Pediatr Pol 2025; 100 (4): 387-393
DOI: https://doi.org/10.5114/polp.2025.156651
Submitted: 04.05.2025, Accepted: 28.07.2025, Published: 17.11.2025

PEDIATRIA Polska
PISMO POLSKIEGO TOWARZYSTWA PEDIATRYCZNEGO
Polish Journal of PAEDIATRICS
OFFICIAL JOURNAL OF POLISH SOCIETY OF PAEDIATRICS

CASE REPORT

Vitamin D toxicity with acute kidney injury in siblings – a case report and literature review

Tomasz Szawłoga[1], Aleksandra Sobieszczańska-Droździel[2]

[1]Students' Scientific Group, Department of Pediatric Nephrology, Medical University of Lublin, Lublin, Poland
[2]Department of Pediatric Nephrology, Medical University of Lublin, Lublin, Poland

ABSTRACT

Vitamin D toxicity is a significant and increasingly diagnosed clinical issue. Despite clearly defined guidelinesw for the prevention and treatment of its deficiency, a growing trend of unsupervised high-dose supplementation is observed. We present two brothers who took 10,000 IU of cholecalciferol daily for over a month. One developed nonspecific symptoms of severe hypercalcemia (4.07 mmol/l) and acute kidney injury (eGFR 63), with serum 25(OH)D of 610 ng/ml. The other patient remained nearly asymptomatic despite a markedly elevated 25(OH)D level (501 ng/ml) and hypercalcemia (3.13 mmol/l). The applied treatment – glucocorticoids, forced diuresis, and electrolyte supplementation – caused normalization of calcemia and renal function. The presented case illustrates the broad spectrum of clinical manifestations associated with vitamin D toxicity and underscores the need to raise public awareness of the dangers linked to its excessive supplementation. It also emphasizes the importance of medical supervision when using vitamin D, particularly in children.

KEY WORDS:
acute kidney injury, hypercalcemia, vitamin D.

INTRODUCTION

Vitamin D is a fat-soluble secosteroid synthesized in human skin as vitamin D_3 (cholecalciferol) upon exposure to sunlight ultraviolet B radiation [1]. Subsequently, the liver and kidneys metabolize vitamin D_3 into circulating calcifediol (25(OH)D) and the active hormonal form, calcitriol (1,25(OH)2D). Notably, 1,25(OH)2D can also be synthesized in various tissues, such as the immune system, parathyroid glands, skin, intestinal epithelium, breast, and prostate, where it functions through autocrine and paracrine signaling pathways. The renal synthesis of 1,25(OH)2D is tightly regulated by parathyroid hormone (PTH), fibroblast growth factor 23, extracellular calcium, and phosphate concentrations. These factors modulate the activity of the renal enzyme 1α-hydroxylase, which catalyzes the conversion of calcifediol to its active form [2].

The primary role of 1,25(OH)2D is the regulation of calcium and phosphorus homeostasis and bone metabolism. Recently, its pleiotropic effects have been increasingly recognized, with significant roles in cell proliferation, oxidative stress reduction, xenobiotic detoxification, neuroprotection, cardiovascular protection, immunomodulation, antimicrobial defense, anti-inflammatory, and anticancer activities. A key benefit of calcitriol is its induction of α-klotho, a kidney-derived hormone that protects against age-related problems such as skin atrophy, hyperphosphatemia, osteopenia, endothelial dysfunction, cognitive decline, hearing impairment, and neurodegenerative disorders [3, 4]. Consequently, vitamin D deficiency may also manifest as symptoms including fatigue, muscle pain, numbness, and, in severe cases, convulsions [3]. The optimal serum concentration of 25(OH)D is regarded as ranging 30–50 ng/ml [5].

