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Lesson of the week

Spurious hyperglycaemia and icodextrin in peritoneal dialysis fluid

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Metabolites of new peritoneal dialysis fluids may cause spurious hyperglycaemia and inappropriate insulin treatment

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Diabetes mellitus, in particular type 2, has become more common, and the trend is likely to continue.[1] Associated comorbidity is also more common—for example, diabetes is now the most common cause of dialysis dependent renal failure in the Western world.[2] In the United Kingdom between 1991 and 1998, the incidence of new patients on dialysis increased from 67 to more than 90 patients per million population, and the prevalence of diabetes in people receiving dialysis has increased from 16% to 19%.[3]

The increasing demand for dialysis and slower growth in capacity for haemodialysis has reinforced the need for an integrated approach to providing dialysis. Peritoneal dialysis is the preferred option for a proportion of patients with end stage renal failure.[4] A subgroup of patients has difficulties with removing fluid. This can be improved with an alternative osmotic agent based on a polymer of glucose—icodextrin.[5] We report a severe potentially clinical consequence of using icodextrin in a diabetic patient, which although mentioned in a specialist journal is still not widely recognised. This issue is even more important given the increasing number of diabetic patients with end stage renal failure. About 500 patients in the United Kingdom use icodextrin daily.

Case report

A 76 year old Sudanese man presented to the emergency department with a four day history of general malaise and shortness of breath. On the day of presentation he developed a cough, which produced white sputum. He had type 2 diabetes controlled with insulin and control of his diabetes had been erratic in the few days before admission. He had been using peritoneal dialysis for four years with icodextrin for the previous year to help maintain ultrafiltration.

In the emergency department the man seemed comfortable at rest but was feverish with a temperature of 37.2°C. His pulse was 85 beats/min and blood pressure 160/80 mm Hg. Oxygen saturation was 94% on air. He had a raised jugular venous pressure and heard crackles at the base of both lungs. A chest x ray showed interstitial shadowing but no focal consolidation. The finger stick glucose reading was 17 mmol/l.

The team diagnosed him as having chest infection and transferred him to a sister hospital. During transfer the patient's consciousness decreased: he became sweaty and developed slurred speech. On arrival at the new hospital, the patient had a grand mal seizure. Finger stick glucose testing gave a reading of 15.4 mmol/l. He was given 5 mg diazepam and the fit subsided. Soon after, laboratory blood tests found that venous glucose concentration was only 1.2 mmol/l. On treatment with intravenous glucose the patient recovered.

The admitting doctors started antibiotics and insulin using a sliding scale. Two hours later, the patient had another grand mal seizure and they gave further bolus of diazepam. The glucose finger stick reading had increased again, to 14 mmol/l, but venous glucose concentration was 1.5 mmol/l. They gave further intravenous glucose and the patient recovered. A sample of blood on test sticks from two different machines gave readings of 15.6 and 16.8 mmol/l. A laboratory sample at the same time, however, gave 5.5 mmol/l. The sliding scale was stopped, and the patient was transferred to the renal unit, where he recovered uneventfully.

Discussion

Peritoneal dialysis fluid usually contains glucose as an osmotic agent to enable water to pass across the peritoneum. Some patients lose the osmotic effect of glucose quickly, but large icodextrin molecules, which are not easily transported across the peritoneal

membrane, maintain an osmotic gradient. This prolongs ultrafiltration.

Icodextrin is not metabolised in the peritoneal cavity, but the polymer can move into the blood stream via the lymphatic system.[6] During systemic circulation, icodextrin is mainly metabolised into maltose, consisting of two glucose molecules, which accumulates due to a lack of circulating maltase. It is the accumulation in the systemic circulation of these metabolites of icodextrin that may lead to the disparity between finger stick and formal blood glucose measurement.

Maltose interferes with glucose assays that use glucose dehydrogenase with coenzyme pyrroloquinolinequinone (GDH PQQ) leading to falsely increased readings. Some machines use this system. Each saccharide chain generated by the metabolism of icodextrin has a free reducing group of glucose located at its end. This can react with the glucose dehydrogenase with coenzyme pyrroloquinolinequinone in the test kit to produce a falsely increased glucose reading.

This method of measuring glucose concentrations can significantly overestimate glycaemia by 3.6 ± 1.4 mmol/l compared with reference values ($P<0.01$).[7] A correction factor cannot be used because overestimation varies widely. Maltose interferes much less in systems which use glucose oxidase or hexokinse. The disparity between the glucose concentrations we found by finger stick measurements and those found in the laboratory are greater than have been found previously.

Our patient was using a system based on glucose oxidase at home with no problems, but the trust changed all finger stick test kits on wards to ones based on glucose dehydrogenase. The literature supplied with these machines mentions potential interference by other reductants. Although the effect of icodextrin on glucose testing has been reported before, both consultants and specialist registrars in diabetes and nephrology seem to be largely unaware of this potentially life threatening problem.[7 8] Careful attention needs to be paid to any change in glucose testing equipment used in these patients to minimise the risk of this problem occurring. Also, when patients start using this type of dialysis fluid, the method by which blood sugar is estimated should be checked. Icodextrin has other less serious laboratory effects, which are reviewed elsewhere.[6]

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A memorable patient

Cyril

Cyril was the last patient at the end of a busy clinic in Leicester, and he came into my room with his daughter. He was an octogenarian with a smart appearance, an upright stance, and a military looking moustache. I just happened to say "Do I detect a slight Welsh accent?" and the response to this casual question has led to three years of an association that has given us both much unexpected pleasure.

He told me that he was originally from Shropshire but had fought during the second world war with the 4th Battalion, The Welch Regiment. My father, who died 30 years ago, had been a colonel of that regiment, and it transpired that Cyril had served with him for seven years from 1939. Initially, they spent some years training with the regiment in Northern Ireland, and in 1944 they took part in the Normandy invasion, losing many of their comrades during that time, and they remained together until 1946. He had married an Irish girl called Pearl while in Ireland, and his daughter and I discovered that we had both been born in Banbridge during that time.

Since that day we have met regularly. Cyril presented me with an account he had written several years ago of his time in the regiment. It contains many anecdotes, some including my father and godfather, and Cyril's testimonial at the end was written and signed by my father, who promoted him to company sergeant major, a post that did not usually go to territorial soldiers. My father never talked about his wartime experiences, and so it has meant a lot to me to listen to so many stories and look at the old military photographs.

The climax of our friendship was a two week trip my husband and I made to France, Belgium, Holland, and Germany this spring. We followed the route taken by Cyril and my father in 1944-5, tracing all the villages and sites of heavy fighting. We compared their appearance now with that in the many old wartime photographs, reading the details of what happened in a regimental book that Cyril lent me and in his own memoirs. It was a very exciting and also sobering experience, begun on the same day as the start of the war with Iraq.

It was a great sadness that I met Cyril because he was losing his sight. I have been so impressed to see the way he has accepted this, with the wonderful help and support of his wife, Pearl. Self pity is not part of his vocabulary. His son now lives in Germany and has a home in France, and I notice with interest that Cyril now has far more time and respect for the Germans than the French.

I continue to be so grateful for this chance meeting, at the end of my career, with the patient who would have to be my most memorable.

Penny Harding *retired associate specialist in ophthalmology, Leicester Royal Infirmary*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. Please submit the article on http://submit.bmj.com Permission is needed from the patient or a relative if an identifiable patient is referred to.