Journal of Paediatrics and Child Health

Dear Editor,

PROPHYLACTIC VITAMIN D SUPPLEMENTATION IN HIGH-RISK BREASTFED INFANTS

Vitamin D deficiency in children is associated with decreased bone mineralisation, deformational rickets and, in severe cases, hypocalcaemic seizure, motor delay and cardiomyopathy.[1] Infant vitamin D status at birth is a reflection of maternal stores, and deficiency less than 50 nmol/L occurs in a striking 11% of all infants in Australia, and in 72% of infants whose mothers have dark skin.[2] Breastfed infants are at higher risk of deficiency, as, despite its benefits, breastmilk contains insufficient vitamin D for infant requirements. Most Australian formulas contain vitamin D supplementation.[1]

Australian position statements recommend routine oral vitamin D supplementation in all breastfed infants who are at high risk of deficiency (e.g. where the mother has known vitamin D deficiency, is dark-skinned or is veiled).[1] Of note, international guidelines in the UK and USA are more interventional and recommend routine oral vitamin D supplementation in any breastfed infant.[2]

Over a 6-month period we have seen four vitamin D-deficient breastfed infants who had not received prophylactic vitamin D, despite being previously known to be at high risk. They presented with complications, including rickets and hypocalcaemic seizures. The mothers in each case had known vitamin D deficiency during pregnancy, with two of the mothers also having dark skin. The infants were born at different hospitals, suggesting a systemic problem of failure to commence prophylactic supplementation in accordance with national guidelines.[3]

We reviewed the protocols of 11 maternal-neonatal units for the prevention of infant vitamin D deficiency. All recommended empiric treatment of breastfed, high-risk group infants for 12 months. The suggested treatment is generally 400 IU cholecalciferol daily.[4] Less commonly Stoss therapy with 50 000 IU cholecalciferol followed by 400 IU daily after 3 months is recommended,[4] although we have seen drug errors associated with the use of the Stoss protocol in neonates.

Unfortunately, there was major inconsistency across the protocols in process for identification of infants requiring preventive vitamin D treatment, and of who is responsible for arranging treatment. Protocols variously suggested a note in the parent-held child health record, or a note to the general practitioner, and in some cases, responsibility was placed solely on the parents to identify and follow up their own infant's supplementation.

Without assessment of whether every newborn is in the high-risk group requiring routine vitamin D supplementation, some children will continue to suffer serious complications following missed treatment. A standardised method for risk assessment is required, and we propose a checkbox is added to the child health record for routine consideration at the birth and early maternal child health visits. This would ensure vitamin D is provided to all infants considered at risk.

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Conflict of interest: None declared.

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Dear Editor,

LATE DUMPING SYNDROME IN A 17-YEAR-OLD FEMALE WITH CHARGE SYNDROME

We would like to draw your attention to a digestive disorder that has not been previously described in CHARGE syndrome. CHARGE syndrome is a mnemonic that stands for some of the common clinical features: coloboma of the eye, heart problems, atresia/stenosis of the choanae, retardation of growth and/or development, genitourinary abnormalities, and ear anomalies. A 17-year-old female with a confirmed genetic diagnosis of CHARGE syndrome presented to her general paediatrician's clinic describing newly occurring symptoms of lightheadedness, dizziness and a blurred visual field with black dots, approximately 1 h after a meal.

Her CHARGE syndrome features[1] included retinal coloboma; choanal stenosis and atresia; semicircular canal hypoplasia; cranial nerve dysfunction (absent sense of smell, facial palsy, hearing and swallowing impairment); ear malformations; heart defects; developmental delay; and genitourinary abnormalities. Past surgical history included two Nissen fundoplication surgical procedures (age 3 and 9 years) to treat her severe gastroesophageal reflux disease. Gastrostomy tube feeding was necessary up until 13 years of age.