ADDRESS FOR CORRESPONDENCE:
Tomasz Szawłoga, Students' Scientific Group, Department of Pediatric Nephrology, Medical University of Lublin, Lublin, Poland, e-mail: tomasz.w.szawloga@gmail.com

Current studies have indicated that during late winter and spring in northern Poland, nearly 90% of 5,775 evaluated adults had 25(OH)D levels below 30 ng/ml [6]. While supplementation is recommended, large epidemiological studies have shown a reverse J-shaped relationship between 25(OH)D levels and all-cause mortality, with concentrations exceeding 48 ng/ml associated with an increased risk [7]. Due to the uncertainty regarding the benefits of higher 25(OH)D levels, it is advisable to avoid concentrations above 50 ng/ml in children [7]. Serum levels exceeding 100 ng/ml are considered toxic [8].

The tolerable maximal daily intake of vitamin D varies by age, ranging from 400 IU for infants to 2,000 IU for children and adolescents [5]. In vitamin D supplementation, dietary intake from milk-based formulas should be considered [5]. Severe vitamin D toxicity is rare and typically results from hypersensitivity or inappropriate supplementation with doses exceeding 10,000 IU/day [8, 9]. Symptoms of excess vitamin D intake, caused by hypercalcemia, are nonspecific and may include abdominal pain, vomiting, dehydration, constipation, depression, confusion, cardiac arrhythmias, nephrocalcinosis, and, rarely, acute kidney injury (AKI) [8, 10].

In recent years, the widespread availability of over-the-counter vitamin D supplements, along with extensive marketing, has led to an increased trend of high-dose vitamin D supplementation for purported health benefits. This report presents the case of two brothers who, influenced by this trend, were administered 10,000 IU of vitamin D_3 daily by their parents for over a month.

CASE REPORT

PATIENT 1

Clinical presentation

A 12-year-old boy was admitted to the Department of Pediatric Nephrology with AKI accompanied by hypercalcemia. Before admission, he had been hospitalized in the Department of Infectious Diseases due to persistent vomiting for about three weeks unrelated to meals. Tests for rotavirus, adenovirus, and norovirus were negative, and a head computed tomography scan showed no abnormalities. According to parental report, the patient had also experienced reduced appetite, generalized weakness, and polyuria a few days before admission.

Further history revealed that the patient had been receiving 10,000 IU of cholecalciferol daily for approximately one month, initiated by the parents on the advice of friends and without medical supervision.

Diagnostics

Initial laboratory findings revealed elevated serum creatinine (eGFR 63 ml/min/1.73 m² estimated using the Schwartz formula), severe hypercalcemia and hypercalciuria, hyperuricemia, hypoparathyroidism, and extremely elevated levels of vitamin D metabolites (Table 1).

Electrocardiography showed nonspecific abnormalities in ventricular repolarization and a delay in intraventricular conduction.

TABLE 1. Follow-up of selected laboratory results in Patient 1

Parameters	Reference range	Admission (day 0)	Discharge (day 17)	Follow- up (day 35)	Follow- up (day 98)	Follow- up (day 201)
Blood						
Creatinine [mg/dl]	0.53–0.79	**1.36**	0.76	0.67	0.69	0.72
Urea [mg/dl]	15–36	**41.8**	36.1	23.8	21	**39**
Uric acid [mg/dl]	1.8–5.4	**7.97**	**6.92**	**6.67**	4.8	**6.3**
Calcium [mmol/l]	2.2–2.7	**4.07**	2.58	2.53	2.6	2.65
Phosphate [mmol/l]	1.05–1.85	1.53	1.25	1.11	1.41	1.51
Parathormone [pg/dl]	10–65	**5.21**	–	12.87	32.97	22.65
25(OH)D_3 [ng/dl]	20–60	**610**	**337.2**	**275.2**	**140**	**89.80**
1,25(OH)D_3 [pg/dl]	19.9–79.3	**624**	–	**84.10**	**101**	**104**
Urine						
Calcium excretion [mg/kg/24 h]	1–4	**12.51**	–	3.83	1.94	–
Phosphate excretion [mg/kg/24 h]	15–20	19.8	**26.89**	18.22	**7.64**	–
Uric acid excretion [mg/kg/24 h]	5–15	12.91	14.11	11.29	10.43	–
Protein excretion [mg/24 h]	0–140	0	–	–	–	–

Values beyond the reference range are highlighted in bold font.

