Acute Brachial Artery Thrombosis: A Rare Complication of Diabetic Ketoacidosis

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Diabetes mellitus is a worldwide disease that leads to several acute complications including diabetic ketoacidosis, hyperosmolar hyperglycemia, and hypoglycemia. In addition, diabetes causes many chronic complications that lead to debilitation and diminished quality of life. Diabetic ketoacidosis is one of the serious acute complications; however, it is usually preceded by infection, acute myocardial infarction, stroke, or other dire events. Rarely does it accompany acute arterial thrombosis. Here, we report on a female patient who suffered from diabetic ketoacidosis combined with acute brachial artery thrombosis. After emergency treatment, including insulin therapy and surgical thrombectomy, the brachial artery was rescued and her prognosis was good.

Key Words: diabetes, ketoacidosis, brachial artery thrombosis
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Diabetes mellitus is a chronic and serious disease whose prevalence is increasing annually, especially with the popularity of the Western diet [1]. The disease causes many complications, one of the most serious and acute of which is diabetic ketoacidosis (DKA) [2]. Many conditions can predispose to DKA, including stroke, acute myocardial infarction, and, most commonly, infection [3].

Acute arterial thrombosis is a rare disease that requires immediate treatment. It has been reported in cases of coagulating factor deficiency, catheter implantation, trauma, anti-phospholipids syndrome, and systemic lupus erythematosus (SLE) [4–6]. This is the first case on which we have reported that combined both acute brachial artery thrombosis and DKA.

Case Presentation

This 33-year-old female had a 4-year history of diabetes, and she received regular outpatient follow-up for medical intervention. Her average fasting blood glucose was around 150 mg/dL. She experienced a painful sensation in the right forearm in the early morning of 3 October 1991, and was sent to our emergency room for help. On examination, her consciousness was clear, her left arm blood pressure was 159/114 mmHg, her body temperature was 37.0°C (98.6°F), and her respiratory rate was 17 breaths/minute. She experienced a severe painful sensation in her right arm, yet there was no wound, puncture lesion, or area of ecchymosis. The arm was cold, soft, cyanotic on palpation; there was no pitting edema. The blood pressure of the right arm was not measurable, while the right ankle blood pressure was 195/105 mmHg (Table 1). No pulsations of the right radial artery, right ulnar artery, or right brachial artery were palpable. The right axillary pulse was weak. There were strong pulsations of the right subclavicular and right carotid arteries. Therefore, there was a strong expectation of an acute thrombosis of the right brachial artery. Following serial confirmatory examinations, an emergency thrombectomy was performed. The coagulated bloody plaque was removed during thrombectomy.

Additional laboratory data collected in the emergency room revealed that her blood glucose was 625 mg/dL; urinalysis showed strong positive ketone bodies (3+). Blood osmolality was calculated at 308 mOsm/kg, and arterial

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Table 1. Four limbs blood pressure data in a female diabetic admitted with a painful right arm*

	Presurgical	Postsurgical
Right arm	—	143/77 mmHg
Right ankle	195/105 mmHg	170/95 mmHg
ABI on right	—	1.19†
Left arm	159/114 mmHg	145/95 mmHg
Left ankle	202/119 mmHg	160/100 mmHg
ABI on left	1.27	1.12

ABI = ankle–brachial pressure index.
*Blood pressure was measured manually, both before and after surgery using the same instrument.
†The postsurgical ABI was within the normal range.

blood gas analysis on room air revealed metabolic acidosis (pH 7.30, P_{O_2} = 97 mmHg, P_{CO_2} = 26.4 mmHg, HCO_3 = 13.2 mM/L). Therefore, DKA was also suspected.

The 12-lead electrocardiogram (EKG) showed myoischemic change during admission, and myocardial perfusion scintigraphy was compatible with myoischemic change over the left ventricular apex. Retinal photography was consistent with diabetic proliferative retinopathy. The albumin-to-creatinine ratio was 86.6 mg/mg, and diabetic nephropathy with microalbuminuria was considered. In other words, she had both diabetic macrovascular and microvascular complications.

Hypercoagulopathy was suspected because of the acute thrombosis event, so complements 3 and 4, prothrombin time, activated partial prothrombin time, anti-thrombin III, and protein C were checked, all of which were within normal limits. The antinuclear antibody and lupus anticoagulant antibody were also checked to rule out autoimmune diseases. All these data are shown in Table 2.

She received regular standard therapy for DKA and 1 day's heparin treatment after thrombectomy, followed by aspirin. The heparin use was to prevent postsurgical restenosis. After 10 days of therapy, her DKA resolved and her right forearm blood pressure returned, up to 143/77 mmHg. The right ankle–brachial index (ABI) was 1.19. She was followed up in our outpatient department for 2 more years while receiving treatment with oral antihyperglycemic agents and aspirin.

