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Diabetic Ketoacidosis and Thromboembolic Disease in Type 1 Diabetes: A Series of 5 Cases

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Abstract

Diabetic ketoacidosis (DKA) is a frequent complication that generally evolves favorably; however, thromboembolic complications may occur. Pulmonary embolism is a rare complication. We report a series of five cases of type 1 diabetic female patients presenting with DKA complicated by thromboembolic disease, along with a review of the literature.

Keywords: Thrombosis; Hypercoagulability; Prophylactic Anticoagulation; Pulmonary Embolism

Introduction

Diabetic ketoacidosis is a severe complication that occurs mainly in patients with type 1 diabetes. It is characterized by the accumulation of ketone bodies, fluid depletion, and electrolyte disturbances [1, 2].

Thromboembolic disease (TED), which includes deep vein thrombosis (DVT) and/or pulmonary embolism (PE), is the third most frequently diagnosed cardiovascular disorder worldwide [3]. It is rare in children and adolescents [3]. Thrombi generally originate in the deep venous system of the lower limbs, but can also occur in the upper limbs, pelvic veins, renal veins, or the right heart [4].

The causes of thromboembolic disease are often multifactorial and may include surgical interventions, recent trauma, immobility and venous stasis, obesity, pregnancy and postpartum, malignancies (paraneoplastic syndrome), hereditary hypercoagulability, oral contraceptives, and smoking [3, 5]. However, the relationship between DKA and thromboembolic disease remains debated.

To document this rare complication, we report a series of 5 cases of type 1 diabetic female patients who developed DKA complicated by thromboembolic disease, along with a review of the literature.

Materials and Methods

This retrospective study was conducted at the Ibn Sina University Hospital Center in Rabat over 5 years (2018–2022).

Personal data were collected for each patient: age, sex, medical history, duration of diabetes, cardiovascular risk factors, the interval between DKA and TED onset, body mass index, bicarbonate levels (HCO_3-), and thrombophilia workup.

Results

Among the five cases, the mean age was 31.6 years, and all patients were female. The mean duration of diabetes was 4.2 years (range: 0–9 years). None of the patients had notable cardiovascular risk factors. Cardiovascular risk assessment indicated a moderate level for all patients according to the 2021 European Society of Cardiology recommendations.

Four patients presented with severe DKA (HCO_3- < 5 mEq/L), and one patient had moderate to severe DKA (HCO_3- = 9 mEq/L).

Causes of DKA included insulin therapy interruption in 3 cases, inaugural DKA in 2 cases, and urinary tract infection in 1 case (associated with voluntary insulin discontinuation). All patients had a normal body mass index.

The mean delay between DKA onset and thromboembolic disease was 2.6 days.

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Table 1: Summary of Patient Data.

Case	Age	Sex	Duration of diabetes	DKA severity	Delay between DKA and TED	Type of TED
1	16	F	5 years	Severe (HCO_3- < 5 mEq/L)	2 days	PE
2	24	F	7 years	Severe (HCO_3- < 5 mEq/L)	2 days	PE
3	64	F	9 years	Moderate to severe (HCO_3- = 9 mEq/L)	3 days	PE
4	28	F	Newly diagnosed	Severe (HCO_3- < 5 mEq/L)	3 days	PE
5	26	F	Newly diagnosed	Severe (HCO_3- < 5 mEq/L)	3 days	Thrombosis of the common and external iliac veins

Pulmonary embolism was identified in four patients, while one patient had thrombosis of the common and external iliac veins. No evident underlying cause of thrombosis was found, and thrombophilia tests were negative. DKA itself was considered the main trigger.

Therapeutic management included rehydration, correction of electrolyte disorders, intravenous insulin therapy by bolus, and curative anticoagulation with heparin, later switched to vitamin K antagonists. Clinical and biological outcomes were favorable in all five cases.

Discussion

Diabetes is a hypercoagulable state marked by endothelial dysfunction, altered coagulation and fibrinolysis, chronic platelet hyperactivity, leukocyte activation, low-grade inflammation, and microparticle activation. This promotes a prothrombotic state contributing to vascular and thromboembolic complications in diabetic patients [6].

Thromboembolic disease may develop even in diabetic patients without vascular complications, as observed in our cases. Subclinical endothelial damage, hypofibrinolysis, and platelet aggregation are major contributors to coagulation activation [7, 8].

Acute hyperglycemia promotes coagulation through increased levels of factors VII and VIII and tissue factor pathway inhibitor (TFPI) [7]. The prothrombotic state seen in DKA is explained by abnormal platelet behavior, coagulation activation [9, 10], endothelial activation [11], and a reduction in natural anticoagulants. A global activation of the fibrinolytic system also occurs during DKA [11].

Additionally, severe dehydration associated with DKA contributes to hypercoagulability by increasing red blood cell rigidity and blood viscosity [12].

Treatment of DKA restores protein C activity and von Willebrand factor levels [11]. Thromboprophylaxis is not yet officially recommended for DKA management, although some centers use it in cases of DKA associated with hyperosmolar hyperglycemia [13].

Conclusion

Diabetes is a hypercoagulable condition that predisposes patients to thromboembolic disease, and this risk is further increased during DKA, which may induce additional hemostatic abnormalities.

Based on the literature and our five observations, patients presenting with DKA—particularly severe forms (HCO_3- < 5)—should be considered at high risk of thromboembolic events, suggesting that preventive anticoagulation may be warranted to avoid potentially fatal complications.

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