

Analyzing the Effect of Intravenous Iron Therapy on Hemoglobin Values in Chronic Dialysis Patients with Anemia and Elevated Ferritin Levels Pediatric Patients with End-Stage Renal Disease Receiving Peritoneal Dialysis: Evaluation of Ultrasensitive C – Reactive Protein as a Cardiovascular Risk Marker

Abstract

The term “renal osteodystrophy” is used to describe the wide variety of skeletal deformities that affect people with chronic kidney disease. The two primary disorders are adynamic bone disease, which has low bone turnover and low levels of circulating PTH, and osteitis fibrosa, which is characterised by fast bone turnover, enhanced osteoclastic and osteoblastic activity, and high levels of circulating parathyroid hormone (PTH).

Renal osteodystrophy is primarily caused by the retention of phosphorus, decreased blood levels of calcitriol, lower levels of serum ionised calcium, decreased numbers of vitamin D receptors and calcium sensors in the parathyroid gland, and skeletal resistance to PTH's calcemic activity. The current strategy for treating renal osteodystrophy will be discussed in this review [1].

Keywords: Cardiovascular diseases • C-reactive protein • End-stage renal disease • Peritoneal dialysis

Introduction

Cardiovascular illness increases the risk of morbidity and death in patients with end-stage renal disease (ESRD). In patients with End-Stage Renal Disease (ESRD), adverse cardiac events occur at rates 30 times higher than in the general population, and at rates 700 times higher in patients receiving renal replacement therapy (hemodialysis or peritoneal dialysis), with a corresponding 10-year drop in life expectancy 10 years after diagnosis. The most common cardiovascular issues in children with ESRD are abnormalities of the left ventricle (first hypertrophic, then dilated cardiomyopathy), arrhythmias, pericardial issues (pericarditis and tamponade), and early atherosclerotic abnormalities, such as thickening of the carotid intima and endothelial dysfunction as determined by brachial artery flow [1].

According to the literature, these patients have a high prevalence of traditional cardiovascular risk factors like diabetes, high blood pressure, smoking, dyslipidemia, obesity, and sedentarism as well as kidney disease-specific risk factors like anaemia, secondary hyperparathyroidism, and hypervolemia. In addition, additional cardiovascular risk factors, such as inflammation and oxidative stress, have been discovered that encourage atherosclerosis in uremic patients [2].

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The use of Ultrasensitive C-reactive protein (USCRP) as a cardiovascular risk measure in adult patients with or without ESRD is a noteworthy new breakthrough. However, this marker has not yet been well evaluated in a juvenile ESRD group in Chile. The acute-phase protein known as C-reactive protein, which is largely released by hepatocytes in response to a variety of stimuli, is a marker for tissue damage, infection, or systemic inflammation. There is evidence that CRP is also created locally in inflamed cells (as happens with atherogenesis); this might affect the function of micro-vascular vasodilation by, for example, inhibiting the generation of nitric oxide synthase. Using conventional methods, readings between 10 and 40 mg/L are suggestive of a viral infection, while anything below 10 mg/L is deemed normal [3].

Researchers have been able to determine USCRP levels in the adult population that correlate with a state of chronic inflammation linked to atherosclerosis and cardiovascular disease. Levels below 1 mg/L are considered to be at very low risk for cardiovascular disease, and levels greater than 3 mg/L are considered to be at high risk [4].

The purpose of this study was to evaluate the efficacy of USCRP in children with ESRD on PD in comparison to other cardiovascular risk factors and early atherosclerosis markers. Additionally, we wanted to assess these individuals' cardiovascular risk, their relationship to metabolic syndrome, and the severity of their cardiovascular damage [5].

Discussion

Adults and kids with ESRD are more likely to have cardiovascular disease, which can lead to major problems that can lower quality of life and increase morbidity and death. Despite the existence of the traditional cardiovascular risk factors, atherosclerosis progresses more quickly in these individuals, mostly as a result of uremia- and inflammation-related variables. Early cardiovascular damage was found in this group of juvenile patients with ESRD on PD, as shown by Left Ventricular Hypertrophy (measured by LVMI) and elevated CIMT, a marker for atherosclerotic disease. These results agree with earlier studies in the literature [6].

According to studies, CIMT is higher in hypertension patients than in healthy controls

(among both adults and children). Elevated CIMT values are seen in ESRD patients, and CIMT is a reliable indicator of worse cardiac outcomes. Values are greater for PD patients as compared to individuals who have had kidney transplants, indicating that the renal replacement process itself encourages the development of early atherosclerosis. Additionally, it has been noted that following a kidney transplant, CIMT readings tend to return to normal, suggesting that the atherosclerosis-causing effects of renal replacement treatment may be removed and the damage can be reversed [7].

The most frequent cardiac anomaly seen in juvenile patients with ESRD is left ventricular hypertrophy, which is also a standalone risk factor for death in adults (38) and is also the condition. Other contributing variables include CIMT, the resolution of post-transplant left ventricular hypertrophy, and elevated blood pressure [8].

Conclusion

In this group, significant CV damage was seen. It wasn't always possible to detect these CV anomalies with the traditional RF. However, USCRP could be more effective than other RF at spotting damage. Despite a high baseline ferritin and low transferrin saturation levels, there was a statistically significant rise in the average haemoglobin at 90 days of treatment. Patients on chronic hemodialysis who are anaemic may benefit from further haemoglobin increases from intravenous iron supplements [9].

This retrospective analysis found that despite increased baseline ferritin and low levels of normal transferrin saturation, there was a statistically significant rise in average haemoglobin with all the examined intravenous iron products at 90 days of therapy. In this specific patient population, intravenous iron products should be taken into consideration as they may lead to additional increases in haemoglobin in anaemic chronic hemodialysis patients [10].

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