These changes can cause many complications, for example, stimulating 1,25(OH)D receptors, calcium-sensing receptors, and FGF-23 receptors in the hyperparathyroidism, including a reduction of expression of Vitamin D FGFR-1-Klotho complex [4]. On the other hand, other factors can cause on the parathyroid gland and suppresses PTH secretion through the concentration of FGF, which further decreases 1,25(OH)D phosphorus in the urine and increases the reabsorption to the blood, improper nephron function may cause a decrease in the excretion of as a compensation effect for a deficiency of calcium in the body. The can activate the parathyroid gland and enhances the secretion of PTH; absorption of calcium and causes hypocalcemia. However, this situation can contribute to the hypersecretion of PTH. The correlations between these predisposing factors of SHPT are explained.

Conclusion: The study showed that SHPT is common among patients with end-stage renal disease. The most complications of SHPT are mineral and bone metabolism disorders and cardiovascular diseases. SHP may play a causal role in the development of vascular calcifications, ischemic cardiovascular events, and cardiac failure. This research aimed to determine the relationship between renal failure and hyperparathyroidism by measuring the levels of creatinine, phosphorus, calcium, and PTH in patients with kidney failure.

Keywords: Chronic kidney disease, Secondary hyperparathyroidism, Parathyroid hormone, Calcium, Phosphate, Creatinine, Vitamin D.
calcium, and PTH). The mean value of serum creatinine was found to be \(10\pm 3.02 \text{ mg/dL}\), while for phosphate, calcium, and PTH were \((6.03\pm 1.13 \text{ mg/dL})\), \((7.59\pm 0.93 \text{ mg/dL})\), and \((975.1\pm 408 \text{ mg/dL})\), respectively. Twenty-five healthy persons with normal kidney functions were also statistically analyzed as normal control and their data were compared with kidney failure patients. The mean value of calcium was found to be \((8.9\pm 0.5 \text{ mg/dL})\) for the control, while their data for phosphate and PTH were found to be \((4.14\pm 0.61 \text{ mg/dL})\) and \((53.96\pm 12.7 \text{ mg/dL})\), respectively, as shown in Figs. 1 and 2. It was found that 32% (32 patients) had a normal level of serum calcium compared to the normal range which is \((8.5–10.2 \text{ mg/dL})\), while 68% (68 patients) found to have serum calcium less than \(8.5 \text{ mg/dL}\), as shown in Fig. 3. A Scatter plot shows that PTH has a significant negative correlation with calcium \((p<0.05)\) (Fig. 4). Results show that there is a significant negative correlation between phosphate and calcium \((p<0.05)\), as shown in Fig. 5. Data show that 28 patients (28%) had a normal level of serum phosphate, 2% (two patients) had a phosphate <3.5 mg/dL, and most patients (70 patients) had a high level of phosphate compare to the normal range which is \((3.5–5.5 \text{ mg/dL})\). The 70 patients who have a high level of phosphate, their serum phosphate was above \(5.5 \text{ mg/dL}\), as shown in Fig. 6. However, the phosphorus level has a significant positive correlation with creatinine in this study \((p<0.005)\), as indicated in Fig. 7. A Scatter plot also shows that PTH has a significant positive correlation between phosphate and creatinine (Figs. 8, and 9).

**DISCUSSION**

SHPT is one of the most common complications among ESRD patients that should be controlled to prevent other complications such as bone and mineral metabolism disorders and cardiovascular diseases. When the mean value of the control is compared to the mean value of patients, we have noticed that there are a significant difference in all parameters, as the mean values of phosphate and parathyroid were higher for kidney failure patients (Figs. 1 and 2), whereas the mean value of calcium (Fig. 1) was higher for control. These results confirmed that there are relationships between these parameters and SHPT as mentioned previously.

