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Case Report

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Pulmonary veno-occlusive disease in a patient with history of major depressive disorder

Abstract

Introduction: Pulmonary veno-occlusive disease (PVOD) represents a specific subgroup of the group 1 of the clinical classification of pulmonary hypertension.

Case presentation: We introduce a young male patient with history of major depressive disorder who was on psychiatric medications and presented with gradually progressive dyspnea and syncope. The preliminary diagnostic tools showed the presence of pulmonary hypertension; further imaging studies and right heart catheterization showed findings consistent with PVOD.

Conclusion: To best of our knowledge, this is the first report of PVOD in a patient taking psychiatric medications.

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Introduction

Pulmonary venoocclusive disease (PVOD) represents a specific subgroup of the group 1 of the clinical classification of pulmonary hypertension. PVOD is an uncommon cause of pulmonary hypertension that characteristically leads to intimal fibrosis of post capillary pulmonary vasculature due to vascular thrombosis. Several synonyms have been used by previous reports to describe this condition, including "isolated pulmonary venous sclerosis," "obstructive disease of the pulmonary veins," and "venous form of primary pulmonary hypertension".[1–3] The etiology of PVOD is largely unknown. However, genetic factors, infections, toxic exposures, thrombotic diathesis and autoimmune disorders have been presumed as probable causes. Herein, we report a case of pulmonary veno-occlusive disease in a young man with history of taking psychiatric medications which have not had exposure to any of the other known causes of PVOD.

Case presentation

A 27-year-old man was admitted to our department for the evaluation of dyspnea and syncope. The patient had begun to experience dyspnea on exertion function class II from one year ago which then progressed to function class III in the following 3 months ago. During this time, he had several syncopal episodes. Past medical history was significant for major depressive disorder and the patient was on olanzapine, lithium, clonazepam, sertraline since several years ago. On admission, vital signs included a body temperature of 36ºC, respiratory rate of 28/min, pulse rate of 98 beats per minute and a blood pressure of 95/60 mmHg. The arterial O2 saturation was 85% on room air. Jugular venous pulsation was elevated to the angle of mandible. Carotid artery pulses were brisk and equal bilaterally. No carotid, aortic, or femoral bruits were heard. Peripheral pulses were intact and symmetrical. On cardiovascular examination, the heart rate and rhythm were regular with a prominent P2 component. There was a systolic murmur of grade III/VI intensity best heard at the lower left sternal border. The lungs were clear. No peripheral edema, hepatomegaly, clubbing, or cyanosis was present. Electrocardiography revealed sinus rhythm with a heart rate of 98/min, right axis deviation and incomplete right bundle branch block (Figure 1). Chest X-ray showed enlarged pulmonary arteries, cardiomegaly and prominent interstitial marking in the lower zones (Figure 2). Echocardiography demonstrated normal left ventricular size and function, normal aortic and mitral valves, severe right ventricular enlargement and dysfunction, severe tricuspid regurgitation and pulmonary arterial pressure of 120mmHg. Routine hematologic workup showed a normal ESR, normal rheumatologic panel (including rheumatoid factor and complement levels), and normal thyroid function tests. Computerized tomographic scan (CT) of the chest revealed multiple enlarged lymph nodes, enlarged main pulmonary artery, bilateral diffuse ground glass opacities and inter lobular septal thickening (Figure 3). Abdominal ultrasonography was normal. Perfusion ventilation scan of the chest demonstrated low probability for pulmonary embolism. The right heart catheterization (RHC) was done via the right subclavian vein and with the method of catheter tip. The RHC data are shown in Table 1. A diagnosis of PVOD was made according the imaging and catheterization data. Unfortunately, the patient did not respond to medical therapy and died after 8 months.