TABLE 2. Follow-up of selected laboratory results in Patient 2

Parameters	Reference range	Admission (day 0)	Discharge (day 11)	Follow- up (day 34)	Follow- up (day 97)	Follow-up (day 200)
Blood						
Creatinine [mg/dl]	0.39–0.73	**0.9**	0.68	0.58	0.61	0.56
Urea [mg/dl]	15–36	**37.3**	34.2	15.1	20	25
Uric acid [mg/dl]	1.8–5.4	**6.77**	5.33	**5.51**	4.3	3.6
Calcium [mmol/l]	2.2–2.7	**3.13**	2.68	2.52	2.48	2.6
Phosphate [mmol/l]	1.05–1.85	1.31	1.38	1.35	1.69	1.63
Parathormone [pg/dl]	10–65	**1.2**	–	**8.3**	18.02	14.69
25(OH)D3 [ng/dl]	20–60	**501**	**334.4**	**285.2**	**121.2**	**98.20**
1,25(OH)D3 [pg/dl]	19.9–79.3	**173**	–	75.3	73.4	61.9
Urine						
Calcium excretion [mg/kg/24 h]	1–4	**11.58**	**12.45**	**7.04**	**2.65**	–
Phosphate excretion [mg//kg/24 h]	15–20	**31.71**	**21.1**	**24.3**	**9.14**	–
Uric acid excretion [mg/kg/24 h]	5–15	**20.14**	**16.94**	**17.56**	8.65	–
Protein excretion [mg/24 h]	0–140	**288.1**	–	–	–	–

Values beyond the reference range are highlighted in bold font.

Treatment

The patient was treated with forced diuresis using furosemide, oral prednisone at a dose of 1 mg/kg/day, and supplementation with potassium and magnesium. A low-calcium diet was introduced.

Follow-up

After 17 days of therapy, serum creatinine and calcium levels normalized, and the patient was discharged without symptoms.

Subsequent outpatient follow-up showed normalization of renal function and calcium levels, though both 1,25(OH)D and 25(OH)D remained elevated (Table 1).

Following discharge, the patient experienced several episodes of chest pain. A thorough evaluation, including ECG, echocardiography, 24-hour Holter monitoring, ambulatory blood pressure monitoring, spirometry, and exercise testing, was conducted. All results were within normal limits except for a prominent tendinous chord in the left ventricle noted on echocardiography. No chest pain or ECG abnormalities occurred during stress testing.

PATIENT 2

Clinical presentation

A 10-year-old boy, the younger sibling of Patient 1, was admitted to the Department of Pediatric Nephrology following identification of hypercalcemia and borderline serum creatinine during outpatient evaluation, performed due to his brother's diagnosis. Apart from mild general weakness and apathy, the patient did not report any other complaints despite receiving the same daily dose of cholecalciferol.

Diagnostics

Laboratory tests on admission revealed hypercalcemia, borderline elevated serum creatinine (eGFR 89.7 ml/min/1.73 m^2), hyperuricemia, suppressed PTH, elevated 1,25(OH)D and 25(OH)D levels, and hypercalciuria (Table 2).

Treatment

Management followed the same protocol as with Patient 1: forced diuresis, prednisone therapy, electrolyte supplementation, and a low-calcium diet.

FOLLOW-UP

After 10 days of treatment, calcium and creatinine levels returned to normal, with a concurrent decline in vitamin D metabolite levels. At the first follow-up visit, three weeks post-discharge, calcium and renal function remained normal, 1,25(OH)D normalized, with a slight reduction of PTH. However, 25(OH)D remained elevated. Subsequent follow-up confirmed normalization of all biochemical parameters, with a gradual decline in 25(OH)D levels (Table 2).

DISCUSSION

Acute kidney injury is a rare complication of vitamin D toxicity. Graidis *et al.* in their systematic review of 74 studies analyzed the relationship between AKI and vi-

tamin D. Notably, between 1984 and 2019, only 115 cases of AKI resulting from hypervitaminosis D were documented. Most patients had been administered extremely high cumulative doses of vitamin D, ranging 240,000–4,500,000 IU, often due to manufacturing errors in over-the-counter formulations, dosing mistakes, or intentional ingestion of products purchased to improve health [10, 11].

