosis of non-thrombotic pulmonary embolisms.

Epidemiological factors are important in order to raise suspicion; however, they cannot always be found. For instance, in this clinical series, only patient 2 had a definitive epidemiological background.

Clinical presentation and diagnostic process

The clinical presentation of hydatidosis depends on the location, size, and integrity of the cyst.[10] To better understand it, it is important to recall the course of *E. granulosus* infection. The human ingests the food contaminated by the eggs. Once in the gastrointestinal system, the larva penetrates the intestinal mucosa reaching the bloodstream, which drives them into the liver. Hence, the liver is the most frequently affected organ, followed by the lungs. Extrahepatic and extrapulmonary localization are rarely seen but have been reported in 2,1% of patients with hydatidosis.[7] In the same way, the clinical presentation of hydatidosis may be conditioned by cyst's compression of the surrounding structures in large cysts. Nevertheless, patients may remain asymptomatic for years and give first symptoms once a cyst rupture occurs.

Therefore, HPE is a rare hydatidosis presentation and it is usually secondary to a hydatid cyst rupture within the right cardiac chambers.[10] Finally, according to these different presentations, it has been classified from a clinical perspective as acute fatal, subacute with pulmonary hypertension, or resulting in chronic pulmonary hypertension.[10]

Table 2 shows HPE characteristics in all clinical cases published in the literature. All of them exhibited involvement of other organs, except from Savaş 2017,[11] who had medical history of heart cystectomy. Noteworthy, no isolated pulmonary artery involvement has been previously described.

Our reported cases also highlight the role of MRI in the hydatidosis diagnosis work-up.[3,12] In case 2, suspicion was raised due to intravascular mass growth within the following 2 years. A positron emission tomography scan was considered necessary to rule out a malignant aetiology,[13] but once excluded, diagnosis was given by MRI, which clearly showed several daughters cysts in the left pulmonary artery lumen. Moreover, MRI was also the key for the diagnosis of a cardiac HC in case number 1, which could not be identified in a CT scan years before. Moreover, in this particular case, it was not possible to detect HC at the time of diagnosis decades ago. We strongly presume that it would have been possible using current imaging techniques like MRI.

Patient 1 also reveals the delicate issue of how to manage hydatid disease during pregnancy. Information on this topic is scarce. It is known that hepatic cysts show an increased risk of rupture throughout gestation due to uterine growth and specially to labour effort. Our patient suffered an acute PE after pregnancy. The spread of the disease could have been related to labour and to the decreased cell-mediated immunity during pregnancy(14–16). Regarding the possibility of a vertical transmission

of the disease, it has been only demonstrated in animals, while data in humans are contradictory,[17] Our patient delivered a healthy baby-girl.

Finally, case 3 constitutes another uncommon clinical presentation with an acute fatal debut aggravated by secondary Kounis syndrome, a rare form of anaphylaxis with hypersensitivity coronary spasm. Although, the most common triggers described for Kounis syndrome are antibiotics (27,4%), and insects' bites (23.4%)(18), Kounis syndrome occurrence secondary to HC rupture, has also been previously described.[19]

Treatment

Patients with HPE are at high risk of anaphylaxis and massive distal embolisms, as illustrated in case 3. Thromboendarterectomy under circulatory arrest is the only definitive treatment.[20,21] However, it carries the risk of cyst rupture and subsequent acute dissemination, and it is not always technically feasible. Several factors need to be taken into account when planning this intervention in HPE patients. Apart from the location of the HC and the thrombotic material, it is essential to evaluate the feasibility of a pneumonectomy prior to surgery as a rescue option if the patient suffers distal embolisms.[4,10–12,22–26]

Among all previously published HPE patients in the last 20 years, (table 2), 83% were suitable for surgical removement (10 patients). Among these 10 patients, 2 declined. From the remaining 8 patients, thromboendarterectomy was scheduled as a second step after cystectomy on other sites in two cases but it had to be abandoned due to extreme surgical risk. Hence, thromboendarterectomy was finally performed in 6 cases, which represents 50% of the HPE published cases. Only two patients had a long-term follow-up.[10,23] In the acute setting, 10 out of the 12 patients were discharged alive. One patient died after surgery and another presented with sudden death.[11,25] No cyst relapse was observed in two of the surgically treated patients, although one of them continued with pulmonary hypertension.[10]

In our report, thromboendarterectomy was considered feasible in patients 2 and 3. In patient 2, it was indicated in spite of normal pulmonary pressures with the aim to prevent cyst rupture. The cardiovascular surgeon had special care to avoid distal cysts embolisms. To that end, they positioned gauzes impregnated in hypertonic serum covering the surgical bed before and after removing the cysts. Surgery was sucessfully performed and the patient enjoyed an uneventful recovery. In the case of patient 3, thromboendarterectomy was scheduled as the second step after abdominal surgery to first remove the biggest hydatic cyst. Unfortunately, the patient presented an anaphylactic shock, probably due to new release of cysts from the inferior vena cava, and died. Regarding patient 1, no surgical approach was planned at diagnosis as no HC were then detected. Presumably, had they been demonstrated during pregnancy, surgery would have been the treatment of choice to minimize the risk of dissemination.

HPE refers to both the hydatids and the surrounding thrombotic material. We advocate for anticoagulant treatment in patients with low bleeding risk to reduce this additional thrombotic load (case 2), but more data to support this approach is needed.. In the particular case of patient 1, anticoagulation was initially started but later withdrawn due to the recurrent haemoptysis episodes.

CONCLUSIONS

HPE are rare, but they should be taken into account in the differential diagnosis of non-thrombotic PE. Clinical presentation is related to the size and location of the cysts. These patients are at a high risk of both anaphylaxis and acute massive embolism.

Surgery is the treatment of choice, but it is not always feasible and should be carefully planned. MRI can be useful to identify daughter cysts when present.

Conflicts of interest

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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Authors' contributions.

All authors contributed equally to the manuscript and read and approved the final version of the manuscript.

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