TECOmedical Group

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Technical Information

FGF-23 Intact, Humain

Facteur de croissance du Fibroblaste 23

Cat. Nr:	CY-4000
Test :	96
Méthode :	**ELISA**
Gamme:	8 - 800 pg/ml
Sensibilité:	3.0 pg/ml
Temps d'incubation :	3.5 heures
Vol.échantillon:	50 µl
Echantillon:	Sérum / Plasma (EDTA & **Héparine**)
Précautions:	Il est préférable de prélever l'échantillon le matin après un jeûne de 12 heures. Le FGF-23 Intact est très instable. Le recueil, le dosage et le stockage des échantillons doivent être effectués rapidement. Conserver les échantillons congelés à -20°C. Eviter les congélations/décongélations répétées.
Valeurs Normales :	10 - 50 pg/ml
Espèces:	Humaine, Rat, Souris

Intérêt clinique:

Le FGF-23, Fibroblast Growth Factor 23, produit par les précurseurs des ostéoblastes, est un régulateur important du métabolisme du phosphate et de la vitamine D.
Le phosphate joue un rôle important dans la stabilité des os du squelette et dans le métabolisme énergétique ainsi que dans la synthèse du DNA et dans les cascades de signaux intracellulaires.

Le FGF-23, en combinaison avec le facteur Klotho, inhibe la réabsorption du phosphate par les cellules des tubules rénaux proximaux par l'intermédiaire des récepteurs du FGF-23 (augmentation des pertes en phosphate, diminution du phosphate sanguin) et diminue la synthèse du calcitriol en inhibant la alpha-1-hydroxylase.

FGF-23 in Osteology

FGF-23 is involved in a variety of diseases accompanied by hypophosphatemia caused by renal phosphate loss. Moreover, the clinical pictures show distinctly reduced calcitriol synthesis and osteomalacia or vitamin D resistant rickets.

1. Tumor-induced osteomalacia / hypophosphatemia (TIO; paraneoplastic overexpression of FGF-23)
2. Autosomal dominant hypophosphatemic rickets (ADHR; due to mutation in FGF-23 protein, FGF-23 cannot be inactivated by endopeptidases)
3. X-linked hypophosphatemia (XHL, mutation in degrading enzyme (PHEX))
4. Craniofacial dysplasia with hypophosphatemia (increased FGF-23 levels caused by mutation of FGF receptor 1)
5. Fibrous dysplasia of bone (overproduction of FGF-23 due to mutation in G-protein subunit G5a/GNAS1)

FGF-23 in Nephrology

1. Elevated FGF-23 values are seen in chronic renal insufficiency and correlate negatively with GFR.
2. Increased serum FGF-23 levels may help maintain normophosphatemia in early chronic renal insufficiency until creatinine clearance is reduced to approximately 30 mL/min and hyperphosphatemia develops due to exhausted regulatory mechanisms and concurrently decreased calcitriol and sHPT.
3. Monitoring of FGF-23 and serum phosphate in early chronic renal insufficiency allows, if necessary, to institute phosphate reduction therapy at an earlier stage.
4. Creatinine levels within the normal range do not exclude disorders of phosphate metabolism.
5. In the ArMoRR study published by Guitierrez et al. in August 2008, it was demonstrated that the FGF-23 level at the beginning of hemodialysis therapy may be seen as an independent risk marker. Patients showing FGF-23 levels within the highest range developed a 5.7fold higher risk of death within one year.

References

Guitierrez et al.: Fibroblast Growth Factor 23 and Mortality among Patients Undergoing Hemodialysis. N Eng J Med 2008; 359: 584-92

Chi-yuan Hsu: FGF-23 and Outcomes Research – When Physiology meets Epidemiology. N Engl J Med 2008; 359 6

Andreas L. Serra et al.: Phosphatemic Effect of Cinacalcet in Kidney Transplant Recipients With Persistent Hyperparathyroidism. American Journal of Kidney Diseases 2008

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