CKD Mineral Bone Disease: An audit of a paediatric population

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INTRODUCTION

Chronic kidney disease associated mineral bone disease (CKD-MBD) in common in those with CKD. It is defined as a systemic disorder of mineral and bone metabolism due to CKD, manifest by either one or more of: abnormalities of calcium, phosphorus, parathyroid hormone (PTH), or vitamin D metabolism; abnormalities in bone turnover, mineralization, volume linear growth, or strength; and Extra-skeletal calcification.
CKD-MBD is associated with poorer morbidity and mortality outcomes, with increased financial costs for delivery of care and episodes of hospitalisation as well as significant impact upon the individual and their family.
The biochemical markers of CKD-MBD (calcium, phosphate, PTH and vitamin D) are routinely measured on attendance at children's outpatient clinic, the frequency of which depending upon CKD stage and prior derangement, in order to intervene as early as possible. Commonly used therapies include prescription of phosphate binding agents (such as calcium carbonate) and vitamin D therapy, and manipulation of the child's diet, such as placing the child on a low-phosphate diet.
This audit will look at the CKD population at Southampton Children's Hospital, and the biochemical measures used in its monitoring and intervention upon found derangement.

STANDARDS ASSESSED

Kidney Disease Improving Global Outcomes (KDIGO) guideline[1]: In patients with CKD stages 2-5, maintain calcium and phosphorus in the normal range and to correct 25(OH)-vitamin D deficiency and insufficiency. Although the optimal PTH levels is now known, those with PTH levels above the normal reference range should be evaluated for derangement in calcium, phosphate and vitamin D. In those with persist elevated PTH, vitamin D therapy should be commenced. Those on dialysis should maintain 2-9-times the upper limit of PTH.

METHODS and SUBJECTS

Children with CKD stages 2-5 under the care of the paediatric nephrology team and Southampton Children's Hospital were reviewed, retrospectively: Serum values, taken as part of usual clinical care, of PTH, corrected calcium, phosphate and 25(OH)-vitamin D were recorded and compared to reference ranges. In those with results that were not in the appropriate range, the electronic patient record was interrogated to determine clinical action, looking particularly for alteration in the patients' medications aimed to correct these abnormal values.

RESULTS

A total of 44 children were reviewed, with a mean GFR (Schwartz) of 46ml/min/1.73m^2 (range: 5.1-98.9), and age of 11.3 years (range: 4 - 17.78). *Figure 1* shows the biochemical values of the audit population. Mean values were : cCa 2.44mmol/l (SD 0.1); iP 1.39 mmol/l (SD 0.3); PTH 10.56pmol/l (SD 15.23) and 12-(OH)-vitD 94.34nmolL (SD 44.98). Percentage of values that fell out of the normal reference range were: cCa 4.5%; iP 9.1%; PTH 32.2%; and 12-(OH)-vitD 13.6%.
The 2 cCa values that were outside of the normal reference range were within 0.02mmol/l of the reference range.
Of the 3 iP values that were high, 2 normalised within 20days, and all had an alteration in their therapy (increased phosphate binder, increased vitamin D, and/or dietary manipulation).
Looking at those with elevated PTH, all had manipulation of therapy. 3 of those with elevated PTH were receiving haemodialyis, in which although the optimal PTH level is not known, higher target values are suggested (x2-x9 upper limit of normal; all of these were receiving vitamin D therapy. The outlier value of PTH (91pmol/l) belonged to a child with known hyperparathyroidism.
6 children had low 25-(OH)-vitD levels, all of which had vitamin D therapy increased or commenced as a result. Although these children had insufficiency (<50nmol/l), no children had vitamin D deficiency (<25nmol/l).

