Unusual presentation of more common disease/injury

An unusual cause of flash pulmonary oedema

Atul Vijay Palkar,¹ Makardhwaj Sarvadaman Shrivastava,² Nivedita D Moulick³¹Department of Internal Medicine, St Vincent Hospital, Worcester, Massachusetts, USA²Department of Internal Medicine, Indraprastha Apollo Hospitals, New Delhi, India³Department of Internal Medicine, Lokmanya Tilak Municipal Medical College & Hospital, Mumbai, India

Correspondence to Dr Makardhwaj Sarvadaman Shrivastava, makar_in@rediffmail.com, makardhwaj.shrivastava@gmail.com

Summary

We report a case of young woman who presented with acute cardiogenic pulmonary oedema and respiratory failure. She underwent emergent endotracheal intubation and was transferred to the intensive care unit. She responded to intravenous diuretics and positive pressure ventilation. Subsequent workup revealed that she had Graves' disease and was in thyrotoxic crisis. Therapy with propranolol and propylthiouracil was instituted to which she showed remarkable improvement.

BACKGROUND

Flash pulmonary oedema is a term used to describe a particularly dramatic form of acute decompensated heart failure and is commonly associated with diastolic dysfunction, hypertensive crisis, renal artery stenosis, coronary artery disease, obstructive sleep apnoea and Tako-tsubo cardiomyopathy.¹ Though thyrotoxic state has a variety of cardiac manifestations, acute heart failure and pulmonary oedema are uncommon particularly in the young.²

CASE PRESENTATION

A 27-year-old woman presented to the emergency department of our hospital with a sudden onset of shortness of breath at rest and orthopnoea. She denied chest pain, palpitations, cough with expectoration, haemoptysis, rash, fever, limb swelling and recent travel. There was no history of blurring of vision, headache, excessive sweating, lightheadedness, syncope or recent change in appetite or weight. She also denied history of oliguria, facial puffiness, cola-coloured urine, fleeting joint pains or involuntary movements.

The patient had no history of hypertension, heart disease and blood clots. She was not on oral contraceptive pills for birth control and had menses 15 days ago. There was no history of hypertension or heart disease in any family member or similar cases in the neighbourhood.

The patient belonged to a low socioeconomic class, with her husband being the sole earning member of their family.

On examination, her general condition was unstable with gasping respiration. She was thin built and afebrile; pulse was 140/min, regular in rhythm and bounding in character. Blood pressure was 120/58 mm Hg in the right upper extremity. Respiratory rate was 32/min with prominence of accessory muscles of respiration. There was mild pallor, jugular venous distension and a small diffuse goitre (figure 1) was noted with no appreciable bruit. She had a 'stare' with an eyelid lag. There was 1+ pedal oedema. The rest of the general examination was unremarkable. An examination of the cardiovascular system revealed a hyperdynamic apex with cardiomegaly. S1 was soft. S2

was normal. S3 gallop was present. No murmur or rub was appreciated. An examination of the respiratory system revealed fine basal crackles. The patient had an anxious look with no focal deficits. Per abdomen examination was normal. There was no renal bruit.

INVESTIGATIONS

The haemogram, blood chemistry and liver function tests were unremarkable. The chest x-ray (figure 2) showed cardiomegaly with peri-hilar interstitial infiltrates. The electrocardiogram showed sinus tachycardia. The arterial blood gas revealed respiratory acidosis with hypoxaemia. Brain natriuretic peptide was elevated to 3465 pg/ml; erythrocyte sedimentation rate and C-reactive protein level were normal. Echocardiography revealed mild mitral regurgitation and moderate global left ventricular dysfunction with an ejection fraction of 40%. Antinuclear antibody was positive with speckled pattern on immunofluorescence; anti ds-DNA antibody titres were negative. Thyroid function tests showed a suppressed thyroid-stimulating hormone level at 0.01 µU/ml and an elevated free T3 and free T4 of 228 ng/dl ($N = 80\text{--}200$) and

Figure 1 Mild diffuse enlargement of the thyroid gland.

Figure 2 Chest x-ray showing cardiomegaly and pulmonary oedema.

22.3 µg/dl ($N = 4.5\text{--}12.5$), respectively. The serum anti-thyroid antibody level was strongly positive at >1000 IU/l, suggesting autoimmune thyrotoxicosis. An ultrasound of the thyroid showed a diffuse goitre with increased vascularity.