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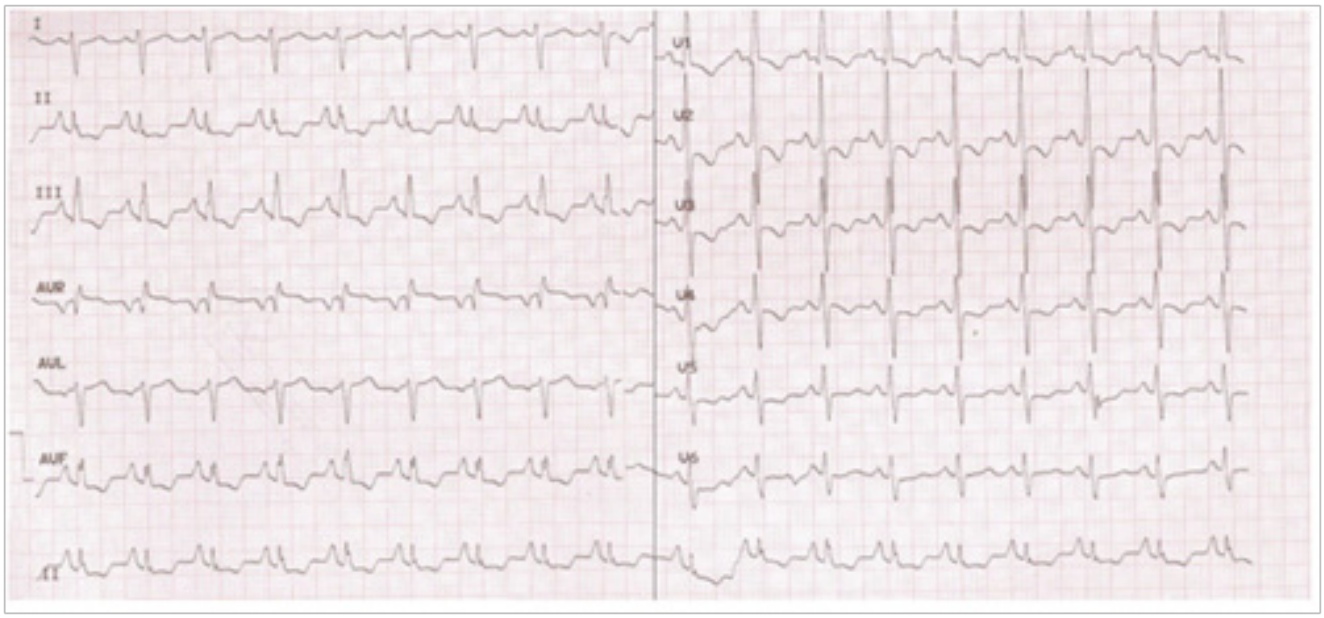

Figure 1 12 Lead ECG shows sinus rhythm, right axis deviation and incomplete RBBB.

