CASE REPORT

Pulmonary hypertension and predominant right heart failure in thyrotoxicosis

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Resuscitation; Pulmonary hypertension; Heart failure; Amiodarone

Summary
In this report we discuss a patient with predominant right heart failure and pulmonary hypertension, caused by thyrotoxicosis due to Graves disease, which deteriorated to asystole, due to amiodarone administration for rapid atrial fibrillation.

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Introduction
Pulmonary hypertension is not a recognized complication of thyrotoxicosis, although there are few reports describing this phenomenon. In most of these reports, the course of events was not life threatening following adequate treatment for thyrotoxicosis. Amiodarone is used to reduce ventricular rate response in rapid paroxysmal atrial fibrillation (RPAF). Amiodarone may also induce thyrotoxicosis, which might trigger or worsen RPAF. Thus, many physicians advise against giving amiodarone for RPAF in urgent situations without first excluding thyrotoxicosis, because other agents are available to treat RPAF.

Case report
A 38-year-old unconscious Philippine female was admitted to the emergency room with hypoglycaemia (30 mg/dL). Consciousness returned following intravenous glucose administration. The ECG showed rapid atrial fibrillation and she was treated with an intravenous bolus of amiodarone by the paramedics. Her medical history revealed a thyroidectomy a few years earlier for an unclear indication. She did not return for further follow-up although she had palpitations, probably due to

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a lack of medical insurance, being an illegal employee in Israel. Physical examination revealed
dyspnoea, diaphoresis, jaundice, high fever, dis-
tended jugular veins, hepatomegaly, ascites, ankle
oedema and diffuse bilateral rales on auscultation
of the chest. A chest roentgenogram showed
prominent vascular marking. Laboratory tests
yielded leukocytosis with neutrophilia, mildly
elevated cellular liver enzymes, mildly unconjugated
bilirubinaemia, and a prolonged prothrombin time
and partial prothrombin time. The patient was
given oxygen through a facial mask, intravenous
furosemide for pulmonary congestion and intra-
venous ceftriaxone as an empirical treatment for
aspiration pneumonia.
An hour later, the patient deteriorated to asys-
tole requiring prolonged cardiopulmonary resusci-
tation before a return of spontaneous circulation.
Further laboratory tests yielded an undetectable
(<0.01 mU/L) serum level of thyroid-stimulating
hormone (TSH), free thyroxin (FT4) serum level of
10.9 ng/dL (normal, 0.7–1.8 ng/dL) and a total tri-
iodothyronine (TT3) serum level of 6.38 ng/dL (nor-
mal, 80–200 ng/dL). Anti-thyroid peroxidase anti-
bodies were positive at 803.0 U/mL. The diagnosis
of thyrotoxicosis (Grave’s disease) was established
and the patient was started on propranolol, propy-
lthiouracil and dexamethasone.
Two hours later, the patient underwent a
transthoracic echocardiogram which revealed a
dilated right ventricle, general reduced right ven-
tricle systolic function, severe elevated systolic
pulmonary artery pressure of 70 mmHg and mod-
erate tricuspid valve regurgitation. The left ventri-
cle was normal sized with an ejection fraction of
55% (Figure 1). A ventilation–perfusion pulmonary
scan demonstrated a low probability for pulmonary embolism.
During the following days the patient’s clinical
condition improved gradually and she was extu-
bated. Two weeks later, a second transthoracic
echocardiogram was performed revealing a normal
sized right ventricle with normal systolic function,
mild elevated systolic pulmonary artery pressure
of 48 mmHg and mild tricuspid valve regurgitation.
Finally, the patient was discharged and visited our
outpatient clinic several weeks later with no signs
or symptoms of heart failure and in good clinical
condition. TSH, FT4 and TT3 serum levels were also
within the normal laboratory range.

Discussion
Pulmonary hypertension is defined as a mean pul-
monary artery pressure higher than 25 mmHg at
rest.1 There are few reports on pulmonary hyper-
tension secondary to thyrotoxicosis2—9; excess thy-
roid hormone might cause pulmonary hypertension
by increasing the cardiac output and by constricting
the pulmonary artery.13 In most of these reports,
thyrotoxicosis is due to Grave’s disease 2—9; thy-
roid auto-antibodies secreted in Grave’s disease
might injure the pulmonary endothelium, further
contributing to pulmonary hypertension.9 Never-
theless, pulmonary hypertension is not recognized
as a complication of thyrotoxicosis,1 probably due
to the favourable outcome described in previous
reports.2—9 We wish to raise the level of suspicious
for pulmonary hypertension secondary to thyrotox-
icosis, in light of our experience.
Amiodarone is a class III anti-arrhythmic agent
which prolongs AV nodal conduction and is used to
lower ventricular rate response in RP AF .10 Amio-
darone, which has a high iodine content, might
induce thyrotoxicosis by loading iodine on the
thyroid gland and by inducing a thyroiditis-like
condition,11 and in turn, may trigger or worse
RP AF.10 This is of significance, since 6.2% of the
patients who present to an emergency department
with RP AF also have thyrotoxicosis.14 Amiodarone
is generally considered to be contraindicated in
patients with RP AF secondary to thyrotoxicosis and
there are strong opinions against empirical adminis-
tration of amiodarone for RP AF without first exclud-
ing thyrotoxicosis.12 We join these calls and ask for
more careful use of amiodarone.

This presentation of thyrotoxicosis was mainly
due to an untreated Grave’s disease and associ-
ated with the administration of amiodarone. The
consequences of health neglect due to lack of med-
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Critical insurance are frequently seen in the emergency departments. There is a large population of foreign employees in Israel, particularly in Tel-Aviv, most of whom are hiding from the emigration authorities and do not have medical insurance. These circumstances are also prevalent in other cities in developed countries. Unfortunately, only severe distress brings these poor people to seek medical treatment.

Conclusions

Pulmonary hypertension and heart failure in a patient with neglected Grave’s disease deteriorating to cardiopulmonary collapse, associated with the administration of amiodarone, have not been reported yet. We wish to raise the level of awareness in patients with pulmonary hypertension and thyrotoxicosis and the danger of amiodarone administration without first excluding thyrotoxicosis.

Conflict of interest statement

None.

References