However, the recent systematic review and meta-analysis conducted by Brustad *et al*. [12], encompassing 32 randomized clinical trials with a total of 8,400 pediatric participants, demonstrated that high-dose vitamin D – administered either daily or as a bolus – was not linked to an increased risk of serious adverse effects. Notably, a daily dose of 10,000 IU was well tolerated in children aged 0–6 years.

In the presented patients, intoxication was caused by a dosing error. Although the manufacturer recommended a dose of 10,000 IU of their vitamin D supplement every 10 days, the patients were administered this dose daily. The parents had not consulted a medical professional and were unaware of current clinical recommendations regarding vitamin D supplementation. Patients with vitamin D toxicity usually present with hypercalcemia, suppressed PTH levels, elevated serum 25(OH)D levels exceeding 150 ng/ml, and normal or increased serum 1,25(OH)2D levels. Notably, both presented patients demonstrated the full spectrum of these biochemical abnormalities (Tables 1, 2) [11, 13].

Furthermore, similarly to the symptoms observed in our sibling, serum calcium and vitamin D metabolite levels may vary among individuals [11]. The spectrum of patients who may require increased vitamin D_3 intake is broad, encompassing musculoskeletal disorders, connective tissue diseases, neurological conditions, as well as lifestyle- and metabolism-related disorders [5]. Individuals with higher body mass index often present with lower circulating levels of 25(OH)D, a phenomenon likely attributable to the augmented storage capacity of adipose tissue [14].

Interestingly, despite receiving identical doses of the same vitamin D supplement, Patient 1 exhibited a serum 25(OH)D level of 610 ng/ml, whereas Patient 2's level was 501 ng/ml. Similarly, serum calcium concentrations differed, with Patient 1 presenting 4.07 mmol/l compared to 3.13 mmol/l in his brother. The observed differences between the siblings may be attributable to individual variations in vitamin D metabolism and calcium homeostasis. Moreover, other factors, including compliance discrepancies, variability in absorption, and concurrent physiological conditions such as hydration status or baseline renal vulnerability, should be included.

Circulating levels of 25(OH)D can be influenced by genetic polymorphisms in genes involved in the synthesis and hydroxylation of vitamin D, as well as by variations in the vitamin D binding protein [11]. Notably, pathogenic variants in the *CYP24A1* gene are responsible not only for idiopathic infantile hypercalcemia (IIH) but also for hypercalcemia across the lifespan [15]. While IIH is extremely rare with an uncertain prevalence, 24-hydroxylase deficiency is estimated to occur in approximately 420 per 100,000 individuals [15]. Such cases are difficult to diagnose due to subtle clinical manifestations without additional triggers such as pregnancy or vitamin D supplementation [15, 16]. Although genetic sequencing was not performed in the presented siblings, mutations in the *CYP24A1* gene may be of potential relevance. However, generally, patients with *CYP24A1* mutations manifest clinical symptoms during infancy, with the majority of pediatric cases diagnosed within the first year of life. This is often associated with features of vitamin D toxicity despite adherence to standard supplementation. Moreover, such mutations are commonly linked to nephrolithiasis and nephrocalcinosis, neither of which was observed in our patients during follow-up assessments [15]. Therefore, genetic testing should be considered in patients presenting with symptoms of vitamin D toxicity despite appropriate supplementation.

Hypervitaminosis D significantly affects kidney function by inducing hypercalcemia, which was detected in both presented patients (Tables 1, 2).

