

Letters to the Editor

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This series is coordinated by Kenny Lin, MD, MPH, Associate Deputy Editor for *AFP* Online.

Pain Management in Patients with ADPKD

Original article: Autosomal Dominant Polycystic Kidney Disease

Issue date: September 1, 2014

Available at: <http://www.aafp.org/afp/2014/0901/p303.html>

TO THE EDITOR: We read this article with interest, and we would like to comment on the issue of pain management in patients with autosomal dominant polycystic kidney disease (ADPKD). We agree that pain management in ADPKD should include evaluation for concomitant illness. Two patients in our practice with ADPKD who presented with severe chronic low back and abdominal pain illustrate the importance of searching for extrarenal causes.

The first patient, a 55-year-old woman with stage 3 chronic kidney disease secondary to ADPKD, required opioid analgesics because of severe chronic back and abdominal pain, which were initially attributed to polycystic kidneys and liver. She was later diagnosed with spondyloarthrosis and osteoporosis with a compression fracture of the sixth thoracic vertebra.

The second patient, a 51-year-old man with ADPKD and stage 2 chronic kidney disease, had severe chronic back pain thought to be caused by enlarged kidneys. Magnetic resonance imaging demonstrated scoliosis and advanced spondyloarthrosis (Figure 1).

In ADPKD, enlargement of renal and liver cysts causes increased abdominal girth, leading to increased lumbar lordosis, which predisposes patients to degenerative changes of the spine. Additionally, asymmetry in renal cyst enlargement leads to chronic postural alteration and lumbosacral disk disease.¹

Biochemical abnormalities in the homeostasis of calcium and phosphorus begin early in patients with chronic kidney disease, leading to an increase in fracture risk, including spinal compression fractures.^{2,3}

To conclude, disorders of the spine should

always be included in the differential diagnosis of patients with ADPKD and back pain.

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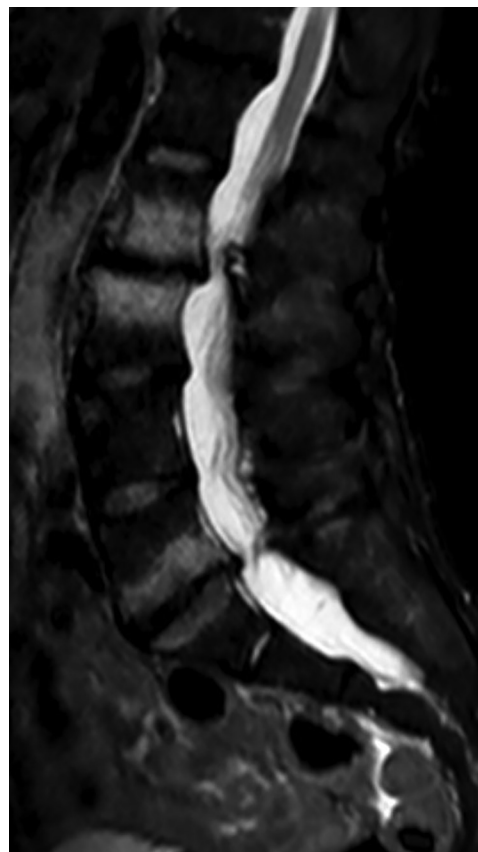


Figure 1. Magnetic resonance imaging shows scoliosis and advanced spondyloarthrosis in a 51-year-old man with autosomal dominant polycystic kidney disease and stage 2 chronic kidney disease.

Letters

IN REPLY: We thank Dr. Niemczyk and colleagues for the additional insight into the evaluation and management of pain in patients with ADPKD and illustrating that the underlying etiology responsible for pain can be multifactorial. Clinicians should also be aware that a debilitating cycle may ensue when chronic pain contributes to or worsens depression and anxiety, negatively affects key factors such as interpersonal relationships and important activities, and worsens the patient's overall disability.¹

Dr. Niemczyk's case presentations are a reminder of how the advancing disease process for ADPKD can indirectly contribute to pain via acceleration of other disease processes, including postural variation and spinal degeneration, and worsening chronic kidney disease leading to renal osteodystrophy. More specifically, for those who have adverse habits related to poor postural tone, the Alexander technique offers a nonpharmacologic approach to diminishing self-propagating and damaging habits, thereby improving coordination, as well as pain management and perception.²

