ASSAY INTERFERENCE CAUSING FACTITIOUS HYPERVITAMINOSIS D

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25-hydroxyvitamin D is most commonly measured with the use of automated immunoassay technology. It is well known that immunoassays are prone to interference from immunoglobulins present in the circulation. Here we present a case of a monoclonal IgM paraprotein interference in the Abbott Architect 25-hydroxyvitamin D assay, a quantitative delayed one-step competitive immunoassay using chemiluminescent microparticle immunoassay (CMIA) technology. A 68-year-old female with newly diagnosed lymphoplasmacytic lymphoma / Waldenström's macroglobulinaemia and a monoclonal IgM paraprotein of 44 g/L, had a 25-OH vitamin D measurement of >400 nmol/L on the Abbott Architect 25-hydroxyvitamin D assay. This result was inconsistent with clinical findings. A 1:2-dilution of the sample returned a non-linear 25-OH Vitamin D result of 84 nmol/L suggestive of an interferent present in the sample. The presence of rheumatoid factor was excluded with a measurement below the limit of quantitation of the assay. Referral of the sample for measurement of total 25-hydroxyvitamin D by liquid chromatography-tandem mass spectrometry (LC-MS/MS) returned a result of 75 nmol/L. The sample analysed was collected before the patient started chemotherapy and we concluded that the most likely interferent is the monoclonal IgM paraprotein.

References

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THE ACCURACY OF BLOOD GAS ANALYSER RESULTS COMPARED WITH CENTRAL LABORATORY TESTING

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Background: Electrolyte and haemoglobin results from blood gas analysers are often used clinically before results from central laboratory testing are available. However, the accuracy of blood gas analyser results is uncertain.

Methods: Patients who presented to ED during 7 consecutive days and with blood gas analyses and central laboratory samples

timestamped ≤ 20 minutes apart were included. Mean differences in sodium, potassium, bicarbonate, haemoglobin and haematocrit between central laboratory results and blood gas results were calculated. Differences were tested using t-tests and linear regression performed, with R^2 calculated.

Results: 55 sets of corresponding samples were analysed. Compared with venous blood gas results, mean central laboratory results were higher for sodium (5.0 mmol/L, 95% CI=4.4,5.5, $p<0.001$, $R^2=0.83$) and potassium (0.31 mmol/L, 95% CI=0.27,0.35, $p<0.001$, $R^2=0.96$). Mean central laboratory concentrations were lower for bicarbonate (−4.4 mmol/L, 95% CI= −4.9,−3.9, $p<0.001$, $R^2=0.93$) and haematocrit (−2.3%, 95% CI= −3.4,−1.2, $p<0.001$, $R^2=0.83$). Mean concentration did not systematically differ for haemoglobin but had lower correlation (3.6 g/L, 95% CI= −0.4,7.6, $p=0.074$, $R^2=0.72$).

Conclusion: Blood gas results may differ systemically and have moderate to excellent correlation with central laboratory testing. Analysers should be calibrated to minimise systematic errors and clinicians should be aware of differences to facilitate appropriate clinical decision making. The blood gas analyser involved has since been replaced.

RECURRENT SEVERE RHABDOMYOLYSIS IN A PREGNANCY AFFECTED BY MYOTONIC DYSTROPHY TYPE 1

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An 18-year-old female G1P0 at 17+5 weeks pregnant with history of myotonic dystrophy type 1 (DM1) presented with bilateral leg ache, intermittent abdominal pain, and dark urine. Blood tests confirmed severe unprovoked rhabdomyolysis with CK 72546 U/L. CK remained higher than baseline throughout pregnancy and she had two further episodes of severe rhabdomyolysis requiring admission. Nonischaemic forearm exercise testing demonstrated normal response to exercise with no evidence of metabolic myopathy; having normal pre-exercise and post-exercise acylcarnitine profile and amino acid profile, appropriately rising lactate (1.0 mmol/L to 2.4 mmol/L), no significant increase in ammonia (29 μ mol/L to 31 μ mol/L), and appropriate deoxygenation (PvO₂ 36 mmHg to 32 mmHg). Three separate urine metabolic screens demonstrated normal glycosaminoglycans (6.0 mg/mmol; 8.6 mg/mmol; 6.3 mg/mmol creatinine; reference range <15.4) and no evidence of fatty acid oxidation defect. A rhabdomyolysis gene test panel involving over 50 genes revealed no underlying cause for rhabdomyolysis. At 33+4 weeks, our patient delivered a stillborn baby strongly affected by DM1 due to anticipation and unable to perform spontaneous respiration. Our patient's CK fell to 936 U/L one day after birth, lower than all levels during pregnancy, confirming pregnancy as the cause of rhabdomyolysis. We believe this is the first case of spontaneous recurrent severe rhabdomyolysis during pregnancy with DM1.