In this research study, it was found that all the patients had very high levels of PTH. The high level might be due to various factors that contributed to the hypersecretion of PTH. The correlation between these predisposing factors of SHPT is explained. Fig. 3 shows that 68% of patients have low levels of calcium, the main reason for this hypocalcemia is due to Vitamin D deficiency. It is worth mentioning that most patients have calcium levels less than the normal levels but close to the normal values; this because PTH compensates for the deficiency. For this reason, a significant negative correlation between PTH and calcium was observed, as shown in Fig. 4. On the other hand, Fig. 5 shows a significant negative correlation between phosphate and calcium, and thus phosphate levels increased. Therefore, calcium binds with phosphate and forms a calcium phosphate complex \((\text{CaHPO}_4)\), this is another cause of hypocalcemia among ESRD patients.
Fig. 6 shows that 70% of patients have an elevation in phosphate levels (hyperphosphatemia), this situation is due to the role of FGF-23, which reduce the expression of type II sodium/phosphate cotransporters (NaPi-2a and NaPi-2c) and allow phosphate excretion [5], but FGFR1-Klotho complex is downregulated in kidneys [6]. However, this contributes to the resistance of the effect of FGF-23 and enhances phosphate retention. There is a significant positive correlation between creatinine and phosphate, as shown in Fig. 7, where Fig. 8 also shows a significant positive correlation between phosphate and PTH. This gives a further significant positive correlation between PTH and creatinine (Fig. 9).

It was found that among ESRD patients, there is low GFR, which leads to an increase in the level of creatinine, and decreased excretion of phosphate, thus, as a result, this causes hyperphosphatemia and hypersecretion of PTH by the mechanism of FGF-23. However, bone and mineral metabolism disorders occur in those patients, as PTH trying to compensate for the decline in calcium by increasing bone resorption. Cardiovascular diseases occur as a result due to the accumulation of calcium phosphate complex, leading to coronary artery calcification. Many other factors cause SHPT, not only kidney failure; these factors are not included in this study.

Fig. 5: Correlation between phosphate and calcium

Fig. 7: Correlation between phosphate and creatinine

Fig. 6: Distribution of phosphate among hemodialysis patients

Fig. 8: Correlation between phosphate and parathyroid hormone

Fig. 9: Correlation between creatinine and parathyroid hormone

Our finding in this study was consistent with the other studies carried out in different countries [4,6]. A study by Arora et al. [6] was found that the mean value of calcium (7.90±1.16 mg/dL) and the mean value of phosphate was (6.44±1.72 mg/dL). Besides, in the same study, it was also found that there is a positive correlation between PTH and creatinine, and a negative correlation between PTH and calcium, which are similar to our results. Moreover, a study by Sliem et al. [4] was determined that the mean value of calcium was (8.2±1.05 mg/dL) and the mean value of phosphate was (5.94±1.7 mg/dL), which are also similar to our finding. Vitamin D was not included in our study because it is not measured regularly for all ESRD patients within the follow-up.
it is only measured to some patients according to the patient situation. Our study has some limitations such as it is only carried out for patient’s reports in 2019 and one hospital.

CONCLUSION

The study showed that SHPT is common among patients with ESRD. The most complications of SHPT are mineral and bone metabolism disorders and cardiovascular diseases. Thus, early detection and treatment of SHPT may control these complications. Further studies with a large sample size and different sites are needed to confirm accurately the different parameter correlations.

ACKNOWLEDGMENT

The authors acknowledge Hebron Governmental Hospital, in Hebron West Bank, Palestine, the Palestinian MOH, and the Palestinian Association for Medical Laboratory Science.

ETHICAL CONSIDERATIONS

The study was approved by the Palestinian MOH. The identities of patients remained unknown and remained confidential and the data only used for research purposes.

CONFLICTS OF INTEREST AND FINANCIAL DISCLOSURE

The authors declare no competing financial interests and no conflicts of interest concerning the authorship and/or publication of this article.

REFERENCES

1. Movahed SM, Mousavi SS, Faramarzi M. Secondary hyperparathyroidism among end-stage renal disease patients in Beharlou hospital, Tehran Province, Iran. J Parathy Dis 2018;6:64-7.
2. Yuen NK, Ananthakrishnan S, Campbell MJ. Hyperparathyroidism of renal disease. Perm J 2016;20:15-127.
3. Slatopolsky E, Brown A, Dusso A. Pathogenesis of secondary hyperparathyroidism. Kidney Int 1999;56:S14-9.
4. Sliem H, Tawfik G, Moustafa F, Zaki H. Relationship of associated secondary hyperparathyroidism to serum fibroblast growth factor-23 in end-stage renal disease: A case-control study. Indian J Endocrinol Metab 2011;15:105-9.
5. Veldurthy V, Wei R, Oz L, Dhawan P, Jeon YH, Christakos S. Vitamin D, calcium homeostasis, and aging. Bone Res 2016;4:16041.
6. Arora K, Goyal G, Soin D, Kumar S, Arora H, Gang C. Correlation of parathyroid hormone levels with mineral status in end-stage renal disease patients. Indian J Endocrinol Metab 2018;22:735-9.