

EDITORIALS

Diabetic ketoacidosis at the onset of type 1 diabetes

Is still common, despite the consistency of predictive factors worldwide

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Despite major advances in the care of diabetes, diabetic ketoacidosis remains a leading cause of morbidity, mortality, and hospital admission in children and adolescents with type 1 diabetes.¹ Deaths are mainly caused by cerebral oedema, which is more common in patients who are younger, have new onset diabetes, or have a longer duration of symptoms.^{1 2} Other possible causes of morbidity and mortality associated with diabetic ketoacidosis include hypokalaemia, pulmonary oedema, cerebral thrombosis or infarction, and rhabdomyolysis.¹⁻³ Given these life threatening complications and the healthcare costs associated with hospital admission, prevention of diabetic ketoacidosis should be the primary goal for clinicians.

In the linked systematic review (doi:10.1136/bmj.d4092), Usher-Smith and colleagues looked at 46 studies of more than 24 000 children from 31 countries to identify factors associated with the presence of diabetic ketoacidosis at diagnosis of diabetes in children and adolescents.⁴ The studies span the late 1980s to 2008. The authors report a high prevalence of diabetic ketoacidosis—between 21.6% and 53.8% in Europe and North America, and 68.2% in Saudi Arabia—similar to that seen in the mid-1990s.¹ In some countries, such as the United Kingdom, the prevalence of diabetic ketoacidosis remained the same—at 27%—before (1985-96) and after (2004-7) the turn of the 21st century.⁴ In a study from Germany and Austria, the prevalence of ketoacidosis at the onset of diabetes also stayed the same throughout the 13 year study period (1995 to 2007).⁵ This is surprising because, with the rising incidence of type 1 diabetes (mean increase of 3-5% a year), especially in young children,⁶ greater awareness of the disease should have resulted in a decrease in the prevalence of ketoacidosis at the onset of diabetes.

The review also shows similarities in predictors of diabetic ketoacidosis across nations and cultures: younger age, misdiagnosis, delayed diagnosis, certain minority ethnic groups, and lack of health insurance were consistently associated with diabetic ketoacidosis. Protective factors that decreased the risk of diabetic ketoacidosis include having a first degree relative with type 1 diabetes, higher parental education, and higher background incidence of type 1 diabetes, all of which are explained by education, recognition, or having experience with diabetes.

Improvements in public and professional awareness of risk factors for and symptoms of diabetes could help prevent diabetic ketoacidosis. This is achievable, as shown by the community and physician awareness campaign in Italy, which reduced the rates of diabetic ketoacidosis from 78% to almost zero two years after the campaign began,⁷ and remained effective eight years later.⁸

The findings from this study should heighten the awareness of clinicians caring for children and adolescents. Continuing education should be provided on the presentation of diabetes. Questions about polyuria and enuresis should always be part of the review of systems during history taking in visits to primary care. Dipstick testing of urine for glucose and ketones should be done routinely in any sick child, especially those under 6 years, whether or not the cause of sickness is known. Blood ketones can be measured at the point of care in clinics or urgent care settings to detect ketonaemia when patients cannot provide urine specimens. To increase public awareness, information about diabetes in children should be given to teachers and to the public, possibly using poster displays at schools and paediatricians' offices, as was done in the Italian campaign,⁷ or electronically through social media networks.

Other benefits of avoiding ketoacidosis at diagnosis should be emphasised to reinforce preventive efforts. Compared with children with ketoacidosis at diagnosis, those without it have significantly better residual β cell function, which results in better glycaemic control and a higher rate of partial remission.⁹⁻¹¹ Several studies have reported an association between more severe ketoacidosis at diagnosis, greater loss of β cell function, and younger age at diagnosis.^{9 11} Delayed diagnosis or difficulty in recognising diabetes in this age group therefore is not the only challenge—it also seems that a more aggressive form of the disease occurs in younger children, whereby rapid β cell destruction speeds up the progression to diabetic ketoacidosis. The higher titres of insulin autoantibodies and islet cell antibodies seen in younger children support the concept of a more aggressive form of diabetes.^{11 12} These differences in pathogenesis of type 1 diabetes in different age groups warrant future study and may provide insights on age specific interventions to decrease the prevalence of ketoacidosis at diagnosis and to preserve residual β cell function.

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