Regional Left Ventricular Dysfunction in a Patient With Severe Prolonged Anemia

Satoshi Murao, MD*; Hiroyuki Fujieda, MD; Taisuke Sakamoto, MD; Tetsuya Sato, MD; Hirohiko Sato, MD

A 47-year-old woman with severe prolonged anemia developed heart failure. After treatment of the heart failure and anemia, she showed regional dysfunction of the left ventricular wall and myocardial fatty acid metabolism was disturbed in these sites. Coronary arteriography showed normal images. It took about 4 months to recover both left ventricular wall motion and fatty acid metabolism. Prolonged decrease of oxygen supply to the myocardium, which is caused by severe prolonged anemia, seemed to affect the myocardial function in this case, which could be another model of anemia-related myocardial dysfunction. (Jpn Circ J 1999; 63: 61–63)

Key Words: Anemia; Fatty acid metabolism; Hibernating myocardium; 123I-[-I]-methyliodophenyl pentadecanoic acid (BMIPP) scintigraphy; Left ventricular dysfunction

Case Report

A 47-year-old woman, with a 14-month history of general fatigue, developed shock after taking nitroglycerine and was transferred to hospital on March 4, 1997. On physical examination, her face was pale, systolic blood pressure was 67 mmHg and heart rate was 87 beats/min. Chest roentgenography revealed cardiomegaly (cardiothoracic ratio 59%) and pulmonary congestion. ECG showed ST segment depression and T wave inversion in I, II, aVF and V3–6 (Fig 1). Severe anemia was noted (red blood cell count 81×104/μl, hemoglobin 1.4 g/dl, hematocrit 6%), which we concluded had evoked her heart failure and ECG abnormalities. With vigorous fluid supplement therapy, transfusion, and the administration of cathecholamine, she recovered from the shock state within 1 h. Transfusion totaling 1040 ml of concentrated red blood cells over 9 h restored her hemoglobin level to 6.9 g/dl.

We re-examined her cardiac function after the anemia improved. Inverted T waves were observed in V3-6 on March 10 and they changed to positive waves by March 26.

Regarding the left ventricular wall motion, hypokinesis of the septum and anterior wall was revealed by echocardiography performed on March 10 (Fig 2), and had partially improved by March 29. Fatty acid metabolism of the myocardium examined by 123I-[-I]-methyliodophenyl pentadecanoic acid (BMIPP) scintigraphy on March 25 demonstrated disturbance in the apex and anterior wall (Fig 3). Hypokinesis of segment 3 was still present on April 4, as...

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Departments of *Internal Medicine and Cardiovascular Medicine, Matsuyama Shimin Hospital, Ehime, Japan
Mailing address: Satoshi Murao, MD, Department of Internal Medicine, Matsuyama Shimin Hospital, Matsuyama, Ehime 790-0067, Japan

Fig 1. Electrocardiography on March 4.
shown by left ventriculography, and coronary arteriography revealed normal coronary arteries except for 50% stenosis of the right ventricular branch and obtuse marginal branch. The cause of anemia was confirmed to be bleeding from the myoma uteri and a hysterectomy was performed on April 17.

In July, her ECG showed no ST segment or T wave change, echocardiography showed normal left ventricular wall motion and BMIPP scintigraphy revealed improvement of fatty acid metabolism in the apex and anterior wall (Fig 3).

**Discussion**

Cardiovascular symptoms may develop in anemic patients. Heart failure results from sustained increases in cardiac output, and angina pectoris may occur when the oxygen supply to the myocardium is inadequate even with normal coronary arteries. The present patient demonstrated common cardiovascular findings observed in severe anemia, such as angina-like symptoms, ECG changes and heart failure. Furthermore, she showed hypokinesis of the left ventricular septum and anterior wall, and disturbance of fatty acid metabolism in these sites. Left ventricular wall motion gradually improved after treatment of the anemia. Myocardial fatty acid metabolism also normalized after 4 months. Prolonged left ventricular dysfunction with disturbed fatty acid metabolism has not been reported as a cardiovascular complication of anemia.

Reversible left ventricular dysfunction is sometimes observed in patients with coronary artery disease. ‘Stunned myocardium’ refers to prolonged left ventricular dysfunction after a period of acute ischemia and coronary reperfusion; whereas ‘hibernating myocardium’ refers to chronic left ventricular regional dysfunction arising from prolonged myocardial hypoperfusion. Although the present patient had no coronary artery disease, there are some similarities between stunned or hibernating myocardium and this case as regards the myocardial oxygen supply. It could be speculated that severe prolonged anemia caused inadequate oxygen supply to the myocardium, and it caused myocardial damage similar to that seen in myocardial hibernation. Falling blood pressure when the patient went into shock further decreased the oxygen supply to the myocardium, and then left ventricular dysfunction developed. Left ventricular function recovered when the oxygen supply to the myocardium was restored by treating the anemia. Chronic hypoxemia in severe anemia and the further decrease of oxygen supply in the shock state seemed to evoke the left ventricular dysfunction in the present case. Besides these mechanisms, there is a possibility that subendocardial infarction developed when she was in the shock state. In patients with coronary artery disease it is sometimes difficult to distinguish subendocardial infarction and stunned or hibernating myocardium as they often coexist. Nevertheless, the present patient’s clinical findings and course could not be explained by subendocardial infarction alone.

Recovery of the left ventricular wall motion took more than 1 month in this case. It is known that prolonged myocardial ischemia causes myocardial degeneration and the severity of the histological changes affects the functional recovery from hibernation! The patient may have had myocardial degeneration during the severe anemia as a result of myocardial hypoxemia.

Impaired fatty acid metabolism and increases in glucose utilization are signs of an ischemic myocardium. Although glucose utilization was not assessed in the present case, a
change in fatty acid metabolism was detected by BMIPP scintigraphy, which is a useful tracer for detecting ischemic myocardium, and the time-dependent change of myocardial fatty acid metabolism reflected the recovery from ischemia.

These abnormal findings were observed only in part of the left ventricle. Myocardial blood flow is known to be heterogeneous at the level of the chamber, the layers of the left ventricular wall, or micro-regions within the layers. These differences reflect the regional differences in work and oxygen demand. Regional left ventricular dysfunction in the present case may be the result of the differences in oxygen demand among the regions of the left ventricular myocardium, as the decreased oxygen supply caused by severe anemia is equal in each part of myocardium.

As indicated in the case reported here, the condition of 'reversible left ventricular dysfunction' may be observed in situations of prolonged decrease of oxygen supply to the myocardium, such as in severe anemia. This could be another model of anemia-related myocardial dysfunction.

References