Abdominal pain, leucocytosis, and abnormal enzymes in DKA

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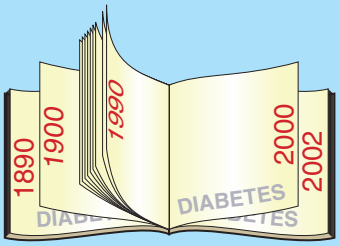
Today's diabetes world is fast-moving and exciting; knowledge is accumulating at an astonishing rate, new discoveries and understanding lead to new ideas and innovations in treating, managing and preventing diabetes. However, there's nothing new under the sun. To help understand the present, it sometimes helps to examine the past.



Tattersall's Tales will enable readers to do just that. In every issue, Robert Tattersall, renowned diabetes sage and guru, will consider an aspect of diabetes and place it in a suitable historical context. Research, treatment, people and products will all feature.

In this instalment, Robert Tattersall discusses diabetic ketoacidosis (DKA) and valuable lessons found in the literature. The moral is not to do unnecessary tests in people with DKA.

When looking at the laboratory results of a patient with diabetic ketoacidosis (DKA), junior doctors (and sometimes even senior doctors) may order a raft of unnecessary secondary investigations when they see a raised white blood cell count, serum amylase or abnormal liver function tests. Yet, in the case of the raised white blood cell count, it has been known for the best part of 80 years that this is part and parcel of severe DKA.

From 1900–30 there was a protracted debate in the US about the value of the white blood cell count as a diagnostic aid in acute appendicitis. The main opponent was the surgeon, John Deaver, who regarded a blood count as 'pernicious and dangerous' claiming that the degree of abdominal tenderness assessed by the experienced surgical hand was the best guide to the need for operation. That leucocytosis did not necessarily indicate appendicitis in DKA was first reported by Joslin et al in 1925. One of their patients had a white blood cell count of 44 000 which fell overnight to 13 000 with rehydration and insulin. A dramatic case was reported by Frank Allan of the Mayo clinic in 1927. A 9-year-old girl who had had diabetes for 2 years was diagnosed by her GP as having appendicitis, was sent on an overnight train journey to the Mayo clinic and unfortunately did not get her evening insulin. On arrival in Rochester, she was critically ill with a white blood cell count of 66 400 (90% polymorphs). The extreme leucocytosis suggested general peritonitis and, as operation was thought too risky, she was treated medically. The next morning she had recovered and her white blood cell count had fallen to 20 200 (Allan, 1927).

In the 1930s, several writers commented on the abdominal symptoms in DKA. In 1936, Baker wrote that:

'...to operate in the presence of uncomplicated diabetes is to expose a seriously ill patient to an unnecessary hazard, yet to disregard this triad of symptoms (abdominal pain, tenderness and vomiting) is to invite disaster occasionally by neglecting a gangrenous appendix which may be causing the acidosis.'

This was not an uncommon problem; 25 of Baker's 108 cases had abdominal pain, vomiting and leucocytosis of more than 12 000, but only three had a surgical cause (Howell, 1995). In a series of 114 cases of DKA reported by Beardwood in 1935, 75% had one or more abdominal symptom. Furthermore, 86% of those with abdominal symptoms had leucocytosis and 79% had a fever which was surprising since hypothermia is common in DKA. One of the many times this issue has been re-examined was in 1975 when Ian Campbell et al found that abdominal pain was sufficiently prominent in 46 of 211 episodes of DKA to cause a diagnostic problem. It was explained by concomitant illness in 17 episodes but was due to pure DKA in 29 episodes.

A possible cause of abdominal pain in DKA is pancreatitis. Here again laboratory tests may mislead. Finn and Cope (1963) found that the serum amylase was raised in 8 of 11 cases of severe DKA. The pancreas was normal in all 5 patients with a raised amylase who had an autopsy. The problem was re-examined in 1973 by Tony Knight and colleagues who found the amylase raised (often grossly) in 60% of 35 consecutive episodes of DKA. All those with gross rises survived and the person with the highest level had no abdominal pain. Other serum enzymes such as creatine phosphokinase (CPK) and transaminases may be raised in DKA and have, in the past, led to erroneous diagnoses of myocardial infarction (Velez-Garcia et al, 1966).

The moral is do not do unnecessary tests in patients with DKA, or any other illness for that matter!

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