Case report:

Hypocalcaemia in a Patient with Chronic Liver Disease: A Case Report

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Abstract:

Acute hypocalcaemia is an uncommon presentation in clinical practice. However, severe acute hypocalcaemia may require immediate resuscitative measures. Thus, the case presented is aimed to highlight the importance of thorough assessment and prompt management in replacing the calcium. This case looks at a 34-year-old woman with underlying chronic liver disease (primary biliary cirrhosis) and hypoalbuminemia presented with symptom of perioral and peripheral paraesthesia for three days and was then infused with slow intravenous calcium gluconate. In avoiding recurrence, she was prescribed daily oral supplementation of calcium carbonate.

Keywords: Hypocalcaemia, chronic liver disease, case study, management.

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Introduction

Hypocalcaemia is an uncommon finding in routine electrocardiogram (ECG). There are many causes including hypomagnesaemia, hypalbuminaemia, vitamin D deficiency, medication or surgical side effects, hypophosphatemia, or PTH deficiency among others[1]. Hypocalcaemia is a common biochemical abnormality that may present as a spectrum from asymptomatic laboratory findings to acute life-threatening crisis. The clinical manifestations of hypocalcaemiadepend on the level of ionized calcium[2]. Most common symptoms of hypocalcaemia are neuromuscular manifestations that include muscle spasms, cramps, tetany, paraesthesia and circumoral numbness[3]. Patients may also present with cardiac features such as electrocardiographic (ECG) changes and cardiac impairment[2].

The normal concentration of serum calcium ranges between 8.9-10.1 mg/dL, whereby the normal range of ionized calcium is 4.65 to 5.25 mg/Dl[4]. Calcium concentration level below this is considered as hypocalcaemia. Interestingly, chronic hypocalcaemia can occur and may produce dermatologic manifestations such as coarse hair, brittle nails, xerosis and cataracts[1].

This case will look at a young woman with chronic liver disease with a history of acute hypocalcaemia with classical ECG changes. She was treated with intravenous calcium gluconate and responded well with it.

Case summary

A 34-year-old woman with underlying chronic liver disease (primary biliary cirrhosis) and hypoalbuminemia presented to the emergency department, andcomplained of numbness and tingling sensations in the perioral area and peripheries for three days. This was associated with muscular ache, over the back and lower legs. This was her first presentation with these symptoms. There was an absence of other neurological symptoms.

On theexamination, her blood pressure was 130/80 mmHg, pulse rate 86 beats per minute, temperature 37^{o}C and oxygen saturation 98%. Her physical examination was unremarkable with no presence of Chvostek or Trousseau's sign.

Laboratory tests were sent; of most significant was the lowered serum albumin level of 3.5 g/dL and serum calcium level 7.5 mg/dL. The corrected

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calcium level was 7.9 mg/ dL which is below the reference range of 8.9-10.1 mg/ dL.

An electrocardiogram (ECG) was done to consolidate the diagnosis of hypocalcaemia. ECG (Figure 1) showed the classical changes of QT interval prolongation. As a result, a diagnosis of acute hypocalcaemia was made and patient was treated with slow intravenous infusion of calcium gluconate over 1 hour. ECG repeated an hour later showed sinus rhythm (Figure 2). Meanwhile serum calcium repeated two hours later showed a normal reading of 9.1 mg/dL.

To avoid recurrence of this problem, patient was prescribed oral supplementation of calcium carbonate 500mg daily. She was seenagain in two weeks which once again showed a normal ECG and serum calcium level of 9.3 mg/ dL. She was advised to come back urgently ifshe experienced any symptoms of hypocalcaemia.