Hypercalcemia induces nephrogenic diabetes insipidus and triggers hypovolemia, which in turn leads to prerenal AKI, establishing a vicious cycle that further exacerbates hypercalcemia. Additionally, hypercalcemia causes renal vasoconstriction, further compromising kidney function [17]. Hypovolemia leads to increased water reabsorption in the proximal tubules and the descending limbs of Henle's loops. However, calcium and phosphate are not adequately reabsorbed, as in the presented patients in whom severe hypercalciuria and hyperphosphaturia were found (Tables 1, 2) [18]. This leads to the deposition of calcium in the renal medulla, aggravating urine concentration ability, which is part of the pathomechanism of nephrogenic diabetes insipidus [19]. Both hypercalcemia and hypercalciuria, responsible for the deposition of calcium in renal parenchyma, may lead to nephrocalcinosis and consequently to the development of chronic kidney disease [19]. Although the presented patients did not exhibit imaging-visible nephrocalcinosis (Figure 1), its microscopic forms cannot be excluded.

Lin *et al*. [20], in their study of vitamin D overdose as a cause of nephrocalcinosis, found that only 4 out of 44 pediatric patients developed AKI. Notably, there were no statistically significant differences in serum creatinine levels or the incidence of AKI between patients with and without nephrocalcinosis.

Interestingly, the half-life of serum 25(OH)D is approximately 15–25 days, whereas the calculated half-life of serum 25(OH)D following the intake of excessive vitamin D can extend up to 82 days [13, 21]. Consequently, normalizing of 25(OH)D concentrations may require many months of monitoring and appropriate management, as it was in the reported cases (Tables 1, 2, Figure 2).

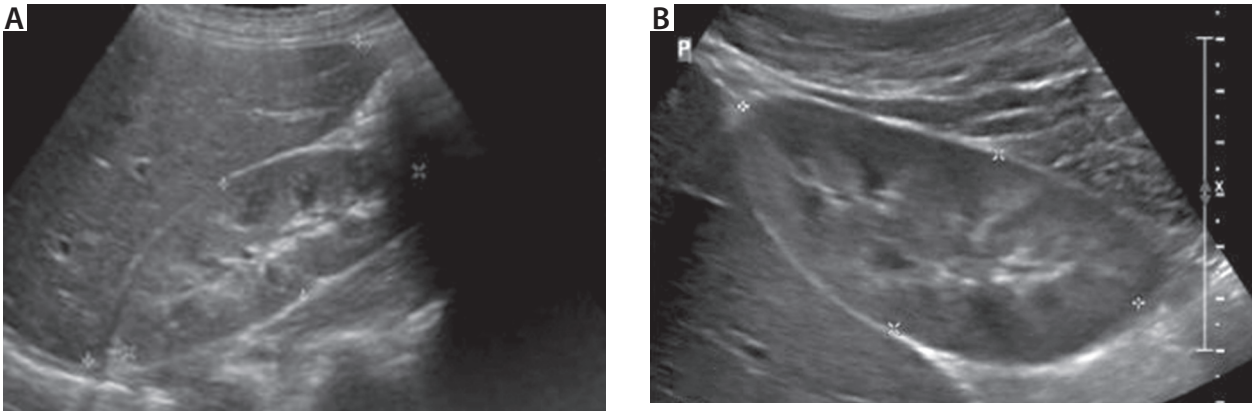

FIGURE 1. Normal renal ultrasound of the presented patients. **A**) Patient 1. **B**) Patient 2 (Department of Pediatric Radiology, Medical University of Lublin)

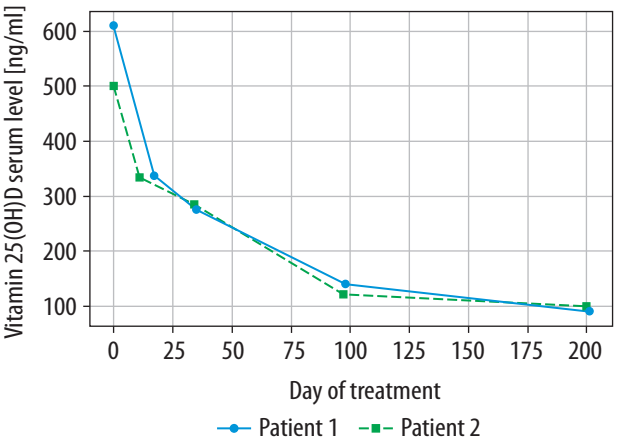

FIGURE 2. Follow-up of the 25(OH)D serum level in the presented patients

In both brothers, an uneven decline in 25(OH)D levels was observed, with the most significant reduction occurring during the first month of treatment (Figure 2). This was likely the result of the comprehensive medical management provided during inpatient care.

