Katalyst Publishing Group

Case Report

Journal of Current Trends in Clinical Case Reports

Hemodialysis Combined with Cinacarbose in the Treatment of a Hemodialysis Patient with Severe Abdominal Pain and Huge Etopic Calcification

Yi Zhao, Ying Huang, Jin-Xuan Su, Yu-Mei Wang*

Department of Nephrology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430022, China

***Corresponding author:** Yu-Mei Wang, MD, Department of Nephrology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430022, China; Tel: 0086-15377581603; Fax: 0086-27-85776343; E-mail: wangyumei75@163.com

Received Date: 23 January, 2021
Accepted Date: 29 January, 2021
Published Date: 09 February, 2021

Citation: Yi Zhao, Ying Huang, Jin-Xuan Su, Yu-Mei Wang (2021) Hemodialysis Combined with Cinacarbose in the Treatment of a Hemodialysis Patient with Severe Abdominal Pain and Huge Etopic Calcification. J Cur Tre Clin Case Rep 2(1): 105

Abstract

Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD) could lead to blood vessel and soft tissue calcification. Vascular calcification attracts more attention from researchers than soft tissue calcification, for vascular calcification is an independent risk factor which significantly increases the incidence of cardiovascular events and mortality. The studies of soft tissue calcification are mostly clinical case reports. For the lack of large-scale clinical studies of soft tissue calcification, there is no final conclusion on its treatment. This paper reported a case of maintained hemodialysis patients with severe abdominal pain and huge calcification lesion in the soft tissue of the right hip joint. This patient experienced recurrent severe abdominal pain and severe right hip pain during hemodialysis treatment. With intensified hemodialysis and low-dose of cinacarbose treatment, the abdominal pain was completely relieved, the calcified lesion was reduced from 16 cm to 6.9 cm, and the joint motion was largely improved.

Keywords: Chronic Kidney Disease-Mineral and Bone Disorder, Hemodialysis, Vascular Calcification, Soft Tissue Calcification

Case presentation

A 40-year-old female patient, with 49kg in weight and 162 cm in height, was admitted on May 8, 2018 for the chief complaint of "8 years of hemodialysis, 3 years of recurrent abdominal pain, and 2 years of swelling and pain of the right hip joint". The patient was received hemodialysis 8 years ago because of uremia stage of chronic renal failure. After one year hemodialysis, she received allogeneic kidney transplantation. However, acute rejection occurred three days after kidney transplantation. The transplanted kidney was removed one week later, and she received hemodialysis treatment again. The dialysis frequency was twice a week and 4 hours each time. 5 years ago, she received thyroidectomy for thyroma, with normal thyroid function after thyroidectomy. 3 years ago, the patient suffered severe persistent tormina-like abdominal pain during hemodialysis in the lower abdomen, without hypotension. The pain could be relieved by intramuscular injection of anisodamine and tramadol. Since then, the abdominal pain repeated frequently about once a week, which seriously hindered the adequacy of hemodialysis. 2 years ago, the patient found the right hip joint mass, which was gradually enlarged, accompanied with severe pain and limited activity. She needed to take talenin and tramadol sustained-release tablets every day to relief pain. In addition, patients had long-term hyperphosphatemia and secondary hyperparathyroidism, and had been treated with oral calcium acetate 2 tablets, three times a day, and intermittent supplement of active vitamin D. In January 2018, the patient visited the orthopedic department because of mass pain which seriously affected her life, and underwent abdominal aorta CTA + right hip plain scan + three-dimensional examination. The report showed that: 1. multiple calcified plaques were found in abdominal aorta, iliac artery, celiac artery, superior mesenteric artery and renal artery, and the local lumen was slightly narrowed; 2. massive calcification was found in the inner side of right hip joint, with the maximum cross-sectional area of 15.1× 9.5cm. However, orthopedic surgeons suggested conservative treatment, for the consideration of the mass size and the complexity of surrounding structures. Then she went to our department for further treatment. Physical examination after admission: blood pressure 152/84 mmHg, heart rate 110 beats/min. She presented chronic kidney disease and anemia face. The cardiopulmonary examination was normal. The abdomen was soft, without tenderness and rebound pain. Tremor was palpated at arteriovenous fistula in the left forearm. A huge swelling mass was inspected at right hip joint, with tenderness and obvious activity limitation. There was no edema in both lower limbs.

Diagnosis and treatment processes

After admission, the relevant examination was completed. The right hip joint plain scan and 3 dimensional scan showed a mass calcification on the inner side of the right hip joint, with a maximum cross-sectional area of about 16× 10cm. The muscle groups of the right thigh were slightly swollen, and osteoporosis was found in the bones of the right hip. There was no obvious dislocation of the joint. Radiologist considered the hip mass as

