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Unexpected Acute Myocardial Infarction Due to Disseminated Intravascular Coagulation

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Abstract An autopsy case of gastric cancer accompanied by disseminated intravascular coagulation (DIC) that resulted in unexpected acute myocardial infarction (AMI) is reported. A 66-year-old man was introduced to our hospital and was diagnosed with gastric cancer accompanied by bone marrow carcinomatosis with DIC. On the fourteenth day of admission, he experienced dyspnea. He died on the fifteenth day. An autopsy was performed. The autopsy findings revealed myocardial infarction from the lateral to anterior and posterior ventricular walls, thromboembolism in the right coronary artery, and nonbacterial thrombogenic carditis of the mitral valve. A diagnosis of AMI was made by the autopsy findings. In cases accompanied by DIC, thrombus may develop in coronary arteries, resulting in AMI. In these patients, the typical symptoms of AMI and ST-segment elevation and pathologic Q waves on electrocardiogram are often not observed. The importance of this phenomenon warrants consideration in clinical practice.

Keywords: *disseminated intravascular coagulation, acute myocardial infarction*

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1. Introduction

Bone marrow carcinomatosis is a complication of cancers that often leads to disseminated intravascular coagulation (DIC) and microangiopathic hemolytic anemia (MAHA). We report of an autopsy case of gastric cancer accompanied by DIC that resulted in unexpected acute myocardial infarction (AMI).

2. Case Presentation

A 66-year-old man was introduced to our hospital, because of subcutaneous bleeding on the body and legs, abnormal blood examination results, severe back pain for 2 months, and tarry stool for a month. He had a history of hypertension and gastric ulcer, but no history of diabetic militaries, hyperlipidemia. He had a took antihypertensive drug. He has smoked 20 cigarettes per day for 45 years. At the admission, physical examination revealed the following: clear consciousness, Eastern Cooperative Oncology Group (ECOG) performance status 1, body temperature 36.6°C, blood pressure 102/73 mmHg, pulse rate 109 beats per minutes and oxygen saturation 96 % in room air. Subcutaneous bleeding was noted on the body and legs. No palpable lymph nodes were observed. Chest auscultation revealed no rales and murmurs. His abdomen was soft, without distention, rebound tenderness, guarding and palpable hepatosplenomegaly. He had no leg edema. His clinical course is shown in Figure 1. Blood examination revealed anemia, thrombocytopenia, leukoerythoblastosis, coagulation disorder, hyperfibrinolysis, and high levels of serum lactate dehydrogenase (LDH), alkaline phosphatase (ALP), serum carcinoembryonic antigen (CEA), and serum carbohydrate antigen 19-9 (CA19-9) (Table 1). A bone marrow biopsy revealed invasion of non-hematopoietic cells in the bone marrow (Figure 2). A diagnosis of bone marrow carcinomatosis complicated by DIC and MAHA was made using bone marrow biopsy results and laboratory data. His anemia and thrombocytopenia were treated with blood transfusions, and back pain was treated with acetaminophen and oxycodone. A chest and abdominal computed tomography (CT) showed thickening of the stomach, bilateral pleural effusions, and mediastinal and axillary lymphadenopathy. High levels of serum CEA and CA19-9 and, CT findings suggested a presence of a malignant gastrointestinal tumor. On the second day, upper gastrointestinal endoscopy revealed a tumor

(Borrmann 4 type) extending from the stomach body to the greater curvature. The pathological diagnosis of the tumor was poorly differentiated adenocarcinoma of the stomach. With gastric cancer as the disease underlying DIC, chemotherapy could not be performed, because of a bleeding tendency. On the seventh day, low- molecular-weight heparin (dalteparin) was introduced for hypercoagulation secondary to DIC. On the eleventh day, he developed shortness of breath, orthopnea and sinus tachycardia (160 beats/minute), and chest auscultation revealed wheezing. Chest X-ray revealed pulmonary congestion. Electrocardiograph (ECG) showed paroxysmal supraventricular tachycardia (PSVT). Congestive heart failure and PSVT were diagnosed. His fluid replacement was reduced, and he was treated with furosemide, digitalis and verapamil. On the fourteenth day, he experienced dyspnea. His clinical course showed deterioration of consciousness with bradycardia and apnea followed by death on the fifteenth day. An autopsy was performed.

