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VITAMIN C DEFICIENCY IN CHILDREN WITH AUTISM SPECTRUM DISORDER

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INTRODUCTION
Avoidant/restrictive food intake disorder (ARFID) is common in individuals with autism. Micronutrients deficiencies should be considered in any physical illness.

RESULTS
We are reporting 2 patients with autism with Vitamin C deficiency. Case A, 9 years old boy, born prematurely 34 weeks with underlying autistic spectrum disorder at 4 years old. He presented with bilateral left lower limb pain since mid June 2020, at 8 years old associated with fever, anorexia and weight loss. Mother reported child is having difficulty with mobilization, refused to walk and required wheelchair. Parents assumed child has pain over his hip and knee due to limitation of his language. Initial diagnosis of Juvenile Rheumatoid arthritis was made and patient was referred to Rheumatologist Hospital Selayang. Vitamin C deficiency was suspected due to corkscrew hair. He has subperiosteal hemorrhage with bilateral knee fixed flexion deformity. He was treated with prednisolone tapering dose over 2 months, Ibuprofen, Multivitamin, Vitamin C and syrup FAC and knee splint. He started walking again after one month of treatment with full recovery after 3 months. Case B, 8 years old boy with underlying autistic spectrum disorder since 5 years old. He presented with bilateral thigh pain since April 2021 associated with difficulties in mobilization. He was treated for reactive arthritis and completed IV cloxacillin for 5 days with regular analgesic and physiotherapy. Vitamin C deficiency was suspected and treated with oral vitamin C. Initial Vitamin C level was low, <5 (normal 28-120). He started walking again after 3 weeks of treatment in June 2021.

CONCLUSION
Micro nutrient deficiencies such as Vitamin C should be considered in Autistic Spectrum Disorder children presenting with musculoskeletal pain.

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NEONATAL SEIZURE IN ASYMPTOMATIC MATERNAL HYPERPARATHYROIDISM

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INTRODUCTION
Primary maternal hyperparathyroidism is a known aetiology causing neonatal hypocalcemia, which usually presents in the first 2 weeks of life. Fetal parathyroid activity will be supressed by maternal hypercalcemia resulting in impaired parathyroid responsiveness to hypocalcemia after birth. Prolonged and severe hypocalcemia may lead to convulsions.

RESULTS
A 10-day-old male infant was admitted to our Neonatal Intensive Care Unit (NICU) due to recurrent episodes of unprovoked generalized clonic seizure for two days. Clinically, his anthropometric measurements were normal. He is not dysmorphic and neurological examination was normal. Laboratory analysis showed hypocalcemia and hyperphosphatemia. The rest of his metabolic panel, liver function studies, CBC, CRP, chest x-ray, and CT scan of the brain were all normal. He had inappropriately low parathyroid hormone (PTH) level for concurrent degree of hypocalcemia and low 25-OH vitamin D. Work up performed on the mother revealed hypercalcemia 3.26 mmo/L, phosphorus 0.85 and PTH 29.6. Mother was seen by endocrinologist for possibility of parathyroid adenoma. The baby was treated with calcium gluconate infusion, oral calcium supplement and vitamin D.

DISCUSSION
Maternal hypercalcemia or hyperparathyroidism leading to suppression of fetal parathyroid gland often causes transient neonatal hypocalcemia. Low level of vitamin D of the patient might have exacerbated hypocalcemia, which may lead to convulsions.

CONCLUSION
Undiagnosed maternal hyperparathyroidism causes severe hypocalcemia in newborn. Therefore, appropriate investigations should be carried out when a newborn presents with hypocalcemic seizure to exclude maternal hyperparathyroidism even if the mother is asymptomatic.