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Case report

Successful balloon pulmonary angioplasty with gadolinium contrast media for a patient with chronic thromboembolic pulmonary hypertension and iodine allergy

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ABSTRACT

A 28-year-old male was referred to our hospital with dyspnea. He was diagnosed as having chronic thromboembolic pulmonary hypertension, and a pulmonary endarterectomy (PEA) was performed. However, exertional dyspnea remained because of residual pulmonary hypertension; therefore, the patient was re-admitted to our hospital 1 year after PEA. We performed computed tomography and pulmonary angiography and found web and band lesions in the distal pulmonary artery with a high pulmonary artery pressure. Although further management was complicated because the patient had an anaphylactic shock to iodine-based contrast media, we eventually completed five sessions of balloon pulmonary angioplasty (BPA) using gadolinium contrast medium. His symptoms and hemodynamics dramatically improved after a series of BPA. After 15 months, mean pulmonary arterial pressure reduced from 67 mmHg to 20 mmHg, and subjective symptoms improved from stage *IV to* I as per the WHO classification system. BPA is a potential procedure for residual pulmonary hypertension after PEA and could be safely performed using gadolinium contrast medium for patients with iodine allergy.

1. Background

The effectiveness of balloon pulmonary angioplasty (BPA) for chronic thromboembolic pulmonary hypertension (CTEPH) is known [1,2]; however, the management of anaphylaxis to contrast media is rarely reported in this context. Here we report a 28-year-old man with no history of iodine allergy who presented with refractory CTEPH and developed anaphylaxis to iodine contrast media during BPA. We describe the case history and use of gadolinium contrast media for BPA.

2. Case presentation

A 28-year-old male had never been diagnosed with an iodine allergy. At 27 years of age, the patient underwent pulmonary endarterectomy (PEA) for central CTEPH caused by anti-phospholipid antibody syndrome. At that time, his mean pulmonary arterial pressure (mPAP) was reduced from 56 mmHg to 20 mmHg, and his subjective symptoms improved from stage III to I as per the World Health Organization (WHO) classification system. Warfarin anticoagulation was initiated before performing PEA, and good control was achieved with a target of a PT-INR of approximately 2.0–2.5. However, 1 year later, the patient's condition worsened because of an acute embolism. At this time, he was classified as WHO stage II and was subsequently hospitalized for additional treatment. Blood analysis indicated elevated lupus anticoagulant and D-dimer levels, along with findings indicative of right ventricular failure, including elevated B-type natriuretic peptide, uric acid, GOT, and GPT levels (Table 1). Chest X-ray revealed an enlarged pulmonary artery shadow with a cardiothoracic ratio of 55% and protrusion of the left second and fourth aortic arches (Fig. 1). Electrocardiogram revealed right axis deviation; a pulmonary P wave in leads II, III, and aVF; and a negative T wave in leads III, aVF, and V1–4 (Fig. 2). Echocardiogram revealed

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Table 1
Blood sample analysis.

WBC (μl)	7600	GOT (IU/l)	40	BUN (mg/dl)	15
RBC (μl)	510×10^4	GPT (IU/l)	75	PT-INR	2.05
Hb (g/dl)	17	LDH (IU/l)	308	D-dimer (μg/ml)	3.4
Plt (μl)	24×10^4	T-bil (mg/dl)	1.9	FDP (μg/ml)	13
Na (mEq/l)	143	UA (mg/dl)	7.6	BNP (pg/ml)	100
K (mEq/l)	3.9	Cre (mg/dl)	1.01	Lupus AC	1.39

WBC: White Blood cell, RBC: Red Blood Cell, Hb: Hemoglobin, Plt: Platelet, Na: Natrium, K: Kalium, GOT: Glutamic Oxaloacetic Transaminase, GPT: Glutamic Pyruvic Transaminase, LDH: Lactate Dehydrogenase, T-bil: Total Bilirubin, UA: Uric Acid, Cre: Creatinine, BUN: Blood Urea Nitrogen, PT-INR: Prothrombin Time International Normalized Ratio, FDP: Fibrin Degradation Products, BNP: Brain Natriuretic Peptide, LA: Lupus Anticoagulant.

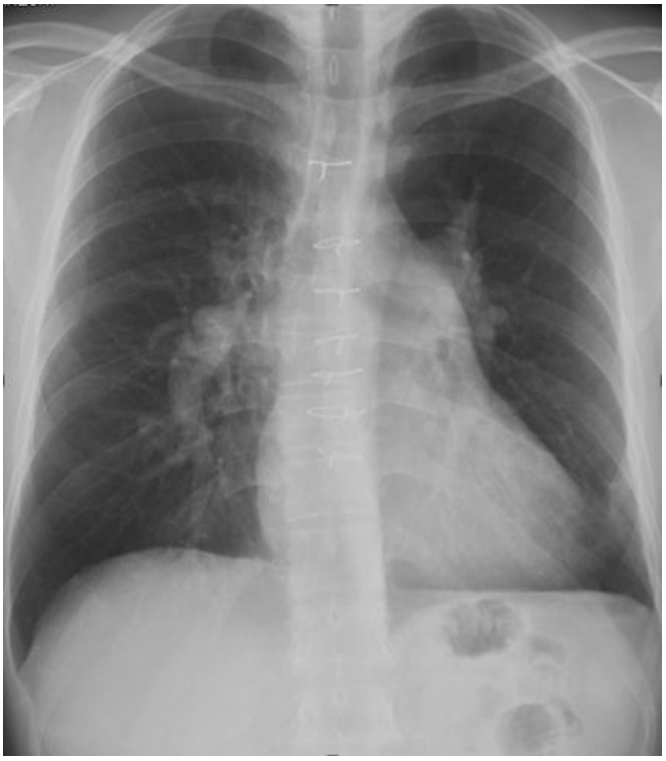

Fig. 1. Chest X-ray Film. Chest X-ray images showing cardiac dilatation with a cardiothoracic ratio of 55%, enlarged pulmonary shadow and enlargement of the left second and fourth aortic arches.

