¤ПЕДИАТРИЯДА КОМПЛЕКС ЁНДАШУВ: ТАЪЛИМ ТЕХНОЛОГИЯЛАРИ, ДИАГНОСТИКА АЛГОРИТМЛАРИ ВА ДАВОЛАШ

мавзусидаги илмий-амалий конференция

ТЕЗИЗЛАР ТЎПЛАМИ

30 май 2025 йил, Тошкент

Ҳурматли _____!

Сизни 2025-йил 30-май куни ***«Педиатрияда комплекс ёндашув: таълим технологиялари, диагностика алгоритмлари ва даволаш»*** мавзусидаги илмий-амалий конференцияда иштирок этишга таклиф этади.

Иштирокчиларни рўйхатга олиш 9:00 да бошланади.

Конференциянинг очилиши - 9:30

КОНФЕРЕНЦИЯНИНГ АСОСИЙ ТАДБИРЛАРИ ДАСТУРИ

Вақти	**Тадбир**	**Ўтказилиш жойи**
9.00	Иштирокчиларни рўйхатга олиш	Асосий бино фойеси (Боғишамол 223)
9.30	Ялпи мажлис	Мажлислар зали (6-маъруза зали)
12.30	Танаффус – Кофе брейк	
13.30	"Педиатрия" секцияси йиғилиши	6-маъруза зали
13.30	"Хирургия" секцияси йиғилиши	Ахборот-ресурс маркази зали (АРМ биноси)
13.30	ГЭОТАР-Медиа кўмагида симуляцион мастер-класслар ва ёш олимлар танлови	Симуляцион марказ зали ва хоналари (Симуляцион марказ биноси)
16.30	Конференция якунларини чиқариш ва конференция резолюциясини имзолаш	6-маъруза зали

kidney failure and improve prognosis for patients. The inclusion of hearing aids and regular ophthalmological monitoring helps reduce the impact of extrarenal manifestations on patients' quality of life. Early detection and timely therapy greatly increase the effectiveness of treatment and improve long-term outcomes in patients with Alport syndrome.

ROLE OF FGF23 IN THE PATHOGENESIS OF MINERAL AND BONE DISORDERS IN CHRONIC KIDNEY DISEASE

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Relevance. Mineral and bone disorders in chronic kidney disease (CKD-MBD) are among the most common and severe complications in patients with renal insufficiency. Fibroblast Growth Factor 23 (FGF23) is an osteocytic hormone that regulates phosphate metabolism and vitamin D homeostasis. Its levels begin to rise in the early stages of CKD and have systemic effects on mineral balance. Excessive FGF23 production disrupts bone mineralization, exacerbates hypophosphatemia, reduces the synthesis of active vitamin D, and is associated with an increased risk of cardiovascular complications. Studying the role of FGF23 in the pathogenesis of CKD-MBD is of high clinical importance for diagnosis, monitoring, and therapeutic strategy selection.

Research Goal. To assess the significance of FGF23 in the development of mineral and bone disorders in CKD, establish its clinical and laboratory correlations, and identify potential directions for targeted therapy.

Materials and Methods. Analysis of scientific publications from the past 10 years, including clinical studies, meta-analyses, and reviews on FGF23 and CKD. Evaluation of phosphate-calcium metabolism indicators, levels of $1,25(OH)_2D$, parathyroid hormone (PTH), and FGF23 in patients at various stages of CKD. Comparison of clinical and laboratory features in patients with elevated FGF23 levels. Data sources included PubMed, Scopus, Web of Science, and eLIBRARY databases.

Results. The analysis revealed that FGF23 levels increase as early as CKD stages 2–3, well before changes in serum phosphorus and PTH levels. This elevation is linked to several key mechanisms. In CKD, the kidneys lose the ability to efficiently excrete phosphates, leading to their accumulation in the blood. In response, bone tissue produces more FGF23 to stimulate phosphate excretion by the kidneys. FGF23 also suppresses the activity of 1-alpha-hydroxylase in the kidneys, reducing the production of active vitamin D ($1,25(OH)_2D$). This impairs the absorption of calcium and phosphates in the intestines, worsening hypophosphatemia and hypocalcemia. In response to decreased blood calcium levels, the body increases PTH production, which promotes phosphate excretion and disrupts phosphate metabolism, further stimulating FGF23 production. FGF23 inhibits sodium-phosphate cotransporters in the proximal renal tubules, enhancing phosphate excretion and lowering blood phosphate levels. These changes lead to impaired bone mineralization, manifesting as osteopenia,

osteomalacia, and an increased risk of fractures. Elevated FGF23 levels are also associated with cardiotoxic effects, particularly left ventricular hypertrophy and increased mortality in CKD patients. In dialysis patients, higher FGF23 levels correlate with the severity of secondary hyperparathyroidism and the need for aggressive therapy. FGF23 is considered not only a biomarker but also a therapeutic target: approaches aimed at reducing its production or blocking its receptors (e.g., anti-FGF23 antibodies) are promising, especially in severe phosphate metabolism disorders.

Conclusion. FGF23 plays a key role in the pathogenesis of mineral and bone disorders in CKD, initiating a cascade of interconnected changes—hypophosphatemia, active vitamin D deficiency, secondary hyperparathyroidism, and impaired bone mineralization. Elevated FGF23 levels serve as an early biomarker of CKD-MBD and contribute to the progression of cardiovascular and skeletal complications. Assessing and controlling FGF23 may be crucial steps in the individualized management of patients with chronic kidney disease.

STREPTOCOCCAL SEPSIS IN YOUNG CHILDREN: IDENTIFYING OPTIMAL APPROACHES FOR EARLY DIAGNOSIS AND TREATMENT

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Relevance. Streptococcal sepsis in young children is a dangerous infection associated with the immaturity of the immune system. The main routes of infection include contaminated birth canals and damaged skin and mucosal barriers. Delayed treatment can lead to severe complications such as disseminated intravascular coagulation (DIC), multiple organ failure, septic shock, and increased risk of death.

Research Objective. To study the pathogenesis, clinical manifestations, and prognostic factors of streptococcal sepsis in children in order to identify optimal approaches for early diagnosis and therapy, as well as to develop recommendations for prevention.

Materials and Methods. The study included children aged 0 to 3 years with confirmed streptococcal sepsis, who were treated in pediatric and neonatal departments. The analysis was based on medical history data, laboratory tests, and instrumental diagnostic methods.Methods of Investigation:Complete blood count (CBC): Leukocytosis (WBC >15×10⁹/L) or leukopenia (<4×10⁹/L in severe sepsis) Neutrophilia with toxic granulation, Elevated erythrocyte sedimentation rate (ESR >30 mm/h), Thrombocytopenia (<100×10⁹/L in DIC),Anemia (hemoglobin <90 g/L). Biochemical blood analysis: Elevated C-reactive protein (>50 mg/L, often >100 mg/L), High procalcitonin levels (>2 ng/mL indicating systemic bacterial infection). Bacteriological studies: Positive blood culture for β-hemolytic group B streptococcus (Streptococcus agalactiae) or Streptococcus pyogenes, CSF culture (in case of meningitis) may reveal

СОДЕРЖАНИЕ