

Hypothyroidism: A Reversible Cause Of Heart Failure

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BACKGROUND

Hypothyroidism is characterized by a decrease in oxygen and substrate utilization by major organ systems of the body. Hence patients with angina pectoris have fewer symptoms if they become hypothyroid¹. Hypothyroidism also results in bradycardia and weakening of myocardial contraction and relaxation. Cardiac preload is decreased due to impaired diastolic function, cardiac afterload is increased, and chronotropic and inotropic functions are reduced². The impairment of the measures of left ventricular performance leads to a reduction in cardiac output³.

CASE REPORT

A 69-year-old male presents after a syncope with cardiac arrest. After receiving CPR, his ECG showed incomplete right bundle branch block and left anterior fascicular block. He was started on a heparin drip for elevated cardiac enzymes. The patient reported no history of angina, syncope or seizure disorder except recent onset fatigue and dyspnea on exertion. He also complained of recent hair loss, hoarse voice and scrotal swelling. The patient had no thyromegaly but had bilateral non-pitting pedal edema with delayed reflexes. Initial results showed, TSH of 122.1 mcU/mL (0.4 – 4.0 mcU/mL), free T4 of <0.02 ng/dL (0.7 – 1.9 ng/dL), total T3 of 22 ng/dL (80–200 ng/dL). Thyroglobulin and thyroid microsomal antibodies were in reference range. His mental status was intact and showed no features of myxedema coma. He was started on a 50 mcg of levothyroxine (LT4) to improve cardiac output without exacerbating an acute coronary syndrome or an arrhythmia. Hydrocortisone was started prior to administering LT4, till co-existing adrenal insufficiency was ruled out. Patient had a transthoracic ECHO that showed severe left ventricular systolic dysfunction with an ejection fraction (EF) of 25% and global hypokinesis with regional variation. Left heart catheterization showed triple vessel coronary artery disease (CAD) without complete vessel occlusion. However from the left ventriculogram, the EF improved to 60% after 3 days of LT4. The free T4 also increased to 0.2 ng/dL. Eventually the patient had a coronary artery bypass grafting (CABG) where intraoperatively, the EF remained stable at 50%.

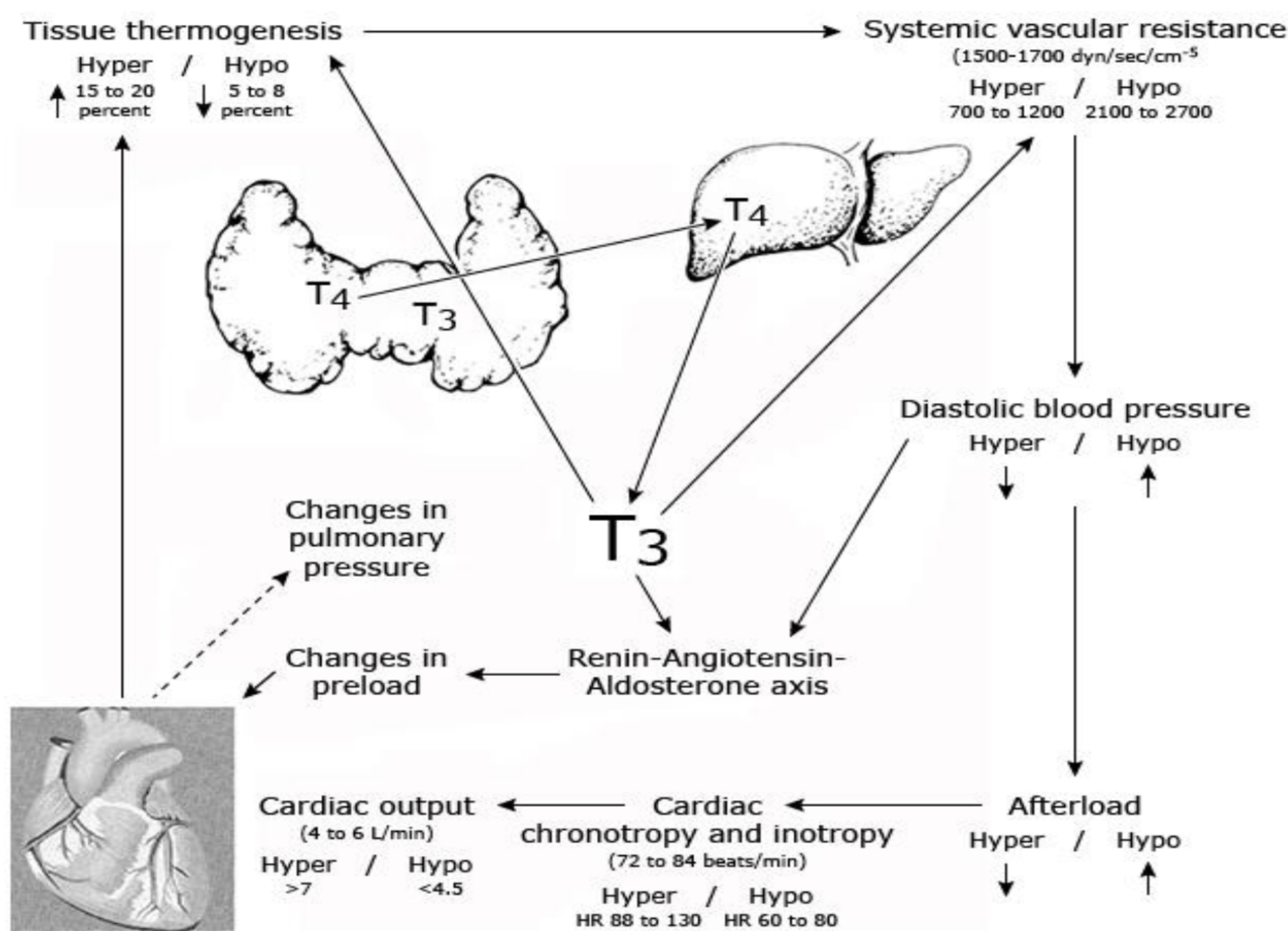


Figure 1¹

DISCUSSION

Our case is distinct in describing a patient who was profoundly hypothyroid, yet had no features of myxedema coma and showed dramatic improvement with initiation of LT4. The improvement in cardiac contractility prior to the CABG demonstrates the relationship between hypothyroidism and left ventricular dysfunction, and its reversible nature with restoration of thyroid function. This is due to the improvement in chronotropic and inotropic function of the heart. Studies exist in children to show that T3 improves cardiac hemodynamics in 48-96 hours⁵ while T4 takes weeks to months. In adults T4 is used traditionally for thyroid replacement. Replacement dose was determined by risk benefit analysis balancing the clinical suspicion for CAD with the severe hypothyroidism and impaired ejection fraction, thus replacement T4 dose was higher than usual in known CAD.

TAKE HOME POINTS

- Heart failure can be caused by hypothyroidism alone, although rare².
- While caution is needed with LT4 replacement therapy with concomitant CAD, there can be beneficial effects of the hormone replacement on cardiac function⁴.
- Untreated hypothyroidism in patients with ischemic heart disease should not be an absolute contraindication to coronary artery bypass surgery⁶.

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