enlargement of the right atrium and right ventricle with left-ventricular displacement (Fig. 3). Marked elevation of 95 mmHg was also noted in the tricuspid regurgitation pressure gradient (TRPG). We confirmed an exacerbation of pulmonary hypertension by finding mPAP of 57 mmHg and a pulmonary vascular resistance of 12.3 Wood units on cardiac catheterization. Pulmonary capillary wedge pressure was 6 mmHg, right atrial pressure was 2 mmHg, and the cardiac index was 2.4 L/min/m^2 (Table 2). Pulmonary arteriography revealed webbing, bands, abrupt narrowing, and disruption (Fig. 4), and pulmonary ventilation/perfusion scintigraphy revealed a mismatch (Fig. 5). The findings were consistent with CTEPH.

We suspected that a new pulmonary thromboembolism had developed and, although we performed thrombolytic therapy with urokinase, the effects were limited. His PT-INR was controlled at 2.5–3.0with warfarin and aspirin administration. However, one month later, his mPAP reached 67 mmHg, and he developed hemoptysis. His condition worsened to WHO stage IV, and he lapsed into a catecholamine-dependent state. Sildenafil (60 mg), 5 mg ambrisentan, and 180 μg beraprost were used as specific therapies to treat pulmonary hypertension, but they had little effect. When iodine contrast medium was used in an investigation after hospitalization, the patient had developed a rash. Therefore, 30 mg prednisolone, 20 mg famotidine, and 2 mg D-chlorpheniramine maleate were administered as premedications before contrast-enhanced computed tomography with iopamidol (Iopamiron 370, Bayer, Land Nordrhein-Westfalen, Germany). Despite our efforts, the patient suffered an anaphylactic shock after the test. The CT images we obtained suggested progression of the pulmonary arterial lesion. However, the anaphylaxis meant that it was difficult to perform tests and treatments that relied on iodine-based contrast agents. Moreover, we had to consider the fact that within a very short period, the patient's condition had drastically worsened and he was in a life-threatening state.

At this point, we examined a report describing percutaneous transluminal coronary angioplasty using gadolinium and digital subtraction angiography for a patient with unstable angina who was allergic to iodine and another report that described the use of percutaneous transluminal angioplasty for a pediatric patient with pulmonary stenosis [3,4]. It was determined that there was a high risk that repeat surgery would worsen the patient's general condition; therefore, we decided to perform BPA with digital subtraction angiography using meglumine gadopentetate (Magnevist; Schering, Berlin, Germany).

The procedure involved placing a sheath (Arrow-Flex; Teleflex, Durham, NC) in the vein and using a 0.035-inch guidewire (Radifocus Guide Wire M; Terumo, Tokyo, Japan) to insert a 6-french-gauge (Fr) wedge-pressure catheter to the bifurcation of the pulmonary trunk. Then, this was replaced with a 6-Fr long sheath (Bright Tip sheath introducer; Cordis/Johnson & Johnson, New Brunswick, NJ) before placing the catheter in the target pulmonary artery. A 6-Fr multipurpose catheter (Mach 1 peripheral MP; Boston Scientific, Natick, MA) was inserted through this and placed in the pulmonary artery trunk. A 0.014-inch wire (Cruise; Asahi Intecc, Tokyo, Japan) was then used to cross the lesion site and was inflated to 6–12 atm using a balloon catheter (2–4 mm, IKAZUCHI PAD, Kaneka, Osaka, Japan). Digital subtraction angiography images were acquired at a rate of 7.5 frames/s with settings of 78 Kv and 41 mAs.

The vessel branches that were dilated in each session were as follows: in session 1, the posterior basal segmental artery of the right lung (Rt.A10a, 10c); in session 2, the lateral basal segmental artery of the left lung (Lt.A9b) and the posterior basal segmental artery of the right lung (Lt.A10b); in session 3, the lateral basal segmental artery of the right lung (Rt.A9b); in session 4, the anterior basal segmental artery of the right lung (Rt.A8); and in session 5, the anterior basal segmental artery of the left lung (Lt.A8). Fig. 6 shows contrast-enhanced images from before and after additional treatment on the right lower lobe branch. The five sessions were concluded without any allergic reactions or complications.

The follow-up right heart catheterization at 4 months indicated improvement to mild pulmonary hypertension with an mPAP of 28 mmHg and a pulmonary vascular resistance of 2.5 Wood units. The pulmonary capillary wedge pressure was 4 mmHg, right atrial pressure 1 mmHg, and cardiac index 3.9 L/min/m^2, indicating increased cardiac output. Improved right cardiac function was shown by the right ventricular ejection fraction increasing from 11%