DIFFERENTIAL DIAGNOSIS

- ▶ Renal artery stenosis
- ▶ Acute renal failure
- ▶ Pulmonary embolism
- ▶ Viral myocarditis
- ▶ Acute rheumatic fever
- ▶ Systemic lupus erythematosus
- ▶ Pheochromocytoma

TREATMENT

In the emergency department, the patient underwent emergent endotracheal intubation and was transferred to the medical intensive care unit with a provisional diagnosis of acute cardiac failure. On the basis of laboratory investigations, she was diagnosed with hyperthyroidism due to Graves' disease and was subsequently given propylthiouracil and low-dose propranolol. She improved clinically with intravenous diuretics and positive pressure ventilation and was weaned off the ventilator. Lugol's iodine or steroids were not given because of improvement in heart failure on diuretics and other supportive therapy.

OUTCOME AND FOLLOW-UP

A follow-up thyroid scan confirmed Graves' disease. She had no new symptoms on outpatient follow-up and continues to be on propylthiouracil.

DISCUSSION

Thyrotoxic crisis is a life-threatening exacerbation of hyperthyroidism, characterised by decompensation of one or more organ systems.³ It is usually associated with Graves' disease, although it can occur with toxic nodular goitre.⁴ It can sometimes present with subtle clinical

signs that can be missed. Thyrotoxicosis has long been known to be associated with several cardiopulmonary complications such as atrial fibrillation (atrial flutter and supraventricular tachycardia are uncommon), systolic hypertension, dyspnoea, functional cardiomyopathy and congestive cardiac failure.⁵ Additionally, few case reports have described unstable angina syndrome,⁶ isolated right ventricular failure^{7 8} and pulmonary hypertension⁹ in patients with hyperthyroidism/thyrotoxicosis. However, thyrotoxicosis manifesting as acute pulmonary oedema is exceedingly rare and extensive search of the literature revealed only one such case reported in a young pregnant woman from Chile.¹⁰

The rapid recognition of the thyrotoxic crisis and the institution of immediate drug therapy are critical in limiting the associated mortality ranging from 20% to 30%.¹¹

Learning points

- ▶ Although rare, flash pulmonary oedema can be the presenting feature of hyperthyroid state.
- ▶ Graves' disease may have subtle physical signs like eyelid lag and present with cardiovascular complications.
- ▶ Thyroid dysfunction is often difficult to diagnose in overt heart failure due to the common occurrence of sick euthyroid syndrome. Testing for thyroid function should be considered for unexplained heart failure even in young patients.
- ▶ Corticosteroids, propranolol, Lugol's iodine and propylthiouracil are recommended in thyrotoxic crisis.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Rimoldi SF**, Yuzepolakayab M, Allemanna Y, *et al*. Flash pulmonary edema. *Prog Cardiovasc Dis* 2009;**52**:249–59.
2. **Siu CW**, Yeung CY, Lau CP, *et al*. Incidence, clinical characteristics and outcome of congestive heart failure as the initial presentation in patients with primary hyperthyroidism. *Heart* 2007;**93**:483–7.
3. **Tietgens ST**, Leinung MC. Thyroid storm. *Med Clin North Am* 1995;**79**:169–84.
4. **Migneco A**, Ojetti V, Testa A, *et al*. Management of thyrotoxic crisis. *Eur Rev Med Pharmacol Sci* 2005;**9**:69–74.
5. **Fadel BM**, Ellahham S, Ringel MD, *et al*. Hyperthyroid heart disease. *Clin Cardiol* 2000;**23**:402–8.
6. **Glikson M**, Freimark D, Leor R, *et al*. Unstable angina syndrome and pulmonary oedema due to thyrotoxicosis. *Postgrad Med J* 1991;**67**:81–3.
7. **McDonough RJ**, Moul MS, Beckman D, *et al*. Isolated right ventricular failure in hyperthyroidism: a clinical dilemma. *Heart International* 2011;**6**:e11.
8. **Berlin T**, Lubina A, Levy Y, *et al*. Graves' disease presenting as right heart failure. *Isr Med Assoc J* 2008;**7**:217–18.
9. **Virani SS**, Mendoza CE, Ferreira AC, *et al*. Graves' disease and pulmonary hypertension. Report of 2 cases. *Tex Heart Inst J* 2003;**30**:314–15.
10. **Diaz NR**, Silva GD. Edema pulmonar agudo como primera manifestacion de hipertiroidismo en una mujer embarazada. [Acute pulmonary edema as a first manifestation of hyperthyroidism in a pregnant woman. Report of one case]. *Rev Med Chile* 2008;**136**:356–8.
11. **Gavin LA**. Thyroid crises. *Med Clin North Am* 1991;**75**:179.

This pdf has been created automatically from the final edited text and images.

Copyright 2012 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.

BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Palkar AV, Shrivastava MS, Moulick ND. An unusual cause of flash pulmonary oedema. *BMJ Case Reports* 2012;10.1136/bcr.03.2012.6087, Published XXX

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow