



Fibroblast growth factor 23 and cardiovascular disease in chronic kidney disease; new trends



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Cardiovascular disease is responsible for high mortality of patients suffering from chronic renal failure (1,2). One of the main explanations for the high rate of mortality due to cardiac disease is various conditions such as heart calcification, diastolic dysfunction, and sudden cardiac death in patients with hypertrophy of left ventricle. The risk of sudden heart death rises with progressive loss of renal function before dialysis (3,4). Around 25% of individuals on dialysis die of sudden heart death as a one hundred increased fold risk compared to the general population. There is a common idea that, the etiology of unexpected cardiac death was hypertrophy of left ventricle and also fibrosis of cardiac tissue. Generally, in chronic renal failure, other major risk factors for hypertrophy of left ventricle are volume overload, anemia and hypertension. However, aggressive correction of these abnormalities leads to regression of hypertrophy of left ventricle in below than 50% of individuals on dialysis, implying the presence of some other parameters specific to chronic renal failure that are responsible for high heart and vessels disease in these group (2-5). Furthermore, it is well documented that individuals with heart and vessel disease have a high risk of kidney disturbance. Additionally, vascular calcification, aberrant deposition of calcium-phosphorus salts in vascular walls, valves, and cardiac tissue, is commonly detected in various conditions such as aging process, diabetes mellitus and chronic renal failure. It is well-understood that vascular calcification can be an independent prognosticator of hear morbidity and mortality in chronic renal failure and end-stage renal disease too. Various factors such as enhanced serum fibroblast growth factor 23, calcification of vessels and hyperphosphatemia are factors related to chronic kidney disease – mineral bone disorder. These factors are also accompanied by cardiovascular risks and mortality (1-4). The raised heart and vessels disease associated with renal insufficiency is partly due to chronic kidney disease–mineral bone disorder (CKD-MBD) syndrome. Even more, recent investigations revealed that serum fibroblast growth factor 23 level predicts mortality and cardiovascular disease, particularly cardiac failure, in patients with chronic renal failure. Fibroblast growth factor 23, is delivered by osteocytes and osteoblasts, and

■ Implication for health policy/practice/research/medical education

Increased serum fibroblast growth factor 23 is capable of increasing intracellular calcium in cardiomyocytes which lead to rise incidence of sudden cardiac death.

■ **Keywords:** Calcium, Chronic kidney disease, Mineral bone disorder, Heart, Calcification, Fibroblast growth factor 23

it is an example of direct bone-kidney interaction or bone and parathyroid interaction in the condition of CKD-MBD (4-7). Levels of fibroblast growth factor 23 increases following mild kidney damage. This increment will extensively strengthen several fold during the late course of chronic renal failure. It has been detected that, the rise of FGF23, is owing to increased osteocyte secretion and also abolished catabolism by the damaged kidney. In fact, fibroblast growth factor 23 levels will increase before disturbances in phosphorus, parathormone or calcium. Thus it may be assumed as an earliest demonstrable biomarkers of the CKD-MBD. It has been detected that heart hypertrophy is the result of strengthen the levels of serum fibroblast growth factor 23 (2-6). Additionally, fibroblast growth factor 23 appears to have different indirect impacts on the extension of heart failure. It has been reported that serum fibroblast growth factor 23 levels were elevated with the induction of myocardial infarction independent of alterations in klotho and parathormone levels. Rise of serum fibroblast growth factor 23 serum levels decreased serum 1,25(OH) vitamin D levels by overwhelming the activation of 25(OH) vitamin D levels (1,4,6,7). It is also possible that aldosterone up-regulate reseals of fibroblast growth factor 23 (8). Moreover, raised serum fibroblast growth factor 23 values augment sodium resorption across increased expression of the Na–Cl co-transporter by the kidney. Hence, along with its effect on myocardium, fibroblast growth factor 23 has various impacts on hemodynamics that bring about to heart failure, comprising increases in sodium and expansion of plasma volume (1,2,8,9). Recently, it has been detected that increased serum fibroblast growth factor 23 is able to increase intracellular calcium in cardiomyocytes which lead to rise incidence of sudden cardiac death too. These

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findings regarding the effects of fibroblast growth factor 23 on CKD-MBD, parathyroid hormone and cardiac involvement imply a complex interaction which requires further investigations (4,8-10).

Author's contribution

AB wrote the manuscript lonely.

Conflicts of interest

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, misconduct, data fabrication, falsification, double publication or submission, redundancy) have been completely observed by the author.

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