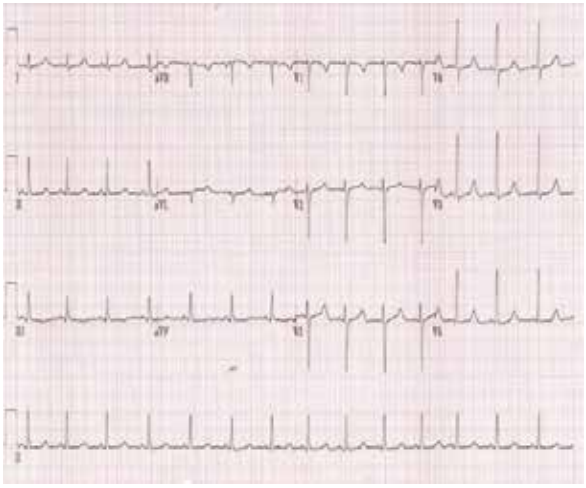

Figure 1: Iinitial ECG with QT prolongation

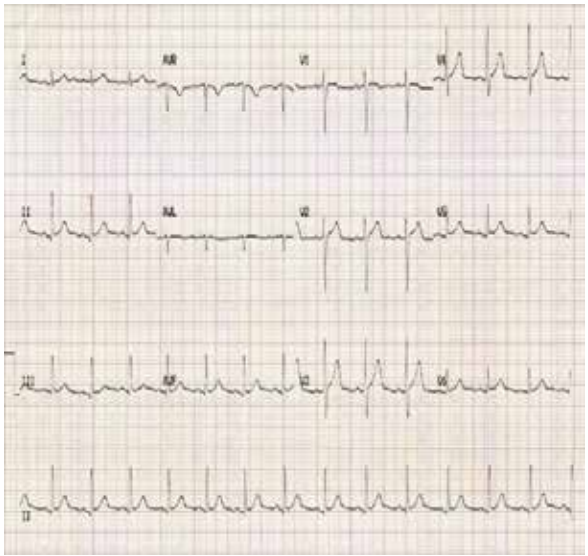

Figure 2: Repeated ECG with sinus rhythm

Discussion& Conclusion

Hypocalcaemia can occur in patients with chronic liver disease due to vitamin D dependent metabolism. In both parenchymal and cholestatic liver disease, the intestinal fat malabsorption may cause vitamin D deficiency which may result to hypocalcaemia[5].

The clinical manifestations of hypocalcaemia are vast and cover across many domains. Presentation of paraesthesias especially around the peri-oral region and the peripheries indicates neuromuscular irritability secondary to hypocalcaemia. Patients can even present with a state of confusion or irritability suggesting neurological or mental effect due to hypocalcaemia. Dermatologic clinical features of hypocalcaemia are dry coarse skin and brittle nails. It can also present as dysphagia and abdominal pain due to the smooth muscle involvement[5].

The classical ECG changes in hypocalcaemia is QT interval prolongation secondary to prolongation of ST segment[6]. QT interval prolongation is directly related to the severity of hypocalcaemia[7]. As the QT interval can be longer or shorter depending on the heart rate (i.e. longer QT interval in slower heart rate and vice versa), Bazett's formula (QT interval divided by the square root of the R-R interval) is used to calculate the corrected QT interval (QTc)[7]. The normal duration of QT interval is 350 - 440ms and a QTc duration of more than 500 milliseconds is associated with increased risk of developing life-threatening cardiac dysrhythmias[8,9]. Other ECG abnormalities reported include T wave changes, arrhythmias and other changes mimicking acute myocardial infarction[8].

Diagnosis of hypocalcaemia will require measurement of both serum calcium and albumin. In order to correct for hypoalbuminemia, 0.8 mg/ dL is added to the total serum calcium for each 1.0 g/dL decrease in albumin level below 4.0 g/dL[1].

It is important to distinguish whether the presentation is acute or chronic as the management will depend on the severity and chronicity of the presentation. In an acute hypocalcaemia case, prompt and rapid correction is needed as it predisposes to life-threatening cardiac arrhythmia[10]. A more aggressive treatment will be needed if severe hypocalcaemia results in tetany, refractory hypotension, seizures or arrhythmias.

Calcium gluconate is the preferred replacement compared to calcium chloride as the latter often causes local irritation[5]. The aim of the treatment is to raise the serum ionized calcium concentration

and control of the symptoms. The calcium gluconate should not be given rapidly as it may cause cardiac dysfunction[11]. One to two ampules of calcium gluconate (90 to 180mg of elemental calcium) diluted in 50mL of 5% dextrose or normal saline should be infused intravenously over 10 to 20 minutes and may be repeated until the symptoms resolve[5,11].

In conclusion, acute hypocalcaemia is an uncommon presentation in clinical practice. This case report is aimed to highlight the importance of thorough assessment and prompt management in replacing the calcium.

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Conflict of Interest

No conflict of interest has been disclosed by the authors.

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Individual authors contribution

Conception and design: NKD, AAR. *Critical revision of the article for important intellectual content:* FM, NKD, AHAM, AAR, NAS. *Final approval of the article:* FM, NKD, AAR, AHAM, NAS.

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