Vitamin D intoxication not only affects renal function. Hypercalcemia, its key consequence, impacts the cardiac conduction system, leading to potential ECG abnormalities. These may include ST-segment elevation, biphasic T waves, prominent U waves, increased J wave amplitude, and QT interval shortening can be observed [22]. ECG of Patient 1 showed only a shorter QT interval, which is the most common finding [22]. Patient 2 with milder hypercalcemia did not present any ECG abnormalities.

Another potential modulator of vitamin D metabolism is uric acid. Hyperuricemia secondary to AKI may inhibit 1α-hydroxylase activity, while elevated vitamin D levels are associated with a lower risk of hyperuricemia [23–25]. Although both patients had elevated or borderline uric acid levels, this interaction does not appear clinically relevant in the presented cases (Tables 1, 2).

The first line of vitamin D overdose therapy is withdrawal of its supplements and normalization of serum calcium. Intravenous fluids correct dehydration, consequently increasing glomerular filtration rate and calcium excretion [11]. Loop diuretics e.g. furosemide, can be used to promote calcium excretion, though their prolonged use requires supplementation of sodium, chloride, and potassium [11, 13]. Glucocorticoids decrease serum calcium levels by reducing renal calcium reabsorption and inhibiting the production of 1,25(OH)2D, thereby reducing intestinal calcium absorption [11, 13]. In adults, a dose of 100 mg/24 h of hydrocortisone or its equivalent normalizes calcium levels within several days. In the presented sibling, serum calcium levels normalized after 17 days in Patient 1 and 10 days in Patient 2. As vitamin D increases the absorption of dietary calcium, a low-calcium diet is recommended as part of the management strategy [13].

Additional approaches to reduce hypercalcemia include the use of calcitonin, bisphosphonates, and azoles [15]. Remarkably, bone resorption is elevated in vitamin D intoxication; therefore, antiresorptive therapy can effectively reduce serum calcium levels [11]. Since the onset of action of glucocorticoids is expected within 24–72 hours, they can be combined with calcitonin to achieve a more rapid reduction in serum calcium. A dose of 2–4 IU/kg of calcitonin administered every 6–12 hours can quickly lower serum calcium levels. However, its therapeutic value is limited due to the potential development of tachyphylaxis after several days of use and the risk of anaphylactic shock and tissue calcium deposition [11, 13]. Guidelines published in 2018 suggest that low doses of pamidronate or zoledronate can be considered in hypercalcemia resistant to dietary management and intravenous hydration [26]. Azoles non-specifically reduce the production of 1,25(OH)2D by inhibiting cytochrome P450 CYP27B1; however, with prolonged use, they may also inhibit other critical cytochrome P450 enzymes [13].

In patients with *CYP24A1* mutations, hypercalcemia may be resistant to steroids; however, that was not

the case in the presented patients. Due to potential adverse effects, including hepatotoxicity associated with azoles and hypocalcemia following bisphosphonate therapy, these treatments should be considered only when conventional approaches fail to adequately reduce hypercalcemia [27, 28].

In patients with refractory vitamin D-induced hypercalcemia, high-cut-off hemodialysis and total plasma exchange with albumin may provide an effective intervention. However, hemodialysis is limited to lowering plasma calcium levels and does not eliminate vitamin D_3 metabolites, as the vitamin D_3 bound to vitamin D binding protein is too large to pass through the dialysis membrane [29].

Due to the rapid normalization of calcemia and renal function along with a favorable response to hydration and prednisone, renal replacement therapy and alternative treatment options were not considered in the presented patients.

Importantly, effective management of vitamin D intoxication, particularly regarding the early detection of patients at risk for AKI, is crucial for optimal outcomes. Recent studies have highlighted biomarkers such as kidney injury molecule-1 and cystatin C as promising tools for the early detection and prediction of AKI development in pediatric patients, thereby facilitating timely diagnosis and intervention [30].