Table 2. Serum laboratory examination data

Item	Results	Normal range
Hemoglobin A_{1c} (%)	11	4–6
Prothrombin time (s)	12.4/11.5 (INR = 1.14)	10.5–14.5
Activated partial thromboplastin time (s)	37.5/31.9 (ratio = 1.76)	24–34
Complement 3 (mg/dL)	136.0	83.1–125.5
Complement 4 (mg/dL)	22.2	17.2–32.8
Anti-thrombin III (%)	128	85–118
Protein C (%)	73	80–132
Antinuclear antibody	Negative	Negative
VDRL test	No reaction	No reaction
White blood cell count (cells/μL)	6,620	4,000–10,000
Blood platelet count (platelets/μL)	242,000	130,000–500,000
C-reactive protein (μg/mL)	< 5	< 5
Aspartate transaminase (glutamic-oxaloacetic transaminase), (IU/L)	40	10–35
Alanine transaminase (glutamic-pyruvic transaminase), (IU/L)	40	10–40
Total cholesterol (mg/dL)	234	140–200
Triglycerides (mg/dL)	147	35–160
Low-density lipoprotein cholesterol (mg/dL)	165	0–100
High-density lipoprotein cholesterol (mg/dL)	30	29–85
Serum urea nitrogen (mg/dL)	10.5	5.0–25.0
Creatinine (mg/dL)	0.51	0.5–1.4
Glutamic acid decarboxylase autoantibodies (U/mL)	1.0	0–1.45

INR = international normalized ratio; VDRL = Venereal Disease Research Laboratory slide test

Discussion

To our knowledge, this is the first published report of DKA combined with an acute brachial artery thrombosis event. The mortality rate from DKA can be as high as 5% [2], but the mortality rate from DKA with thromboembolic complications can reach 20% [7]. The diagnosis of DKA is based on the clinical data. The key pathogenesis of DKA is insulin deficiency [8], which consists of hyperglycemia, acidemia, and ketonemia [9]. DKA always accompanies stressful conditions, therefore, many conditions can precipitate it. Infection, myocardial infarction, stroke, trauma, surgery, lack of insulin use, and poor compliance can all contribute to the development of DKA. In this rare distinct case, no precipitating condition was clinically found, except for poor control of blood glucose.

DKA causes many complications, including cerebral edema, pulmonary thromboembolism, cerebral venous thrombosis, and myocardial infarction. Batra et al [10] reported a case of combined DKA and acute myocardial infarction in a 12-year-old child.

DKA leads not only to hyperglycemia but also to dehydration caused by osmotic diuresis, which leads to hyperosmolarity and hypercoagulation. These may contribute to the acute thrombosis event. Keane et al [11] reported on a case of combined DKA and cerebral venous thrombosis in a 5-year-old child, with dehydration as the precipitating condition.

Brachial artery thrombosis is a rare coexisting condition. Coagulation deficiency, trauma, aneurysm, puncture site injury, catheter implantation, SLE, and even Raynaud's phenomenon should be considered as secondary causes [6]. Atherosclerosis is the leading cause of occlusive arterial disease of the extremities in patients older than 40 years of age [5]. Acute arterial occlusion results in the sudden cessation of blood flow to an extremity. There are two principal causes of acute arterial occlusion: embolism and thrombus *in situ*. The most common sources of arterial emboli are the heart, aorta, and large arteries; usually such patients have cardiac disorders including atrial fibrillation. Acute arterial thrombosis *in situ* occurs most frequently in atherosclerotic vessels or arterial bypass grafts [6]. Oral contraceptives, which have been considered a risk factor in thrombosis events, may also contribute to the artery thrombosis [12, 13]. However, our patient did not have these precipitating conditions. Hyperlipidemia may be related to arterial thrombosis, especially in association with myocardial infarction in children [14]. However, we found no report of a peripheral acute artery thrombosis associated with hyperlipidemia.

Diabetes causes many chronic complications in both large and small vessels. Large vessel diseases include peripheral artery occlusion and coronary artery disease. In this case, coronary artery disease was diagnosed based on 12-lead EKG and myocardial perfusion scan. Diabetic nephropathy and retinopathy were also recognized. Therefore, she had both large and small vessel diseases.

In this case, two conditions could have contributed to the development of acute artery thrombosis: dehydration secondary to hyperglycemia and hyperlipidemia. It is important to recognize all complications of DKA, and some of these may require emergency treatment. In this report, we demonstrate a case of DKA combined with acute right brachial artery thrombosis. After immediate surgical intervention and further treatment, the DKA resolved and her right arm was saved.

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急性肱動脈栓塞，糖尿病酮酸中毒罕見的併發症病例報告

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糖尿病是一個世界性的疾病，會引起相當多的急、慢性併發症，如酮酸中毒、高血糖症候群等等，會危及生命，降低生活的品質。其中，糖尿病酮酸中毒是一個常見、也是會危及生命的急性併發症。糖尿病酮酸中毒通常都是由一些原因引起，如敗血症，急性心肌梗塞等等，也會合併許多併發症如靜脈栓塞等等，它很少會合併急性動脈栓塞。我們在這裡提出一個罕見的病例，不但有糖尿病酮酸中毒，且合併急性右肱動脈阻塞。經過適當的胰島素治療，並立即開刀施行血塊清除術處理，病人的預後相當不錯，持續在門診追蹤治療。

關鍵詞：糖尿病，酮酸中毒，急性肱動脈阻塞，糖尿病急性併發症

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