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# Rickets treatment improves more than bone health in toddler with autism spectrum disorder: A brief report

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**Abstract**

Children with autism spectrum disorder have been found to have lower levels of vitamin D than their peers. Our case report supports the hypothesis that vitamin D may be an effective treatment for developmental delay in autism. In addition, we review the literature surrounding vitamin D deficiency as a potential cause of autism spectrum disorder and the role that vitamin D may play in treatment.

**Keywords**

Neurology, general pediatrics, child psychiatry

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## Introduction

Nearly 50% of the worldwide population, with peak incidence in childhood and menopausal women, is affected by hypovitaminosis D, which can be attributed to poor dietary intake, breastfeeding without vitamin supplementation, lack of exposure to sunlight, frequent use of sunscreen, darker skin tones, and use of a variety of different medications including anticonvulsants and antivirals.[1] While vitamin D deficiency has been independently linked to increased mortality, it is also found to be a risk factor for many chronic diseases including cardiovascular disease, breast cancer, colon cancer, prostate cancer, diabetes, and most recently neuropsychiatric disorders.[2] We present a case supporting vitamin D supplementation as a potential treatment for symptoms of autism spectrum disorder (ASD). Informed consent was obtained in writing from the below patient's mother to share all information presented in the article.

## Case

A 3-year-old African-American boy presented to the emergency department with his mother for complaints of decreased urine output, decreased intake of both solids and liquids, and increased fussiness for 2 weeks. The patient had also been vomiting and refusing to walk for 3–4 days. During the earlier portion of this 2-week illness, the patient did have rhinorrhea and cough which had resolved upon this visit. Prior to this emergency department visit, over the last 14 days, the patient had been diagnosed with viral illness and thrush, having been treated with intravenous fluids and nystatin upon two different visits to our emergency department and a local urgent care. The mother denied any fever, rash, or diarrhea in the child over the last several weeks. His only known sick contact was an older brother with recent rhinorrhea and cough.

Significant medical, psychiatric, and surgical history for this child included ASD without any development of spoken language to this point, hydronephrosis with bilateral ureteropelvic junction repairs, and intra-abdominal tests with bilateral orchiopexy. Immunizations were up to date, no routine medications were given to the patient, and family history was noncontributory. The boy lived at home with his mother and three siblings, and there was no concern for abuse, neglect, or exposure to drugs and alcohol in the home. His mother was particularly concerned that recent decreased

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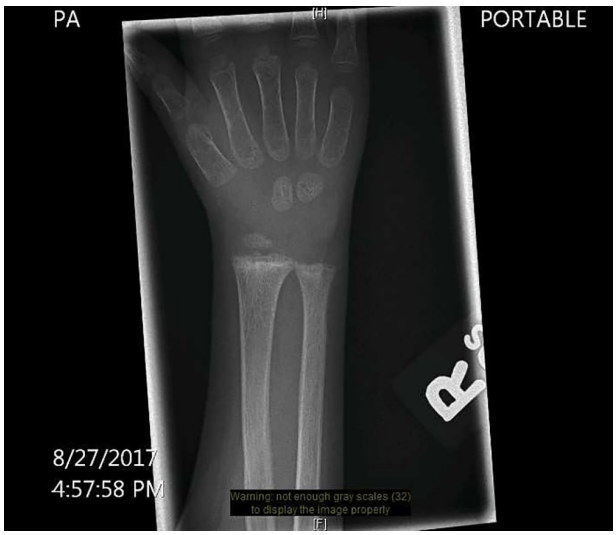


**Figure 1.** Radial and ulnar metaphyseal fraying.

urine output may be related to his history of hydronephrosis, prompting her to bring him to the emergency room again.

In the emergency department, the patient was ill-appearing on examination with dry oral mucosa and hypoactive bowel sounds. However, he had an otherwise normal physical examination, was not in any acute distress, and had normal vital signs. He was given two boluses of normal saline causing spontaneous urine output. An electrolyte panel was obtained showing low calcium at 6.0 mg/dL, low K+ at 2.9 mg/dL, and low albumin at 3.2 mg/dL. Alkaline phosphatase was elevated at 644. The patient was admitted to the children's hospital and treated with fluids including potassium and calcium replacement overnight. Other laboratory tests obtained in the emergency department were normal including viral panel, blood cultures, complete blood count, C-reactive protein, urinalysis, and additional electrolytes.

