Myocardial infarction with ventricular septal rupture and cardiogenic shock

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Figure. Electrocardiogram recorded on the patient's arrival in the emergency department. See text for explication. Also, note that there was sinus rhythm at a rate of 106 beats/min with the next to last QRS complex being a fusion of a premature ventricular complex with a sinus-initiated complex.

A 72-year-old woman with a history of systemic arterial hypertension presented to the emergency department with chest pain, and an electrocardiogram showed acute anterior myocardial infarction with striking ST-segment elevation, upright T waves, and large Q waves in leads V_1 to V_4 (Figure). Furthermore, the ST-segment elevation > 0.25 mV in lead V_1 , the ST depression ≥ 0.1 mV in lead III, and the ST elevation ≥ 0.1 mV in lead aVL indicated that the left anterior descending coronary artery was occluded proximal to both the first major septal perforating branch and the first diagonal branch (1–4).

Anterior infarcts from such proximal left anterior descending occlusions tend to be quite large and are often associated with cardiogenic shock (2, 4, 5), which this woman manifested soon after her arrival in the emergency department. An echo-

cardiogram showed a left ventricular ejection fraction of approximately 40% with akinesis of the apex and distal two-thirds of the ventricular septum. The right ventricle was considerably dilated. Through a defect in the distal ventricular septum, blood shunted from left to right during systole with reversal of flow during diastole, suggesting an elevated right ventricular diastolic pressure. Laboratory studies of the patient's blood confirmed

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Table. Laboratory results from blood drawn on admission

Test	Flag	Value	Reference range
CBC			
White blood cell count (10 ³ /μL)	High	22.1	4.5–11.0
Red blood cell count (10 ⁶ /μL)	Low	3.43	4.0–5.2
Hemoglobin (g/dL)	Low	10.4	12.0–16.0
Hematocrit (%)	Low	31.4	35–46
Mean corpuscular volume (fL)		91.4	80–100
Mean corpuscular hemoglobin (pg)		30.2	26–34
Absolute neutrophil count–segmented (10 ³ /μL)	High	17.02	1.8–8.0
Absolute neutrophil count–bands (10 ³ /μL)	High	2.65	0–0.9
Basic metabolic profile			
Sodium (mmol/L)		145	135–146
Potassium (mmol/L)		3.8	3.6–5.2
Chloride (mmol/L)		98	96–107
CO ₂ (mmol/L)	Low	11	24–32
Glucose (mg/dL)	High	275	65–109
Blood urea nitrogen (mg/dL)		18	7–25
Creatinine (mg/dL)	High	2.9	0.7–1.2
Calcium (mg/dL)	Low	6.4	8.4–10.3
Magnesium (mg/dL)	High	3.6	1.5–2.6
Phosphorous (mg/dL)	High	10.6	2.5–4.7
Cardiac profile			
Creatine kinase (U/L)	High	710	<190
Creatine kinase MB (ng/mL)	High	68.6	<4.0
Creatine kinase MB index (%)	High	9.7	<3.0
Cardiac troponin (ng/mL)	Panic high	17.85	<0.05

myocardial damage and indicated chronic kidney disease and acute metabolic acidosis (Table).

The patient had a run of ventricular tachycardia while in the emergency department. Once stable, she was transported to the cardiac catheterization laboratory; an intra-aortic balloon was placed via the left femoral artery, and 1:1 counterpulsation was begun. Coronary arteriography, performed via the right femoral artery, showed proximal total occlusion of the left anterior descending coronary artery, diffuse disease of the left circumflex coronary artery with a totally occluded first obtuse marginal branch and 90% narrowing of the third obtuse marginal branch, and diffuse disease of the right coronary artery with a long 80% to 90% narrowing in its mid segment. Despite repeated inflations of first a 2.5 × 20 mm balloon and then a 2.0 × 25 mm balloon in the proximal and distal portions of the left anterior descending artery, there was no flow. Hypotension, bradycardia, and acidosis (arterial blood pH = 6.9; reference = 7.4) were temporarily combated with atropine, epinephrine, and sodium

bicarbonate, and the patient was sent to the intensive care unit with an intravenous dopamine infusion and 1:1 intra-aortic balloon counterpulsation. She died several hours later.

Ventricular septal rupture formerly occurred in 1% to 3% of patients with acute myocardial infarction and did so within the first week in more than 75% of them (6). Subsequently, among patients treated with fibrinolysis, the incidence of ventricular septal rupture was only 0.2%, and the median time from the onset of the infarct was 1 day (7). Approximately one-half of ventricular septal ruptures are in the setting of an anterior infarct, and one-half occur in patients with inferior infarcts (8, 9). At least two-thirds of patients with myocardial infarction and ventricular septal rupture die, and many of these, like our patient, are older and have cardiogenic shock; early operation by superb surgeons seems to improve the chances of survival (10).

The large upright fused ST segments and T waves seen in the figure have been referred to as “tombstoning,” probably because of the appearance of tombstones and because they are signs of more extensive myocardial damage and of a poor prognosis (11). Sclarovsky believes that this pattern of deep anterior Q waves with persistently elevated ST segments and tall T waves indicates lack of reperfusion of the myocardium due to a severely damaged microcirculation (12).

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