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CEREBRAL EDEMA SECONDARY TO DIABETIC KETOACIDOSIS: A DEVASTATING COMPLICATION

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INTRODUCTION

Diabetic ketoacidosis (DKA) is characterized by metabolic acidosis, hyperglycemia, and ketonemia/ketonuria. This is a metabolic emergency, commonly associated with type 1 Diabetes Mellitus, triggered, in most cases, by infections and poor adherence to medication treatment. The signs and symptoms of DKA are generally: diffuse abdominal pain, nausea and vomiting, dehydration and hyperventilation. Furthermore, depending on its severity and the time required for diagnosis, DKA can develop into potentially fatal complications, including the most devastating: cerebral edema.

OBJECTIVES

Present general aspects and evidence on the relationship between CAD and cerebral edema.

METHODOLOGY

This is a literature review whose search for evidence occurred through the UpToDate and PubMed databases. Furthermore, consultations were carried out on the electronic portal of the Brazilian Diabetes Society.

RESULTS

Cerebral edema secondary to DKA is an uncommon complication, more common in children than adults. Although considered atypical, this condition has a high mortality rate and risk of permanent neurological sequelae. A retrospective observational study of adults with DKA and hypersomolar hyperglycemic syndrome found that the mortality rate was more than 30 times higher in the group with cerebral edema than in

the group without cerebral edema. Clinical manifestations commonly include: altered level of consciousness, papilledema, headache and cranial nerve palsy, and generally occur within 12 hours of starting DKA treatment. However, symptoms may be present before starting treatment. The pathophysiological mechanism of cerebral edema is not fully understood. For some time, theories about osmotic changes were proposed as responsible for the development of cerebral edema. However, current evidence points to changes in cerebral blood flow and neuroinflammatory responses as factors directly related to the pathogenesis of brain injury. Furthermore, studies have shown that individuals with high levels of urea nitrogen, hypocapnia and severe acidosis are more prone to this neurological complication. It is worth noting that cerebral edema related to DKA does not have a specific treatment, and the therapy used in this condition is generally mannitol and hypertonic solution.

CONCLUSION

Cerebral edema secondary to DKA is an acute complication and neurological emergency. Given such severity, early diagnosis and adequate management of the condition are essential. Furthermore, further research is necessary to better understand the pathophysiology of this condition.

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