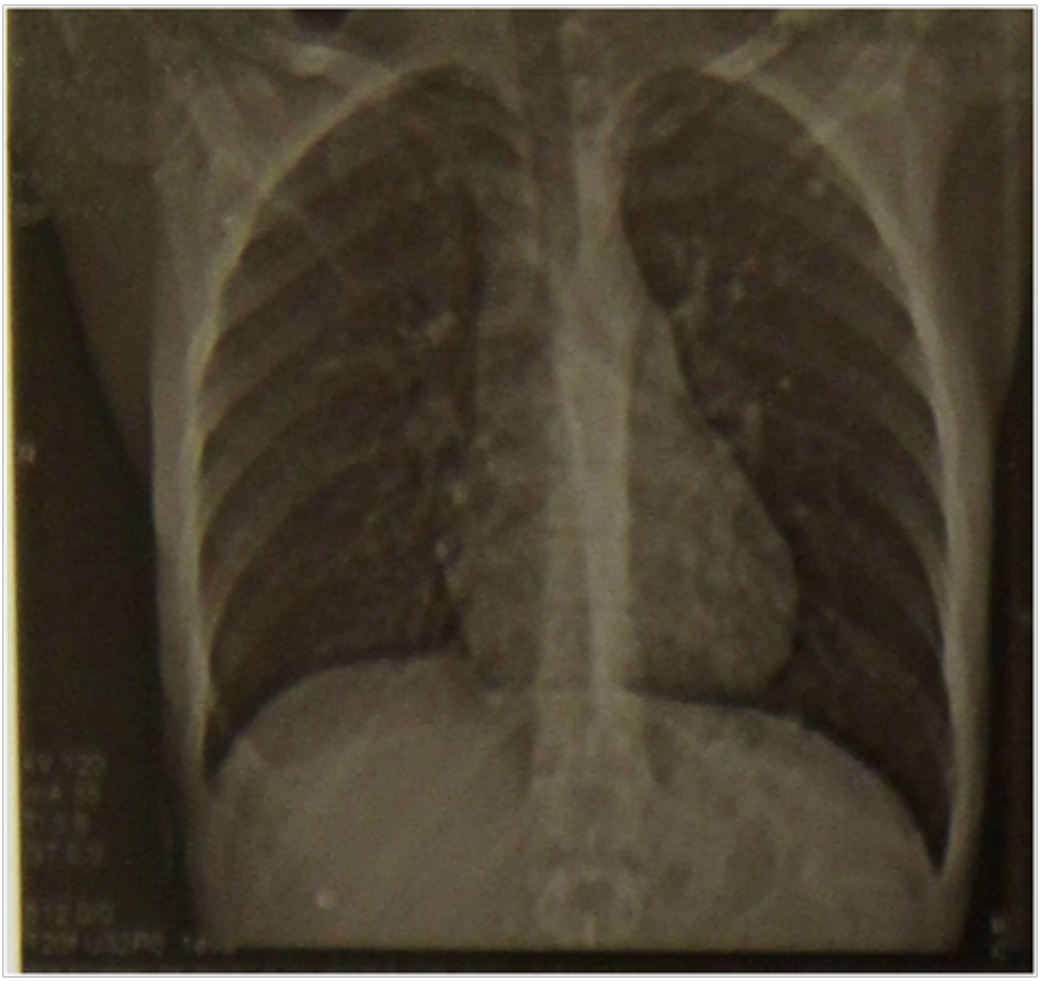

Figure 2 CXR shows enlarged pulmonary arteries, cardiomegaly and prominent interstitial marking in the lower zones.

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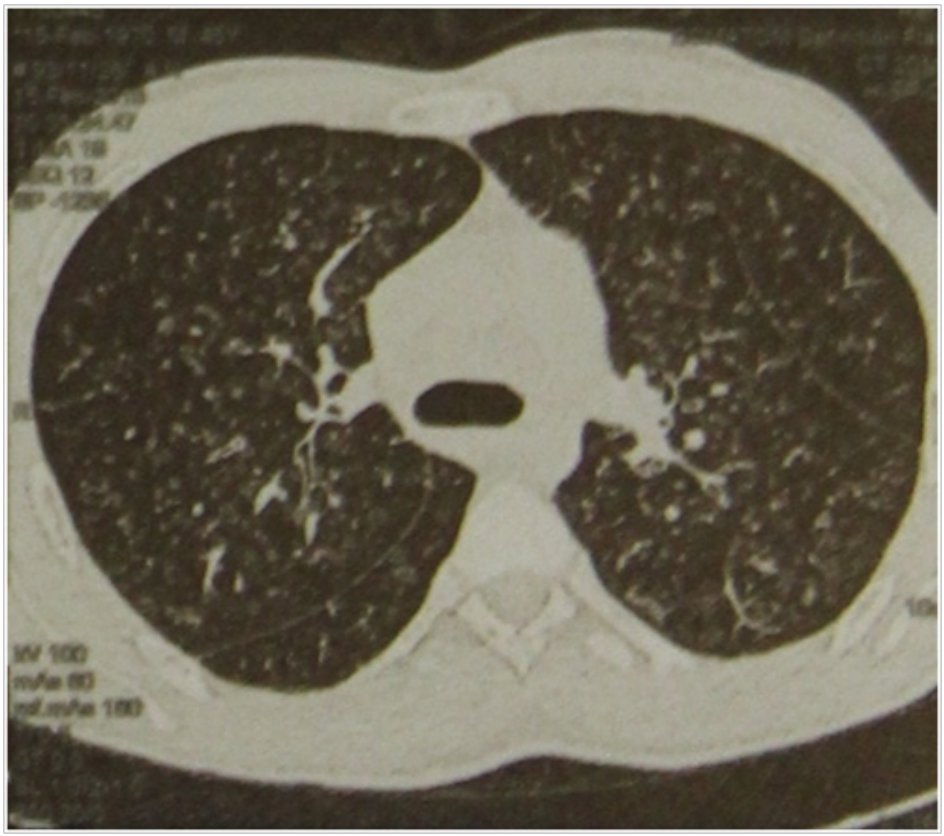

Figure 3 Computerized tomographic scan of the chest reveals multiple enlarged lymph nodes, enlarged main pulmonary artery, bilateral diffuse ground glass opacities and inter lobular septal thickening.