On day 2, vitamin D and parathyroid hormone (PTH) levels were obtained and found to be critically abnormal with a vitamin D level of 7.6 ng/mL (normal 20–100 ng/mL) and a PTH of 912 (normal 8–97). Urine calcium was also obtained and found to be low at less than 5 mg/dL. X-rays of the patient's wrists were obtained (Figure 1) and showed radial and ulnar metaphyseal fraying and cupping suggestive of rickets. Chest films were within normal limits. Given the patients symptoms including refusal to walk, labs including low vitamin D, serum calcium, urine calcium and high PTH, and the patient's wrist films, he was diagnosed with hypocalcemic rickets secondary to malnutrition. His mother endorsed extremely picky eating since beginning table food as an infant, often having extreme difficulty convincing him to eat anything other than crackers, potatoes, and juice. Renal disease was deemed less likely to be the cause of his acute illness as a renal ultrasound showing stable hydronephrosis was obtained along with normal creatinine and urine output once fluids were administered.

During the remaining week-long hospitalization, the patient's electrolytes and vitamin D levels were trended and he received supplementation of fluids, calcium, vitamin D, magnesium, and eventually protein via total parenteral nutrition (TPN) once it was felt his electrolytes were stable. He was discharged home with continued calcium carbonate (1000 mg twice daily) and vitamin D (4000 IU daily) supplementation and instructed to follow-up with his outside primary care provider and endocrinologist.

One month after discharge, our team followed up with the mother and patient. He had continued taking vitamin D and calcium supplements at prescribed doses. His protein intake had increased as his electrolytes continued to normalize. To our surprise, our patient's symptoms of autism spectrum disorder had also improved. Prior to supplementation, at the time of diagnosis, the patient had 18 failed Modified Checklist for Autism in Toddlers (M-CHAT) items, with only 8 failed items following treatment of rickets. The patient had also begun speaking 3–4 words as opposed to biting and scratching to communicate. He had returned to not only walking but also began running. Previously, he had not interacted with other children. He had begun interacting and playing with his siblings and their friends. The patient did not begin a new therapy regimen during this time or have any significant life changes outside of improved nourishment that would explain the improvement in his social responsiveness and other symptoms of ASD.

## Discussion

Recent evaluation of vitamin D levels has shown a statistically significant positive relationship between the diagnosis of ASD and lack of vitamin D in both children and their mothers during pregnancy.[3,4] A recent population-based cohort study of 4229 children showed that low vitamin D levels in mothers mid-gestation or at birth are related to lower scores on a social responsiveness scale, which is used to identify autism-related traits.[5] An additional population study of 639 children in 2016 yielded similar results.[6] In addition, the improvement of autistic traits in our patient after vitamin D replacement indeed suggests that vitamin supplementation may be a safe and effective treatment for children with ASD. While reports in the literature are not plentiful, our case report is not the first. The American Academy of Pediatrics Journal also reported a marked improvement of autistic traits after administering vitamin D to a child with an ASD diagnosis.[7] A randomized clinical trial completed in 2016 involving 109 children with autism disorder treated with high dose of vitamin D showed significant improvement of symptoms without frequent adverse outcomes.[8] While these data suggest that there is a potential for high-dose vitamin D as a symptomatic or preventive treatment, additional large-scale clinical trials are needed to determine whether this is truly effective and safe.

Although improvement of autistic traits is one important benefit of vitamin D supplementation, data suggest that patients with autism are more likely to have selective eating

and resultant vitamin deficiencies of multiple types.[9–11] With the knowledge that vitamin D deficiency is linked to multiple additional chronic diseases mentioned above, it would be reasonable to recommend vitamin supplementation at doses that are currently known to be safe, especially in children with autism disorder due to their high risk of deficiency.

## Conclusion

Given multiple studies above suggesting that low vitamin D is one factor contributing to the pathogenesis of ASD, it is prudent that clinician's obtain levels of this vitamin when concerned for developmental delay or early autism to determine whether this level may in part explain symptoms being observed. Pending further clinical trials, supplementation with vitamin D may be an effective treatment to at least in part ameliorate the severity of ASD. In addition, clinician's should be familiar with the signs and symptoms of rickets disorder when treating a population of patients with ASD. While rickets is more common in developing countries, restrictive eating related in the setting of ASD and other developmental disorders may put patients at a higher risk of this uncommon complication in any location.

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All authors claim legitimate contributorship.

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