Beyond a prolonged QT interval

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A hypocalcaemia case is presented with convulsions, electrocardiogram (ECG) changes and laboratory findings suggestive of osteomalacia. This case constitutes a rare presentation of osteomalacia-induced hypocalcaemia.

Case presentation

A 14-year-old girl was admitted with generalized tonic-clonic convulsions for 2 min along with tongue biting. Her family reported previous recurrent attacks of muscle cramps and carpo-pedal spasms of four months’ duration prior to admission but they had never previously sought medical help.

The patient had a good appetite and no diarrhea, with no prior history of cardiovascular disease or symptoms. She was not on any regular medications. She lived mainly indoors, avoided sunlight, and wore a veil outdoors mainly due to traditional beliefs. Her family noticed an abnormal gait eight months prior to presenting at our clinic and she usually asked for help to stand from a squatting position.

Assessment

The patient had circumoral paresthesia and muscle spasms upon flexion of the metacarpophalangeal joints associated with finger extension. There was no goitre. Severe proximal myopathy involving the shoulder and pelvic girdle was noted.

The patient’s blood pressure was 120/60, with a heart rate of 60 beats per minute, and her body temperature was normal. Auscultation of the lung fields and heart was normal. The calcium was low and alkaline phosphate was high. The magnesium and phosphate levels, complete blood count and liver enzymes were normal. Other laboratory test results are reported in Table 1.

A 12-lead surface ECG showed bradycardia and prolonged QT interval of 0.64 s with a corresponding QTc of 0.666 s (using Bazett’s formula [QTc = QT/√RR – interval]). A second-degree heart block with a 2:1 block and rate was evident (Figure 1). The echocardiogram and thyroid function test were normal.

Management

Calcium gluconate was infused intravenously for several days, and the clinical status of the patient improved. As the serum calcium returned to normal values, 24-h ECG monitoring demonstrated the gradual disappearance of the QT-interval prolongation and the atrioventricular block.

The atrioventricular block disappeared after 4 h of calcium gluconate infusion, but it took seven days for the QT interval to return to normal after the initiation of calcium supplementation.

Discussion

The patient presented with hypocalcaemia without any other electrolyte disorder. The ECG changes were two-fold: type II second-degree atrioventricular block and the prolongation of the QT interval. The latter change is known to be caused by hypocalcaemia, which prolongs the duration of phase two of the action potential (AP) of the cardiac muscle.

In addition, calcium channel function and calcium influx during phase two are modulated by the rate of change of extracellular calcium, all of which affect the QT interval.1

Hypocalcaemia delays ventricular repolarization, thus increasing the QTc interval (normal, 0.35–0.44 s). When electrical systole is prolonged, the ventricles may fail to respond to the next electrical impulse from the sinoatrial node and a 2:1 heart block results. The prolongation of the QT interval is a moderately reliable ECG sign of hypocalcaemia, not for the population as a whole but on a patient-by-patient basis.2 Thus, although monitoring the corrected QT interval (corrected according to the heart rate) is useful, it is not always an accurate means of monitoring hypocalcaemia in any given patient.
Low extracellular calcium levels decrease the slow inward current and intracellular calcium concentration during the AP plateau. The latter decreases the outward current, prolonging phase two of the AP, the total AP duration and the duration of the effective refractory period. As a consequence of low intracellular calcium, contractility decreases. Moreover, hypocalcaemia slightly decreases the rate of diastolic depolarization in the Purkinje fibres and increases excitability through direct interaction with the sarcolemma.3

Table 1. Laboratory results and progression of the ECG QT interval.

| Variable                  | 0 h  | 6 h  | 2nd day | 5th day | 7th day | Reference range |
|---------------------------|------|------|---------|---------|---------|-----------------|
| Serum calcium, mmol/L     | 1.12 | 1.42 | 1.56    | 1.91    | 2.27    | 2.2–2.45        |
| Albumin, gm/L             | 38   | –    | –       | –       | 38      | 34–50           |
| Alkaline phosphatase, U/L | 1017 | –    | –       | –       | 990     | 40–129          |
| PTH, pmol/L               | 28.7 | –    | –       | –       | 19.2    | 1.59–6.89       |
| QT, s                     | 0.64 | 0.4  | 0.4     | 0.4     | 0.38    |                 |
| Corrected QT, s           | 0.67 | 0.52 | 0.48    | 0.46    | 0.45    |                 |
| 25-HVitD, ng/ml           | <3   | –    | –       | –       | –       | 30–75           |

Figure 1. ECG performed on admission, showing second-degree heart block and a prolonged QT interval.
Hypocalcaemia-induced second- and third-degree atrioventricular block has been described in the neonatal and paediatric populations. We describe here the first case of osteomalacia presenting with both hypocalcaemia convulsions and second-degree atrioventricular block. We believe that seizures were related to low calcium as no ventricular arrhythmias were recorded.

Vitamin D deficiency is the most common cause of osteomalacia worldwide, but in the Western world, gastrointestinal disorders causing vitamin D deficiency and hypophosphatemic-osteomalacia are the most common causes. Vitamin D deficiency and osteomalacia are a common problem among veiled Kuwaiti women despite an abundance of sun light; women tend to avoid the sun, and traditional customs play an important role in the prevalence of osteomalacia.

Conclusion

In this case, the correction of hypocalcaemia led to the rapid disappearance of the second-degree heart block and to slower decrease in the QT prolongation. To our knowledge, this is a rare case of second-degree heart block secondary to osteomalacia-induced hypocalcaemia. No other treatment was administered, and the rest of the clinical parameters were normal, which led us to the conclusion that the occurrence of the second-degree heart block was caused by the patient’s hypocalcaemic status.

Declarations

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Guarantor: ZB

Contribution: ZB conceived the idea for the case report and wrote it. OM and MB provided the editorial input, and together with HA were also responsible for the care of the patient

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