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Case Report

Hyperlactatemia Due to Thiamine Deficiency in a Peritoneal Dialysis Patient

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Abstract

Although prevalent in clinical practice, hyperlactatemia and lactic acidosis occasionally present a diagnostic challenge and are frequently linked to adverse clinical outcomes, particularly in the vulnerable dialysis population. While tissue hypoperfusion states typically lead to elevated lactate levels, they can also be an indication of a range of other conditions. Long-term usage of lactate-containing peritoneal dialysis solutions as a standard of care has not been linked to lactic acidosis. Although one of the known causes of lactic acidosis is thiamine deficiency, this is frequently disregarded, especially in people without obvious underlying predisposing conditions. Intriguingly, thiamine deficiency appears to affect the dialysis population more frequently than one might anticipate for the various reasons mentioned in the discussion. Here, we describe a rare case of hyperlactatemia caused by thiamine deficiency in a peritoneal dialysis patient and emphasize the significance of considering thiamine deficiency as a differential diagnosis, particularly in the peritoneal dialysis population.

Keywords: Hyperlactatemia; Lactic acidosis; Peritoneal dialysis; Thiamine deficiency

Introduction

Hyperlactatemia is described as an elevated lactate level of 2 to 5 mmol/L, whereas lactic acidosis is generally defined as a lactate level of 5 mmol/L or higher [1]. Although hyperlactatemia/lactic acidosis is a prominent issue in clinical practice, the etiologies can have a wide variety of differentials and are frequently linked to adverse clinical outcomes [2]. Serum lactate concentration is an indication of the balance between production and clearance [2]. While decreased tissue oxygenation results in anaerobic metabolism and increased lactate production which frequently causes lactic acidosis, it can also occur in non-hypoxic diseases that cause delayed clearance, such as liver disease, alcoholism, and thiamine deficiency [2]. When lactic acidosis coexists with sepsis or low-flow situations, mortality is raised by a factor of almost three, and the severity of the outcome worsens with increasing lactate levels [3,4]. Metformin uses, particularly in individuals with renal failure, and ingestion of toxic alcohols such as methanol or ethylene glycol are additional causes [5]. We present a case of persistently elevated serum lactate levels in a patient receiving Peritoneal Dialysis (PD), raising questions about the potential role of lactate from PD fluid in the development of elevated lactate.

Previously, this article was presented as a conference abstract at the ASN Kidney Week on Nov 06, 2015, in San Diego, California.

Case Description

A 44-year-old woman who was undergoing peritoneal dialysis for the end-stage renal disease was admitted with generalized weakness and poor oral intake. Her test results showed leukocytosis, metabolic acidosis (CO2 17 MM/L), an elevated anion gap (19 mEq/L), and a lactate level of 6 (normal 0.9-1.7 MM/L). She also had hypotension and tachycardia. Fungal peritonitis was discovered during further testing, and it was treated with fluconazole. The patient declined to have the PD catheter removed as a standard protocol for fungal peritonitis but agreed to exchange the PD catheter. Fortunately, subsequent cultures showed resolved fungal infection. Although the illness cleared up and the blood pressure improved, the serum lactate levels remained elevated (4-6 MM/L). As there was no clinical evidence of hypoxic etiology, we looked at non-hypoxic causes for elevated lactic acid levels. Apart from a low level of serum albumin, her liver function

tests were normal. Thiamine, which is essential for the metabolism of lactate and glucose, was found to be low at 59 nmol/L (normal range: 78–185 nmol/L) and pyruvate levels elevated at 2.08 mg/dL (normal range: 0.3–1.5 mg/dL), even though the patient denied ever consuming alcohol. After beginning treatment with 50 mg of oral thiamine per day, the serum lactate level soon returned to normal, reaching 1.6 MM/L (Figure 1).