The adolescent was diagnosed with late dumping syndrome, which refers to symptoms and signs of reactive hypoglycaemia when food reaches the small bowel too rapidly, as the presence of glucose in the jejunum is a strong stimulus for insulin secretion.[2] It can occur after a gastrectomy, oesophageal surgery or even after no surgery. Symptoms include hypoglycaemia, perspiration, palpitations, hunger, weakness, confusion, tremor and syncope.[2] First line treatment consists of consuming smaller amounts of food in one sitting, delaying liquid intake until at least 30 min after a meal and avoiding sweetened foods.[2]

Nissen fundoplication is one of the most common surgical procedures undergone in CHARGE syndrome to treat gastroesophageal

Table 1 Medical and psychosocial issues that can emerge in adolescence and adulthood in CHARGE syndrome[3]

Scoliosis†
Delayed puberty†
Late dumping syndrome†
Overactive bladder†
Sleep apnoea
Abdominal colic
Retinal detachment/cataract
Migraines
Urinary tract infections
Seizures/epilepsy
Hypoglycaemia†
Overstuffing of food in one's mouth†
Pocketing of food in cheeks†

†Experienced by our patient.

reflux disease, with some individuals undergoing this procedure two or three times due to a high failure rate.[1,3] Late dumping syndrome has not been previously reported in the CHARGE syndrome literature. Its presentation may be unique in CHARGE syndrome, with symptoms presenting up to a decade after surgery. Furthermore, the treatment recommendations can be difficult to implement as individuals often display difficult feeding behaviours such as pocketing of food in cheeks, over-stuffing one's mouth with food or consuming texture-restricted diets (e.g. puree only) (Table 1).[3] These feeding challenges are often not elicited in the general history. It is known from the CHARGE syndrome Facebook page and the International CHARGE conferences that the symptoms of late dumping syndrome are not unusual.

Continual surveillance for late dumping syndrome should be a standard part of health care management in CHARGE syndrome. The newly developed checklist[4] can aid in health supervision across the lifespan, making sure that new issues are not missed.

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Conflict of interest: None declared.

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Dear Editor,

SHORT SLEEP DURATION AND OBESITY AMONG CHILDREN

I read with interest the report by Li *et al.*, who conducted a meta-analysis of prospective studies to know the associations between sleep duration and obesity/body mass index (BMI) in children.[1] The pooled relative risk (RR) (95% confidence interval) of short sleep duration for obesity was 1.45 (1.14–1.85). After excluding two cohorts to avoid heterogeneity, RR (95% confidence interval) was 1.30 (1.20–1.42). The authors concluded that short sleep duration increased the risk of childhood obesity. I have some concerns about their study.

First, Li *et al.* reported that sleep duration was negatively associated with BMI, waist circumference (WC), fat mass percentage (FAT%) in 6–12-year-old children by adjusting serum high-molecular-weight adiponectin.[2] In addition, sleep duration was negatively associated with BMI, WC and FAT% in 13–18-year-old children by adjusting serum retinol-binding protein 4. They adopted multiple linear regression analysis, and negative association between sleep duration and obesity in both generations was observed by adjusting different confounders. Although BMI, WC and FAT% are closely associated, there is a need of setting cut-off of obesity from different obesity indices. It should be paid with caution to calculate RR of short sleep duration for obesity.

Second, Navarro-Solera *et al.* evaluated the association between sleep duration and cardiovascular risk factors in 7–16-year-old obese children.[3] Short sleep duration was significantly associated with cardiovascular risk factors, including thyroid-stimulating hormone, retinol-binding protein 4, homocysteine and mean arterial pressure by logistic regression analysis. In contrast, there was no significant association between sleep duration and inflammatory markers, adipokines or BMI. This is a cross-sectional study and causality of the association cannot be confirmed. On this point, a meta-analysis of prospective studies by Li *et al.* has an advantage on the relationship.

Finally, definition of short sleep duration among children was presented by National Sleep Foundation, which recommended that sleep range in children aged between 6 and 13 and teenagers aged between 14 and 17 were 9–11 h and 8–10 h, respectively.[4] As there are some definitions in short sleep duration among target articles for a meta-analysis, risk assessment of short sleep duration for obesity among children should be conducted by stratifying age.

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Conflict of interest: None declared.