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Vitamin D Supplementation in Premature Infants

Original article: Common Questions About Outpatient Care of Premature Infants

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Available at: <http://www.aafp.org/afp/2014/0815/p244.html>

TO THE EDITOR: Thank you for this very informative article. It did not specifically discuss the importance of vitamin D supplementation. The American Academy of Pediatrics (AAP) recommends that all infants consume a minimum of 400 IU per day of vitamin D beginning shortly after birth, particularly breastfed or partially breastfed infants.¹ Human breast milk is low in vitamin D, containing less than 25 IU per L.² Even with adequate oral intake, a premature infant would still receive far less than the recommended daily amount of vitamin D.

The article does mention that breast milk can be supplemented with a multinutrient fortifier, but that benefit postdischarge is not clear. Although common commercial preparations, (e.g., Similac Human Milk Fortifier, Enfamil Human Milk Fortifier) contain vitamin D, breastfed infants discharged without fortified milk will not benefit from this type of vitamin D supplementation. Sequelae of vitamin D deficiency among breastfed infants, such as rickets and hypocalcemia, are not common; however, supplementation is essential to prevent potential illness in this already vulnerable patient population.

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IN REPLY: We thank Dr. Shipley for the astute observation regarding vitamin D deficiency, osteopenia, and metabolic bone disease in graduates of neonatal intensive care units. Prematurity and the metabolic demands of sick newborns can put them at high risk of poor bone growth and health.¹

Our article focused on guidelines for the outpatient follow-up of newborns discharged from neonatal intensive care units that deviate from routine newborn care and guidelines. As Dr. Shipley stated, the AAP recommends that all infants, shortly after birth, begin receiving 400 IU per day of vitamin D.^{2,3} This can be achieved with consumption of greater than 1 L per day of formula (which usually occurs by approximately one month of age) or vitamin D supplementation for those infants consuming breast milk or less than 1 L per day of formula (i.e., mixed feeding). This recommendation does not differ for graduates of neonatal intensive care units.

Regarding fortification of human breast milk, our discussion was to address concerns of extrauterine growth failure, not vitamin deficiencies. If breast milk is to be fortified as an outpatient, it is typically done with formula, as human milk fortifiers are not readily available other than for inpatient use. Because the amount of vitamin D provided would likely be less than 400 IU per day, supplementation is recommended.²⁻⁴

Recently published studies suggest increasing the recommended vitamin D dosage to 800 IU per day. In a randomized double-blind trial (n = 96), the prevalence of vitamin D deficiency was significantly lower in ►

preterm infants receiving 800 IU per day than in those receiving 400 IU per day at 40 weeks (38% vs. 67%) and at three months corrected age (12% vs. 35%). However, there was no improvement in bone mineralization between the two groups. One infant receiving 800 IU per day had vitamin D excess.⁵ In 2008, the AAP released new recommendations increasing the dosage of vitamin D from 200 to 400 IU per day based on growing evidence of vitamin D deficiencies in infants receiving lower dosages. Until larger trials are completed, 400 IU per day of vitamin D supplementation is the best evidence-based recommendation.

Interestingly, a longitudinal survey from 2010 revealed that very few infants were receiving adequate vitamin D supplementation. Only 5% to 13% of breastfed infants, 9% to 14% of mixed-fed infants, and 20% to 37% of formula-fed infants met the AAP recommendation.⁶ This evidence underscores the importance of vitamin D supplementation. Clinicians are encouraged to inquire about ingestion of vitamin D in this at-risk population.

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Correction

Incorrect vaccine name. The article "ACIP Releases 2015 Childhood Immunization Schedules" (February 15, 2015, p. 265) contained an error in the third paragraph of the right-hand column regarding the tetanus, diphtheria, and pertussis vaccine. The article should have stated that there is a small but real association between febrile seizures and concurrent administration of influenza plus pneumococcal 13-valent conjugate vaccine and/or diphtheria and tetanus toxoids, and acellular pertussis (DTaP) vaccines, not the tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis (Tdap) vaccine. The online version of this article has been corrected. ■

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