showed a high Calcium of 2.89 mmol/L with a low urine Calcium supporting the likely diagnosis of familial hypocalciuric hypercalcaemia. Molecular analysis of AP2S1 gene was abnormal. This result confirmed a molecular diagnosis of FHH type 3.

Discussion FHH is an autosomal dominant condition. FHH1 is caused by loss of function in CASR gene, FHH2 by a mutation in GNA11 gene and FHH3 identified as mutation in AP2S1 gene. Mutations lead to an elevation of the normal set point for maintaining normal plasma calcium levels. This causes mild to moderate hypercalcaemia with ionised calcium levels usually within 10% of the upper limit of normal. Urine calcium/creatinine ratio tends to be low in FHH whereas it is usually elevated in primary hyperparathyroidism. In the classic description of FHH, individuals are asymptomatic with normal bone density and no intervention is usually required.

P41 NOT YOUR TYPICAL RICKETS CASE

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10.1136/archdischild-2019-epa.396

Presentation A healthy caucasian 3 year old girl was referred due to bowing of her femora, apparent since she started walking at 13 months. She was reported to be clumsy and tire easily. There was no history of fractures or leg pain. Her height had dropped from the 75th centile to between the 25th and 50th centile. Initial investigations showed mildly low corrected calcium and phosphate, slightly raised alkaline phosphatase and a sufficient vitamin D level of 50nmol/L. She was treated with oral Vitamin D supplements. X-ray of knees was normal. On review after 6 months, bowing had progressed and height had fallen further. This led to the consideration of rarer forms of rickets.

Further investigations Urinary calcium creatinine ratio was normal at 0.07 but urinary phosphate: creatinine ratio was elevated at 4.36; with reduced tubular reabsorption of phosphate - in keeping with a diagnosis of X-linked hypophosphataemic rickets. This was confirmed by detection of a mutation in the PHEX gene.

Skeletal survey showed lower limb abnormalities, including flared metaphyses, widening of the growth plates and buttressing of the femur and tibia. Renal ultrasound showed no nephrocalcinosis. Parental blood tests showed a slightly low phosphate level in her mother.

Progress and treatment She was treated with oral phosphate supplements and alfacalcidol. Since commencing treatment her growth has improved and she remains active but still tires easily. She was subsequently diagnosed with moderate sensorineural hearing loss, another feature of hypophosphataemic rickets, and has had bilateral hearing aids fitted.

X linked hypophosphataemic rickets Rickets is a disorder of the growth plate, due to inadequate supply of phosphate to growing bones. Mutations in the PHEX gene cause increased levels of fibroblast growth factor 23 (FGF23), resulting in reduced absorption of phosphate in the proximal renal tubule. Although rare, it is the most common form of hereditary rickets, and usually presents in the first 2 years of life. Investigations show low serum phosphate and the key feature is significant phosphaturia (calculated by TmP/GFR). Patients are at increased risk of dental complications, enthesopathy, lumbar lordosis and hearing

impairment. Phosphate supplements replace renal losses and calcitriol increases phosphate absorption from the gut and reduces PTH, preventing nephrocalcinosis. A new treatment, Burosumab is a monoclonal IgG1 antibody that binds excess FGF23. This normalises phosphate levels and improves bone mineralisation.

P42 SEPTIC ARTHRITIS OF ELBOW JOINT IN AN 11 MONTH BABY FOLLOWING CHICKENPOX, A CASE REPORT

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10.1136/archdischild-2019-epa.397

An 11 month baby girl presented with 1 week history of left elbow pain and swelling with restricted mobility, on and off fever treated with paracetamol at home. 3 weeks prior to her presentation she had chickenpox with generalized skin rashes. Baby has no past medical history and up to date with her vaccinations. Clinically elbow was swollen with redness at the dorsal lateral side mainly, very tender to touch with almost no range of motion at a slight flexed position. Routine bloods showed significant increase in the inflammatory markers, ultra-sound of the elbow show moderate joint effusion with fluid in the anterior and posterior aspects of the joint with small foci of debris within the effusion, Synovial thickening and increased vascularity with no irregularity of the underlying bony contours. As simple aspiration was difficult to be performed given her age, patient was taken to theatre and urgent drainage on the day of presentation was done, with about 10–15ml of yellowish discharge drained from the lateral side of the elbow. Blood culture along with the joint fluid showed growth of *Streptococcus Pyogenes* which was sensitive to Penicillin. After 4 weeks of Intravenous Penicillin and another 4 weeks of oral Amoxicillin, baby showed complete recovery both clinically and on her routine bloods.

P43 THE POWER OF THE MIND

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10.1136/archdischild-2019-epa.398

Introduction Somatoform disorders are a challenge for all branches within medicine but particularly for those working within the paediatric sphere. They are characterized by physical symptoms that are inconsistent with or not fully explained by any underlying medical or surgical diagnosis. In order to reach the diagnosis of a somatoform disorder, children are typically subjected to a number of investigations.

Somatoform disorders can have a significant impact within two domains; the economic impact on healthcare resources in reaching a diagnosis and the effect on children in terms of the underlying emotional stress, the practical impact on families, school absenteeism and the treatment of such a condition. For the purposes of this study, we examined the incidence of somatoform disorders within an orthopaedic cohort of patients.