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Short Communication

Risk Factors of Metabolic Bone Disease of Prematurity in Children

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DESCRIPTION

The multifactorial systemic disease known as Metabolic Bone Disease of Prematurity (MBDP), which is characterised by a decrease in bone-like tissue and bone mineral content as well as potential biochemical changes in calcium and phosphorus metabolism, is influenced by dietary and biomechanical factors. The main point is that preterm infants' bone mineral reserves are insufficient for normal bone growth and development. As a result, imaging changes like hypophosphatemia, hyperalkaline phosphatase, bone mineralization deficiency, and other manifestations may also occur in addition to blood biochemical changes like these. According to earlier research, the incidence of MBDP is 32% and 54%, respectively, in preterm children with Very Low Birth Weight (VLBW) and Extremely Low Birth Weight (ELBW) [1].

The diagnosis of metabolic bone disease of infancy requires a thorough evaluation of the patient's medical history, clinical manifestations, biochemical indicators, and imaging tests because the diagnostic criteria for the condition are not always consistent. The early stages of MBDP are asymptomatic until substantial bone demineralization takes place [2]. It has an insidious onset. The most noticeable clinical manifestations are cranial deformities, including enlarged cranial sutures, an enlarged anterior fontanelle, a forehead bulge, cranial softening, thickening of the rib and rib cartilage junction, thickening of the carpal joints, and, in severely affected patients, rib or long bone fractures. Biochemical markers and imaging tests are used to assess the condition of the bones in newborns. Serum calcium, phosphorus, Alkaline Posphatase (ALP), Parathyroid Hormone (PTH), and 25-hydroxyvitamin D (25(OH)D) are the most often utilised blood biochemical markers. Both calcitonin and parathyroid hormone are involved in controlling the body's blood calcium levels. When blood calcium levels drop, the body keeps them stable by releasing calcium from bones under the control of parathyroid hormone. When the body loses calcium, blood calcium can be normal or even high. It only starts to fall when bone calcium reserves are used up in the late stages of MBDP. Therefore, using blood calcium to diagnose MBDP at an early stage is useless. Hypophosphatemia is one of the initial blood chemistry alterations seen in infants or kids with MBDP. A steady decline in blood phosphorus levels indicates insufficient phosphorus intake and an elevated risk of osteoporosis. Blood phosphorus concentrations are a helpful indicator to review bone phosphorus reserve. A state of calcium depletion results from persistent hypophosphatemia, which also causes increased bone resorption and increased renal calcium excretion. MBDP is linked to elevated blood alkaline phosphatase levels, and elevated blood alkaline phosphatase levels can appear before clinical symptoms. Plasma calcium ion concentration is primarily responsible for controlling PTH secretion. By encouraging calcium reabsorption by the renal tubules, mobilizing osteolysis, and phosphate excretion, the blood calcium level is kept stable [3]. Neonatologists uses plasma parathyroid hormone as a supplement to aid in the screening, diagnosis, and monitoring of MBDP, but it is not exploited to its full potential. While serum 25(OH)D can be normal, decreased, or even raised, the main cause of MBDP is calcium and phosphorus deficiency, hence 25(OH)D is not employed as a diagnostic marker for MBDP. Urine calcium, phosphorus, urinary calcium/creatinine, urinary phosphorus/creatinine, and Tubular Reabsorption of Phosphorus (TRP) are examples of urinary biochemical markers. Urinary calcium and phosphorus levels are higher, which suggests improved bone mineral deposition [4]. Bone mineral density is measured via imaging procedures, mostly using X-rays and Dual Energy X-ray Absorptiometry (DEXA). X-rays of MBDP patients may reveal fractures, subperiosteal new bone development, cupping or burr-like changes at the epiphysis, enlarged rib ends, and osteoporosis at the ends of long bones. X-rays should only be used to diagnose severe MBDP in cases when there is considerable osteoporosis or bone fractures since they may not discover osteoporosis in cases where there has been 20% to 40% bone loss. Consequently, X-rays are not appropriate for early diagnosis even if they are highly specific for the diagnosis of MBDP. While reflecting only the two-dimensional area density of the bone and not its three-dimensional density, DEXA is the gold standard for the diagnosis of osteoporosis. DEXA is technically challenging to employ for MBDP screening, making it unsuitable for routine screening. The diagnosis of MBDP is now primarily based on common clinical symptoms and radiographic abnormalities, however by that point; the bone mineral density may have drastically diminished. Since the majority of MBDP

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does not manifest any overt clinical symptoms, early clinical screening and surveillance are the mainstays of its diagnosis.

For metabolic bone disease of prematurity, prevention is more important than treatment, and the aim of managing bone health in preterm newborns is to provide appropriate calcium and phosphorus intake to support normal bone formation. Age in days, calcium, phosphorus, lactose intake, and intake of fat are all positively connected with postnatal calcium and phosphorus absorption rates in preterm newborns. Vitamin D levels also have an impact. For preterm infants with high-risk factors, preventive measures should be put in place. For infants with very low birth weights, nutritional management, particularly calcium, phosphorus, and vitamin D consumption, should be strengthened. Drugs that alter bone metabolism should not be used for extended periods of time, and biochemical signs should be carefully watched. Infants at high risk for MBDP should continue receiving nutritional formula after discharge until the full-term correction or when there is no longer any evidence of combined MBDP on routine clinical monitoring.

CONCLUSION

After obtaining complete enteral feeding, infants at risk for MBDP can be educated in daily passive activities to avoid MBDP. If MBDP is identified, thorough nutritional management measures should be implemented right away. In order to correct abnormal metabolic states like hypophosphatemia, secondary hyperparathyroidism, and vitamin D deficiency as soon as possible, the key to treatment is supplementation with calcium, phosphorus, and vitamin D preparations on the basis of intensive nutritional formula feeding, making sure it reaches the target amount. Phosphorus supplements alone can exacerbate the body's calcium and phosphorus balance, resulting in secondary hyperparathyroidism and worsening bone lesions. Therefore, it is stressed that in addition to stronger formula feeding, newborns with MBDP should also receive supplemental calcium and phosphorus supplements. Vitamin D supplementation is required concurrently for infants with MBDP in order to enhance intestinal absorption of calcium and phosphorus. After a few weeks, increasing enteral or parenteral mineral supplementation will improve the imaging results. When the course of treatment has lasted 6–8 weeks, imaging can be used to evaluate its effectiveness. There are a lot of unidentified factors that affect the prognosis of MBDP. Regular follow-up and monitoring are stressed in preterm infants with MBDP risk factors in order to decrease MBDP problems and improve their linear growth as well as their short- and long-term prognosis. In order to avoid excessive urinary calcium excretion, it is important to maintain normal blood calcium and phosphorus levels as well as the ideal growth of indicators like length, weight, and head circumference.

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