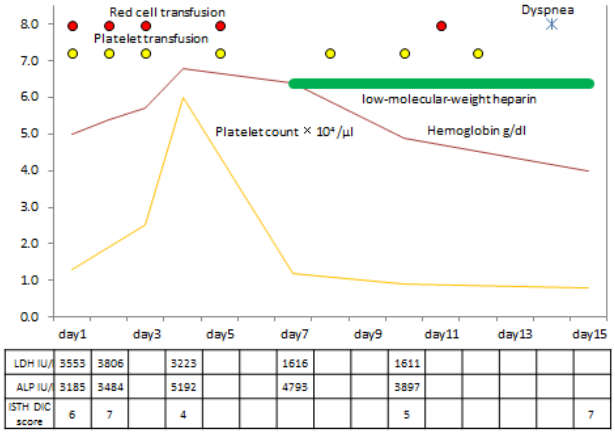

Figure 1. Clinical course after admission

The frequent red cell and platelet transfusion failed to keep enough hemoglobin levels and platelet counts. Although, on the seventh day, low- molecular-weight heparin was introduced, he experienced dyspnea supposed to be secondary to AMI on the fourteenth day. High levels of serum LDH and ALP suggest the existence of bone marrow carcinomatosis, which lead to DIC. High levels of ISTH DIC score suggest severe DIC.

ISTH DIC score: Score the test results: Platelet count (>100×10^9 /l = 0, <100×10^9 /l = 1, <50×10^9 /l = 2), Elevated fibrin marker (e.g. D-dimer, fibrin degradation products) (no increase = 0, moderate increase = 2, strong increase = 3), Prolonged PT (<3 sec = 0, >3 but <6 sec = 1, >6 sec = 2), Fibrinogen level (>1 g/l = 0, <1 g/l = 1), The International Society for Thrombosis and Haemostasis (ISTH), reference [3].

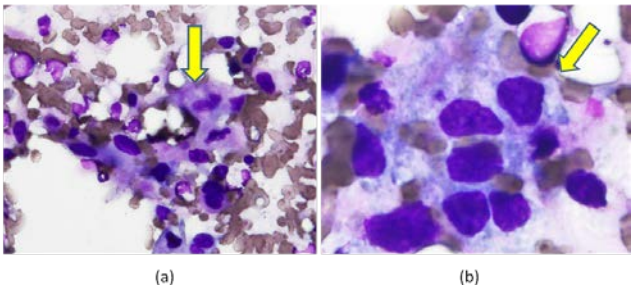

Figure 2. Microscopic findings of bone marrow liquid smear

Diffuse invasion of non-hematopoietic cells (arrow). Normal hematopoietic cells are rarely seen. (a) ×600, (b) ×1000 (May–Giemsa stain).

Table 1. Laboratory data on admission

WBC	13500	/μl	TP	7.1	g/dl	Peripheral blood smear		
Hb	5.0	g/dl	Tbil	1.8	mg/dl	Neu	60.0	%
Ht	14.8	%	AST	119	IU/l	Lym	24.5	%
MCV	87.6	fl	ALT	69	IU/l	Mon	4.0	%
MCH	29.6	pg	LDH	3553	IU/l	Eosino	4.0	%
MCHC	33.8	g/dl	ALP	3185	IU/l	Basophil	0.5	%
Plt	1.3×10^4	/μl	BUN	74.4	mg/dl	Blast	1.0	%
Ret	5.1	%	Cr	1.2	mg/dl	Myelo	7.5	%
PTINR	1.6		UA	11.4	mg/dl	Metamyelo	1.5	%
aPTT	35.8	sec	Amy	95	IU/l	Erythroblast	20.0	%
fibrinogen	106	mg/dl	Na	129	mEq/l			
D-dimer	29.8	μg/ml	Cl	92	mEq/l			
ATⅢ	124	%	K	5.4	mEq/l			
FDP	156.2	μg/ml	Ca	9.2	mg/dl			
			CRP	4.3	mg/dl			
			BS	265	mg/dl			
			ferritin	2497	ng/ml			
			CEA	247	ng/ml			
			CA19-9	43460	U/ml			

