

Shock in a patient with anorexia nervosa

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46 yo, female

- General fatigue, dyspnea on exertion
- Diagnosed as anorexia nervosa at her 20's.
- After a few psychiatric consultation, she stopped seeking treatment and continued a diet consisting of vegetables and tofu.
- 149 cm, 23 kg
- BP 99/76, HR 98
- TP 4.5 g/dL, Alb 2.9 g/dL, T-ch 91 mg/dL, TG 1 mg/dL, Na 128 mEq/L, K 3.4 mEq/L, BNP 876.9 pg/mL

(Sakamoto Y, Nutrition 2017;35:148)

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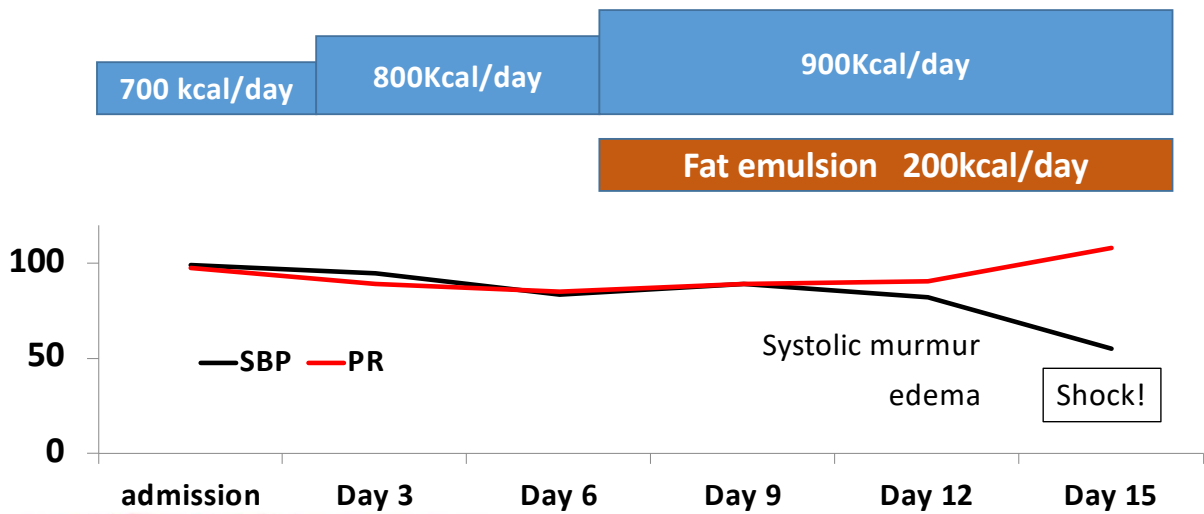
Echo on admission



LVEDD 40 mm, LVESD 35 mm
IVSTh 5 mm, PWTh 6 mm
IVC 5 mm
EF 21%
Diffuse hypokinesis

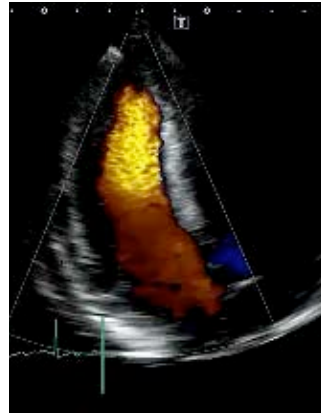
Severe depletion of the energy substrates may cause systolic dysfunction

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Echo on Day 15



LVEDD 19 mm, LESD 10 mm
 IVSTh 12 mm, PWTh 12 mm
 IVC 2 mm
 EF >70%

Mid LV obstruction Δ PG 93 mmHg

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What happened?

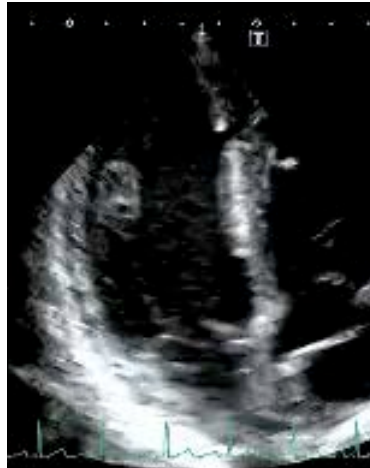


Hyperdynamic state and small LV leads to intra LV obstruction.
 Oversupply of fatty acid may activate cardiac metabolism overly and cause hypercontraction.

Fat emulsion was stopped and fluid replacement was started.
 She recovered from shock within 24 hrs.

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Echo on Day 40



LVEDD 28 mm, LVESD 17 mm
 IVSTh 9 mm, PWTh 9 mm
 IVC 15 mm
 EF 71%

No intra LV obstruction

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Cardiac complications in anorexia nervosa



Prolonged severe malnutrition induces fat and protein catabolism leading to diminished intracellular protein and glycogen synthesis.

- QT prolongation
- Decrease in LV mass
- MV prolapse
- Pericardial effusion
- LV dysfunction
-

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Refeeding syndrome

Refeeding activates anabolic processes, protein and glycogen synthesis, glucose phosphorylation, and glycolysis.

These lead to a drastic increase in the cellular uptake of phosphorus, potassium, magnesium, thiamine and water.

- Hypophosphatemia
- Hypokalemia
- Hypomagnesemia
- Vitamin B1 deficiency
- Fluid overload
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- Hypercontraction⇒Intra LV obstruction

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