CONCLUSIONS

Although vitamin D is generally regarded as a safe fat-soluble vitamin, excessive intake can lead to a range of complications, including AKI. With growing public awareness of its health benefits, the use of over-the-counter vitamin D supplements – often without medical supervision – has become increasingly common. This trend may elevate the risk of overdose and toxicity, particularly in susceptible individuals.

Supplementation should be provided with medical oversight and follow established guidelines, with appropriate dosing and regular monitoring of serum calcium and 25(OH)D levels. Patients ought to be informed about the potential risks of vitamin D overdose, including the possibility of toxicity and related health complications.

In children presenting with nonspecific symptoms accompanied by AKI, hypercalcemia, or hypercalciuria, vitamin D toxicity should be considered as part of the differential diagnosis.

DISCLOSURES

1. Institutional review board statement: Not applicable.
2. Assistance with the article: The written informed consent to publish the cases was obtained from the mother of the affected patients.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

REFERENCES

1. Delrue C, Speeckaert MM. Vitamin D and vitamin D-binding protein in health and disease. Int J Mol Sci 2023; 24: 4642.
2. Bikle DD. Vitamin D: production, metabolism and mechanisms of action. In: Feingold KR, Anawalt B, Blackman MR (eds). Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000. Available from: https://www.ncbi.nlm.nih.gov/books/NBK278935/ (accessed from: 23.10.2024).
3. Zhang X, Liu Z, Xia L, Gao J, Xu F, Chen H, et al. Clinical features of vitamin D deficiency in children: a retrospective analysis. J Steroid Biochem Mol Biol 2020; 196: 105491.
4. Gil Á, Plaza-Diaz J, Mesa MD. Vitamin D: classic and novel actions. Ann Nutr Metab 2018; 72: 87-95.
5. Płudowski P, Kos-Kudła B, Walczak M, Fal A, Zozulińska-Ziółkiewicz D, Sieroszewski P, et al. Guidelines for preventing and treating vitamin D deficiency: a 2023 update in Poland. Nutrients 2023; 15: 695.
6. Płudowski P, Ducki C, Konstantynowicz J, Maciej Jaworski. Vitamin D status in Poland. Pol Arch Med Wewn 2016; 126: 530-539.
7. Wan M, Patel J, Rait G, Shroff R. Hypervitaminosis D and nephrocalcinosis: too much of a good thing? Pediatr Nephrol 2022; 37: 2225-2229.
8. Shroff R, Wan M, Nagler EV, Bakkaloglu S, Fischer DC, Bishop N, et al. Clinical practice recommendations for native vitamin D therapy in children with chronic kidney disease stages 2–5 and on dialysis. Nephrol Dial Transplant 2017; 32: 1098-1113.
9. Braegger C, Campoy C, Colomb V, Decsi T, Domellof M, Fewtrell M, et al. Vitamin D in the healthy European paediatric population. J Pediatr Gastroenterol Nutr 2013; 56: 692-701.
10. Graidis S, Papavramidis TS, Papaioannou M. Vitamin D and acute kidney injury: a two-way causality relation and a predictive, prognostic, and therapeutic role of vitamin D. Front Nutr 2021; 7: 630951.
11. Vogiatzi MG, Jacobson-Dickman E, DeBoer MD. Vitamin D supplementation and risk of toxicity in pediatrics: a review of current literature. J Clin Endocrinol Metab 2014; 99: 1132-1141.
12. Brustad N, Yousef S, Stokholm J, Bønnelykke K, Bisgaard H, Chawes BL. Safety of high-dose vitamin D supplementation among children aged 0 to 6 years: a systematic review and meta-analysis. JAMA Netw Open 2022; 5: e227410.
13. Marcinowska-Suchowierska E, Kupisz-Urbańska M, Łukaszkiewicz J, Płudowski P, Jones G. Vitamin D toxicity-a clinical perspective. Front Endocrinol (Lausanne) 2018; 9: 550.
14. Tobias DK, Luttmann-Gibson H, Mora S, Danik J, Bubes V, Copeland T, et al. Association of body weight with response to vitamin D supplementation and metabolism. JAMA Netw Open 2023; 6: e2250681.
15. Cappellani D, Brancatella A, Morganti R, Borsari S, Baldinotti F, Caligo MA, et al. Hypercalcemia due to CYP24A1 mutations: a systematic descriptive review. Eur J Endocrinol 2021; 186: 137-149.
16. Silvestre C, Aragües JM, Bugalho MJ, Jones G, Kaufmann M. Idiopathic infantile hypercalcemia presenting in childhood but diagnosed in adulthood. AACE Clin Case Rep 2018; 4: 256-262.
17. Deshpandey RP, Kumar HSA, Mathew S. Dyselectrolytemias and acidbase disorder in acute kidney injury. Clin Queries Nephrol 2012; 1: 70-75.
18. Markowitz GS, Perazella MA. Acute phosphate nephropathy. Kidney Int 2009; 76: 1027-1034.
19. De Francesco Daher E, Mesquita Martiniano LV, Lopes Lima LL, Filho NCVL, de Oliveira Souza LE, Palácio Duarte Fernandes PH, et al. Acute kidney injury due to excessive and prolonged intramuscular injection of veterinary supplements containing vitamins A, D and E: a series of 16 cases. Nefrología 2017; 37: 61-67.
20. Lin TH, Lu HJ, Lin CH, Lee MD, Chang BPH, Lin CC, et al. Nephrocalcinosis in children who received high-dose vitamin D. Pediatr Nephrol 2022; 37: 2471-2478.