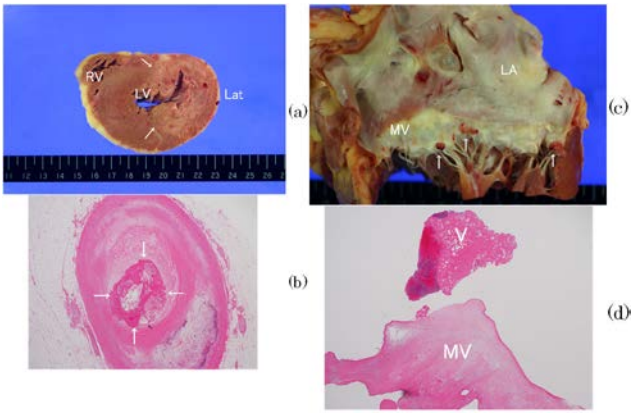

Figure 3. Pathological findings of the heart

(a) Transection of the papillary muscle attachment. Acute necrosis and fibrosis from lateral to anterior and posterior ventricular walls. Acute myocardial infarction (arrow), RV: right ventricle; LV: left ventricle; Lat: lateral ventricular wall.

(b) Microscopic findings: thromboembolism (arrow) in the right coronary artery (hematoxylin–eosin stain, ×20).

(c) Vegetation (arrow) on the mitral valve, LA: left atrium; MV: mitral valve.

(d) Microscopic findings: nonbacterial thrombogenic caditis on the mitral valve (hematoxylin–eosin stain, ×20), V: vegetation; MV: mitral valve.

The autopsy findings were as follows: myocardial infarction from the lateral to anterior and posterior ventricular walls [Figure 3 (a)], thromboembolism in the right coronary artery [Figure 3 (b)]. nonbacterial thrombogenic carditis of the mitral valve [Figure 3 (c-d)]. enlarged heart (470 g) with pericardial fluids (25 ml). The gastric tumor (Borrmann 4 type) was noted from the stomach body to the greater curvature, with the diameter of 24 cm. The histologic types were mostly poor differentiated adenocarcinoma and partly moderately differentiated tubular adenocarcinoma. The depth of cancer invasion was subserosa. Metastases were observed in lymph nodes of the left supraclavicular fossa, the

pulmonary hilum and the para-aortic region. Bone marrow carcinomatosis was detected, as a distal metastasis. The bone marrow was necrotic and fibrotic. Also, a phagocytosis of red cells and siderosis was observed in the bone marrow. Extramedullary hematopoiesis was observed in the spleen.

3. Discussion

Bone marrow carcinomatosis is a complication of cancer that often leads to disseminated intravascular coagulation (DIC) and microangiopathic hemolytic anemia (MAHA). Pathologically, bone marrow invasion of cancer impairs hematopoiesis and activates coagulation system. Subsequent formation of fibrin thrombi leads to DIC and systemic microvascular obstruction. The high shear rates of blood passing through these obstructions result in fragmented red blood cells, and lead to MAHA [1].

A solid tumor with bone marrow carcinomatosis is rare, and gastric cancer accounts for the majority of cases. The characteristics of gastric cancer with bone marrow invasion are as follows: young age, high levels of serum LDH, and ALP, and widespread bone invasion with osteolytic destruction. The histologic types of gastric cancer are signet cell carcinoma and poorly differentiated adenocarcinoma [2]. In the present case, autopsy and laboratory findings revealed typical findings of bone marrow carcinomatosis accompanied by DIC, and MAHA.