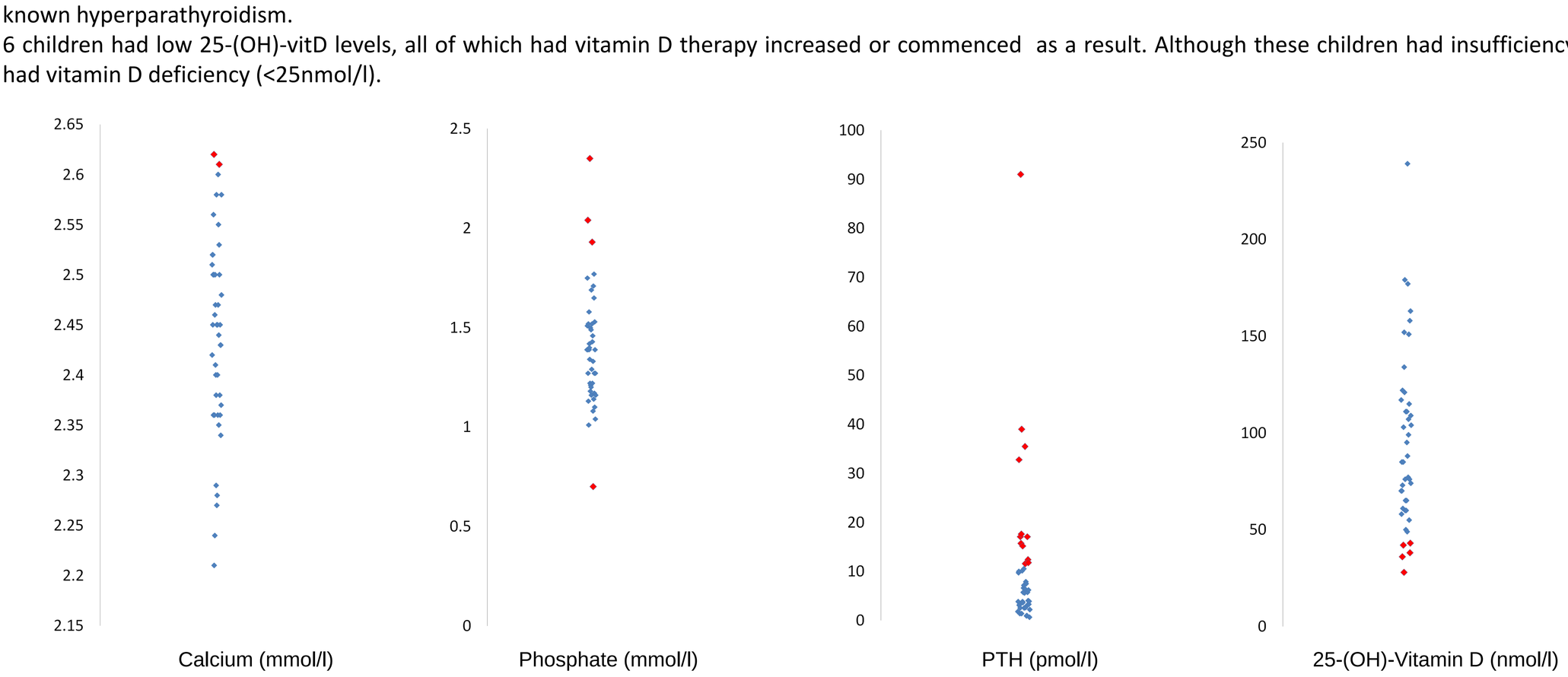

Figure 1. Biochemical values associated with MBD-CKD: Corrected calcium, inorganic phosphate, parathyroid hormone and vitamin D status. Values that fall outside of the normal reference ranges are in red.

DISCUSSION

This audit demonstrates a good compliance with guidance, demonstrating biochemical measures of CKD-MBD within the ranges defined by the KDIGO guidelines in a sample group of children with CKD at Southampton Children's Hospital. Children in whom values fell out of the appropriate ranges received manipulation of therapy to try to correct this. Documentation of such changes were generally clear in the electronic record, although retrospectively gathering of data regarding dietary restriction (of phosphate, for example) with which CKD patients are commonly managed, was challenging, due to the poor recording of this information in the electronic patient records.
We found that 13.6% of children had vitamin D insufficiency. The UK has an childhood rate of vitamin D insufficiency of 35%[2], our population had a significantly lower percentage than this. This is likely due to our cohort having had previous investigation and treatment, as well as having more sun exposure than most of the UK and a mostly Caucasian population.
Although this audit uses a lower limit of 25-(OH)-vitD of 50nmol/l, there is evidence that higher levels may be beneficial in the CKD population having an anti-proteinuric effect, and potentially preserve renal function.

RECOMMENDATIONS

•Improved documentation of dietary restrictions placed on the child. A suggestion is that this be documented at the start of the clinic letters so that all data is on a single reference document.
•Ongoing monitoring of biochemical parameters of CKD-MBD; depending upon CKD stage and prior derangement.
•Regular clinical audit to assess future compliance.

REFERENCES

1. Kidney Disease: Improving Global Outcomes (KDIGO) CKD–MBD Work Group. **KDIGO clinical practice guideline for the diagnosis, evaluation, prevention, and treatment of chronic kidney disease–mineral and bone disorder (CKD–MBD).** *Kidney International* 2009; 76 (Suppl 113): S1–S130.
2. Absoud M, et al. **Prevalence and Predictors of Vitamin D Insufficiency in Children: A Great Britain Population Based Study.** *PLoS ONE* 2011, 6(7): e22179