21. Martinaityte I, Kamycheva E, Didriksen A, Jakobsen J, Jorde R. Vitamin D stored in fat tissue during a 5-year intervention affects serum 25-hydroxyvitamin d levels the following year. J Clin Endocrinol Metabol 2017; 102: 3731-3738.
22. Chorin E, Rosso R, Viskin S. Electrocardiographic manifestations of calcium abnormalities. Ann Noninvasive Electrocardiol 2016; 21: 7-9.
23. Ejaz AA, Mu W, Kang DH, Roncal C, Sautin YY, Henderson G, et al. Could uric acid have a role in acute renal failure? Clin J Am Soc Nephrol 2007; 2: 16-21.
24. 24. Isnuwardana R, Bijukchhe S, Thadanipon K, Ingsathit A, Thakkinstian A. Association between vitamin D and uric acid in adults: a systematic review and meta-analysis. Horm Metab Res 2020; 52: 732-741.
25. Zhang YY, Qiu HB, Tian JW. Association between vitamin D and hyperuricemia among adults in the United States. Front Nutr 2020; 7: 592777.
26. Simm PJ, Biggin A, Zacharin MR, Rodda CP, Tham E, Siafarikas A, et al. Consensus guidelines on the use of bisphosphonate therapy in children and adolescents. J Paediatr Child Health 2018; 54: 223-233.
27. Sayers J, Hynes AM, Srivastava S, Dowen F, Quinton R, Datta HK, et al. Successful treatment of hypercalcaemia associated with a CYP24A1 mutation with fluconazole. Clin Kidney J 2015; 8: 453-455.
28. Kilci F, Jones JH, Çizmecioğlu-Jones FM. Successful management of severe hypercalcemia with zoledronic acid: a report of two pediatric cases. J Clin Res Pediatr Endocrinol 2024; 16: 224-228.
29. Heister DJ, Bohnert BN, Heyne N, Birkenfeld AL, Artunc F. Two cases of severe vitamin D3 intoxication treated with therapeutic plasma exchange and high cut-off hemodialysis. J Nephrol 2023; 36: 1461-1467.
30. Hasan HR, Al-Fatlawi ACY, Al-Obaidy QM. Evaluation of the levels of kidney injury molecule-1 and cystatin C as early biomarkers for prediction of acute kidney injury complications in paediatric male patients – a case-control study. Pediatr Pol 2025; 100: 38-51.