An Unusual Case of Decompensated Heart Failure in a Young Man

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CASE DESCRIPTION

A 21-year-old man presented to the emergency department (ED)[3] with chest pain, worsening shortness of breath, lethargy, nausea, vomiting, and palpitations. He presented to another ED the previous day with similar symptoms and was sent home with a diagnosis of anxiety and panic attack. His only relevant past medical history was repaired Chiari malformation. There was no family history of ischemic heart disease. His lipid concentrations were as follows: total cholesterol, 79 mg/dL [2.05 mmol/L; reference interval (ref), <200], HDL cholesterol, 26 mg/dL (0.67 mmol/L; ref, 40–199), LDL cholesterol, 22 mg/dL (1.14 mmol/L; ref, 0–129), triglycerides, 43 mg/dL (0.49 mmol/L; ref, 0–150), and he had a BMI (body mass index) of 29. The patient was otherwise in normal health.

On arrival, his troponin I was 47.94 ng/mL (Siemens Dimension; ref, <0.24). Electrocardiography (ECG) was performed that revealed sinus tachycardia (140 beats/min) without elevation of ST-segments. As a result, a β blocker was administered for non-ST-elevation myocardial infarction. The patient also developed severe hypoxic respiratory failure with an arterial PO2 of 62 mmHg (ref, 80–100), which required oxygen by nonrebreathing mask at 25 L/min. He was later intubated due to increased respiratory fatigue (respirations 26/min), hypotension (blood pressure 85/57), and increased oxygen requirements. The patient also became oliguric with creatinine increasing to 1.9 mg/dL. He was acidotic, hyperkalemic, and his liver function test results were high (Table 1). There was clinical concern for infection, but all cultures were negative. Furthermore, a urine drug screen, antinuclear antibody, antineutrophil cytoplasmic antibody, and coagulation testing were all normal. Thyroid testing was performed that revealed a thyroid stimulating hormone (TSH) <0.04 mcIU/mL, free thyroxine (FT_4) of 0.6 ng/dL (7.7 pmol/L) (ref, 0.7–1.3), and free triiodothyronine (FT_3) >20.0 pg/mL (30.8 pmol/L; ref, 2.4–4.2).

Twenty-four hours after admission, bedside ECG demonstrated evidence of acute decompensated heart failure (HF) with worsening left ventricular dysfunction and an ejection fraction of 21%. Therefore, the β blocker was stopped and dobutamine was started despite tachycardia. The patient's heart rate soon improved to 110, his urine output improved, and troponins trended down. Three days following admission, his symptoms improved dramatically, and extubation was performed; no further complaints of dyspnea or chest pain were received.

3 Nonstandard abbreviations: ED, emergency department; ref, reference interval; ECG, electrocardiogram; TSH, thyroid stimulating hormone; FT_4, free thyroxine; FT_3, free triiodothyronine; HF, heart failure.

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Table 1. Selected laboratory results.[a]

	Reference interval	Admission	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Discharge
Troponin I	<0.24 ng/mL	47.94	32.13	19.52					0.29
Creatine kinase	30–200 Units/L	1760	1634	1291					
Triglycerides	0–150 mg/dL (0–1.70 mmol/L)	43 (0.49)							
HDL cholesterol	40–199 mg/dL (1.04–5.15 mmol/L)	26 (0.67)							
LDL cholesterol	0–129 mg/dL (0–3.34 mmol/L)	44 (1.14)							
Total cholesterol	<200 mg/dL (<5.18 mmol/L)	79 (2.046)							
TSH	0.35–5.50 mcIU/mL	<0.04	0.04						2.94
FT_4	0.7–1.3 ng/dL (9.0–16.7 pmol/L)	0.6 (7.7)	0.83 (10.68)						
FT_3	2.3–4.2 pg/mL (3.5–6.5 pmol/L)	>20.0 (3.08)	8.4 (12.9)	5.4 (8.3)					
Aspartate transaminase	11–47 Units/L	95	204	211	156	954	527	252	106
Alanine transaminase	7–53 Units/L	144	147	187	1605	1523	1111	642	587
Creatinine	0.7–1.3 mg/dL (61.9–114.9 µmol/L)	1.27 (112.3)	1.21 (107.0)	0.91 (80.4)	1.9 (168.0)	1.63 (144.1)	1.4 (123.8)	1.3 (114.9)	1.11 (98.1)
Sodium	135–145 mmol/L	138	141	142	141	143	143	140	139
Potassium	3.3–4.9 mmol/L	4.5	4.3	4.4	6.6	5.2	4.6	4.4	4.2

[a] Results listed as conventional units (SI units).

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QUESTIONS TO CONSIDER
• What are some causes of decompensated heart failure in otherwise healthy, young individuals?
• What are the indications for thyroid testing in acutely ill patients?
• What are some common causes of acute thyrotoxicosis?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the January 2019 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol65/issue1 and follow the link to the Clinical Case Study and Commentaries.

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