While thrombocytopenia and coagulation factor deficiency may lead to bleeding, fibrin thrombus may cause thromboembolism [3]. A significant proportion of patients with chronic DIC have nonbacterial thrombogenic carditis mainly involving the mitral and aortic valves. These heart lesions can be a source of arterial embolization, leading to infarction of the myocardium, kidneys, and brain [4].

Treatment of the underlying disease is a basic strategy for the treatment of DIC. Rhee et al. reported on 21 patients with gastric cancer with DIC; 18 (85.7%) had bone marrow metastasis, and 9 patients (42.9%) had the hemorrhagic complication of DIC. The most common factor influencing the decision to abandon palliative chemotherapy was uncontrolled bleeding [5]. Recommendations for the treatment of hypercoagulation secondary to DIC were described by Levi et al. In cases of DIC where thrombosis is predominat such as arterial or venous thromboembolism, severe purpura fulminans associated with acral ischemia, or vascular skin infarction, treatment with heparin should be considered. In cases associated with co-existing high risk of bleeding, it may be beneficial to use continuous infusion of unfractionated heparin [3]. In the present case, because of the bleeding tendency caused by DIC, chemotherapy for treating of the underlying disease was not introduced. Although low-molecular-weight-heparin was considered useful for the hypercoagulation in DIC, nonbacterial thrombogenic vegetation appeared on the mitral valve, resulting in AMI as a fatal thromboembolism.

Ueda et al. reported the incidence and pathologic features of cardiac lesions in 184 autopsied aged patients with DIC. Coronary thrombosis was noted in 31 (16.8%) patients, fresh myocardial necrosis in 60 (32.6%), and massive myocardial hemorrhage in 49 (26.6%). Fresh myocardial infarction was present in 16 (8.7%) patients, 13 of whom manifested coronary thrombosis. Only 3 of 16 patients with DIC and AMI had typical cardiac symptoms (chest pain, chest discomfort, dyspnea, and hypotension). In most patients, the ECG showed neither ST-segment elevation nor pathologic Q waves [6].

Reports of cases [7,8,9] accompanied by AMI due to DIC are shown in Table 2. In cases 2 and 3, the typical AMI symptoms were observed. ST-segment elevation on ECG was observed in cases 1 and 2. In the present case, AMI was presumed to have developed on the fourteenth day when dyspnea appeared. Because of the absence of chest pain, AMI was not considered as a differential diagnosis. The diagnosis of AMI was made by autopsy findings.

Table 2. Reports of cases accompanied by acute myocardial infarction caused by disseminated intravascular coagulation

case	age/ sex	predisposing condition of DIC	cardiac symptoms	ECG signs	reference
1	54/ female	sepsis	none	ST-segment elevation	7
2	71/ male	acute myeloid leukemia	tight chest pain	ST-segment elevation	8
3	40/ female	sepsis	substernal and epigastric pain	sinus tachycardia	9

Most cases of AMI develop secondary to arteriosclerosis. The coronary risk factors are as follows: hypertension, diabetic mellitus, hyperlipidemia, smoking, family history, and chronic kidney disease. The patient had two risk factors (hypertension and smoking). However, the autopsy findings did not demonstrate arteriosclerosis of the coronary arteries, and AMI was considered to be caused by DIC.

4. Conclusion

In cases accompanied by DIC, thrombus may develop in coronary arteries, resulting in AMI. The typical AMI symptoms and ST-segment elevation on ECG are often not observed. The importance of this phenomenon warrants consideration in clinical practice.

Acknowledgements

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Competing Interests

The author declares no competing interests.

List of Abbreviations

Disseminated intravascular coagulation: DIC; microangiopathic hemolytic anemia: MAHA; acute myocardial infarction: AMI; Eastern Cooperative Oncology Group: ECOG; lactate dehydrogenase: LDH; alkaline phosphatase: ALP; carcinoembryonic antigen: CEA; carbohydrate antigen 19-9: CA19-9; computed tomography: CT; Electrocardiograph: ECG; paroxysmal

supraventricular tachycardia: PSVT; The International Society for Thrombosis and Haemostasis: ISTH

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