Table 1 The right heart catheterization data of the patient

Variable	
Cardiac output (Thermodilution) (L/min)	3
Cardiac index (L/min/m2)	1.8
Mean central venous pressure (mmHg)	4
Pulmonary arterial pressure (mean) (mmHg)	55/35 (41)
Pulmonary capillary wedge pressure (mmHg)	15
Pulmonary vascular resistance	8.6
Systemic arterial O2 saturation	85
Mixed venous O2 saturation	78

Discussion

Pulmonary veno-occlusive disease (PVOD) is a less common cause of pulmonary hypertension. Clinical presentation is non-specific and similar to other causes of pulmonary hypertension including dyspnea, fatigue and cough.[4] The diagnosis of PVOD needs a high clinical suspicion in a patient with no secondary causes for pulmonary hypertension. Up to 5-20% of these patients are classified as idiopathic pulmonary arterial hypertension (PAH).[5–7]

Imaging studies such as high-resolution CT may show the characteristic findings including pulmonary venous congestion and mediastinal lymphadenopathy.[8] The presence of normal spirometry findings with decreased diffusing capacity and the absence of a V/Q mismatch on perfusion-ventilation scan are also characteristics of PVOD.[8] On right heart catheterization, normal mixed venous saturation and elevated mean pulmonary artery pressure, pulmonary capillary wedge pressure and pulmonary vascular resistance will be seen, as in our patient. Although definite diagnosis is made by histopathologic findings on lung tissue specimen, bronchoscopic transbronchial lung biopsy is not recommended because of the risk of life-threatening bleeding due to the elevation in both pulmonary venous and arterial pressures.[8]

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The exact pathophysiology of PVOD is unknown, although genetic factors, infections such as HIV virus, toxic exposures, thrombotic diathesis and a wide range of autoimmune disorders such connective tissue disorders, sarcoidosis and granulomatous angiitis have been presumed as predisposing factors. Among many other causes of PVOD, there are only a few chemical exposures reported to be linked to POVD. Two cases of PVOD have been reported with oral contraceptive therapy.[9,10] PVOD has also been linked to chemotherapeutic agents such as bleomycin, mitomycin, and carmustine (BCNU).[11–13] Powdered cleaning products containing silica, soda ash, dodecyl benzyl sulfonate, and trichloro-s-triazinetrione are the other reported chemical exposures which were associated with the development of PVOD.[14]

In the present report, we introduce a case of PVOD in a young male with past history of major depressive disorder who was administered a combination of psychiatric medications including olanzapine, lithium, clonazepam and sertraline. To best of our knowledge, this is the first report of PVOD in a patient taking psychiatric medications.

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None.

Conflict of interest

The author declares no conflict of interest.

References

1. Brown CH, Harrison CV. Pulmonary veno–occlusive disease. *Lancet*. 1966;2(7454):61–65.
2. Heath D, Segel N, Bishop J. Pulmonary veno–occlusive disease. *Circulation*. 1966;34(2):242–248.
3. Weisser K, Wyler F, Gloor F. Pulmonary veno–occlusive disease. *Arch Dis Child*. 1967;42(223):322–327.
4. Huertas A, Girerd B, Dorfmuller P, et al. Pulmonary veno–occlusive disease: advances in clinical management and treatments. *Expert Rev Respir Med*. 2011;5(2):217–229.
5. Rounds S, Cutaia MV. Pulmonary hypertension: Pathophysiology and clinical disorders. In: Baum GL, et al. editors. *Textbook of Pulmonary Diseases*. Philadelphia, Pa: Lippincott–Raven; 1998. p. 1273–1295.
6. Wagenvoort CA. Lung biopsy specimens in the evaluation of pulmonary vascular disease. *Chest*. 1980;77(5):614–625.
7. Thadani U, Burrow C, Whitaker W, et al. Pulmonary veno–occlusive disease. *Q J Med*. 1975;44(173):133–159.
8. Montani D, O'Callaghan DS, Savale L, et al. Pulmonary veno–occlusive disease: recent progress and current challenges. *Respir Med*. 2010;104(Suppl 1):S23–S32.
9. Townend JN, Roberts DH, Jones EL, et al. Fatal pulmonary venoocclusive disease after use of oral contraceptives. *Am Heart J*. 1992;124(6):1643–1644.
10. Urisman A, Leard LE, Nathan M, et al. Rapidly progressive pulmonary venoocclusive disease in young women taking oral contraceptives. *J Heart Lung Transplant*. 2012;31(9):1031–1036.
11. Joselson R, Warnock M. Pulmonary veno–occlusive disease after chemotherapy. *Hum Pathol*. 1983;14(1):88–91.
12. Knight BK, Rose AG. Pulmonary veno–occlusive disease after chemotherapy. *Thorax*. 1985;40(11):874–875.
13. Porres–Aguilar M, Al–Bayati I, Porres–Muñoz M, et al. Pulmonary veno–occlusive disease as a cause of pulmonary arterial hypertension. *Proc (Bayl Univ Med Cent)*. 2015;28(3):350–352.
14. Liu L, Sackler JP. A case of pulmonary veno–occlusive disease: Etiological and therapeutic appraisal. *Angiology*. 1973;23:299–304.