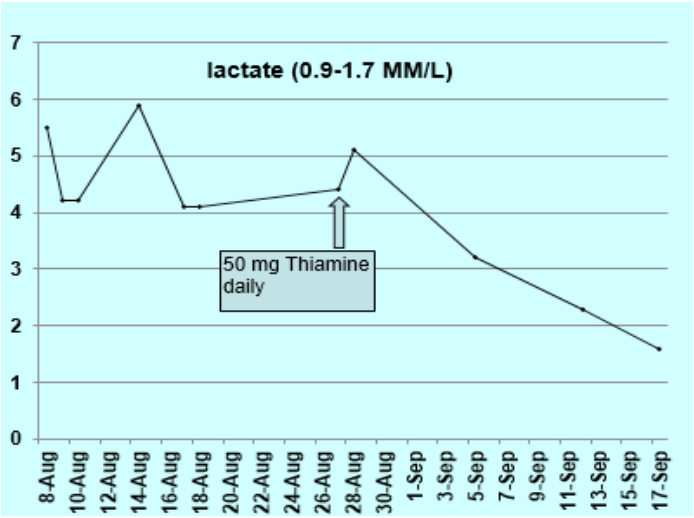

Figure 1: Serum lactate levels (MM/L) before and after supplementation of oral thiamine 50mg.

Discussions

To treat metabolic acidosis, bases such as lactate and bicarbonate are added to PD fluids. Studies have demonstrated that lactate does not accumulate even when using a higher volume of PD fluid than what was given to our patient (4 exchanges of 1.5% dextrose solution over 24 hours with a 1 L fill volume) [6-8]. Like in our patient's case, where she was also discovered to have peritonitis, an unwanted side effect of peritoneal dialysis [9,10], which can also cause sepsis and lactic acidosis, the diagnosis of the etiology of lactic acidosis can be difficult at times. Sepsis as the underlying cause was ruled out since lactatemia persisted even after the infection had cleared up.

In the Krebs cycle, thiamine functions as an enzyme cofactor for pyruvate dehydrogenase and alpha-ketoglutarate dehydrogenase [11]. Pyruvate builds up in thiamine deficiency and is converted into lactate-by-lactate dehydrogenase (Figure 2). The most frequent causes of thiamine deficiency in the general population are inadequate intake and chronic alcoholism, which are followed by pregnancy and parenteral feeding [12]. Because thiamine is essential for carbohydrate metabolism, the carbohydrate load from PD fluid leads to increased thiamine metabolic demands. Few studies have previously examined changes in thiamine levels in individuals with end-stage renal disease receiving maintenance dialysis therapy [13]. Poor oral intake, dialysis clearance, and the presence of uremic toxins were found to be the causes for thiamine deficiency [13].

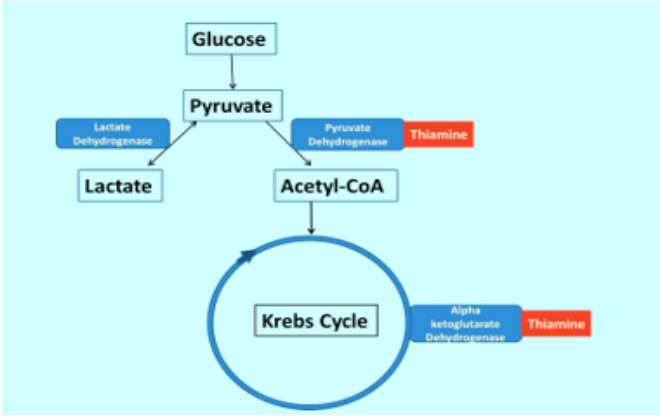

Figure 2: Thiamine deficiency leads to accumulation of lactate.

Thiamine has a relatively low molecular weight (266.4 Da), which makes it more dialyzable [14-16]. While data on PD patients is scarce, the research found that the mean reduction in thiamine diphosphate (TDP) concentration following hemodialysis was 43.9% (95% CI 34.9-52.9). Elevated pyruvate levels and the temporal relationship between thiamine supplementation and lactate normalization strongly support thiamine deficiency as the cause of this patient's elevated lactate level. Although thiamine deficiency-related lactic acidosis in the context of parenteral nutrition has been documented, this case is, to our knowledge, the only instance of thiamine deficiency-related hyperlactatemia in the context of PD.

Conclusion

To our knowledge, our case is the only illustration of hyperlactatemia secondary to thiamine deficiency in a peritoneal dialysis patient. It seems unlikely that the higher serum lactate levels are caused by the lactate in PD fluid. It's crucial to rule out any other potential diagnoses, notably sepsis like the patient in this case. Thiamine deficiency has been discovered to be more common than one would anticipate in the dialysis population for several reasons that were mentioned in the discussion. We stress the significance of incorporating thiamine deficiency as one of the primary differential diagnoses, particularly in the peritoneal dialysis population, as this helps to prevent delays in diagnosis and management.

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