ectopic calcification due to uremia. Clinical diagnosis: chronic kidney diseases, uremia, right hip joint giant ectopic soft tissue calcification, multiple artery calcification, renal anemia, renal osteopathy, renal hypertension, and hyperuricemia. She was given adequate hemodialysis 3 times a week, 4 hours each time, hemodiafiltration once a week, hemodialysis + perfusion once a week, oral administration of cinacacet 25 mg/d, lanthanum carbonate 500 mg, 3 times a day, and beraprost sodium 40 μg/d, subcutaneous injection of recombinant human erythropoietin-β (Rockman 5000 IU) 2 ~ 3 times a week, blood pressure was controlled around 130/80mmHg. After 3 weeks of treatment, the right hip pain symptoms of the patients were significantly relieved, analgesics were stopped, and the frequency of abdominal pain was also reduced, about 1 time a month. Two months later, she could not tolerate the gastrointestinal side effects of lanthanum carbonate, she stopped taking lanthanum carbonate by himself and took 2 tablets of calcium acetate, 3 times a day. The reexamination found that the blood calcium and phosphorus were significantly increased, calcium acetate was stopped and sviram was given, 2.4g, three times a day, and maintained until now. In recent three months, abdominal pain and the right hip joint pain were basically relieved, the mass was significantly reduced, and the daily activities were almost not limited. In April 2019, the patient received re-examination (biochemical indexes before and after treatment were shown in Fig.1). CTA of whole aorta + plain scan + three-dimensional of right hip joint showed: 1. Calcified plaques were found scattered in aorta, iliac artery, celiac trunk and kidney; 2. the wall of superior and inferior mesenteric artery was smooth and the lumen was unobstructed; 3. soft tissue mass with size of 6.9× 5.2cm was scattered in the inner edge of right gluteus maximus muscle, with patchy and spotty calcification inside the mass (Fig.2 presented CT scan of calcification lesion before and after treatment).

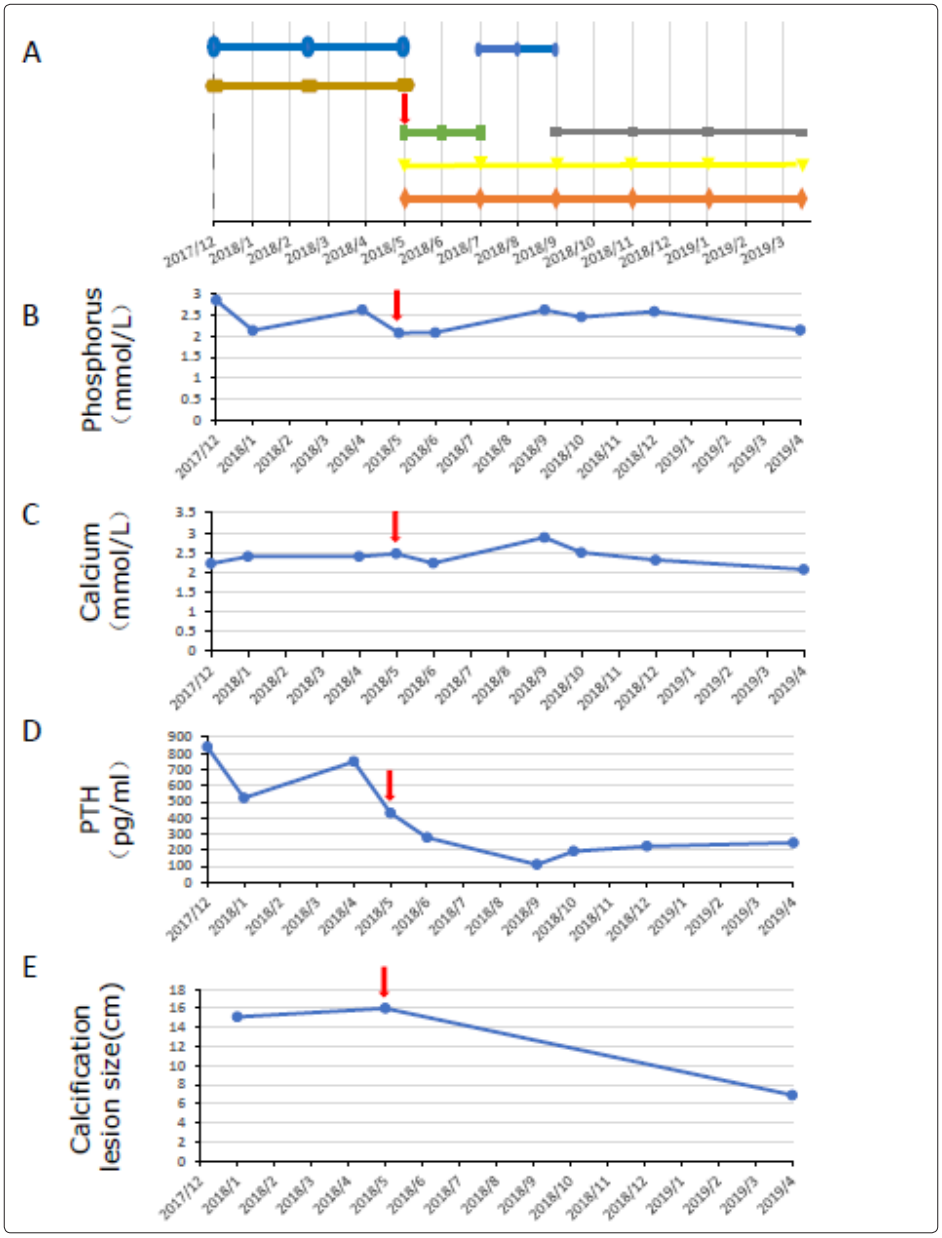

Figure 1: Changes of serum phosphorus, serumcalcium, parathyroid hormone levels, and the size of ectopic calcification at different treatment time points. **A**. Therapeutic method. Blue line represents oral calcium acetate, brown line represents oral active vitamin D, green line represents oral lanthanum carbonate, yellow line represents oral cinacacet, grey line represents oral sviram. Orange line represents intensive hemodialysis. **B**. Changes of levels of serum phosphorus, **C**. Changes of levels of serum calcium, **D**. Changes of levels of serum parathyroid hormone, E. Size of calcification lesion. Red arrow represents initiation of intensive treatment.

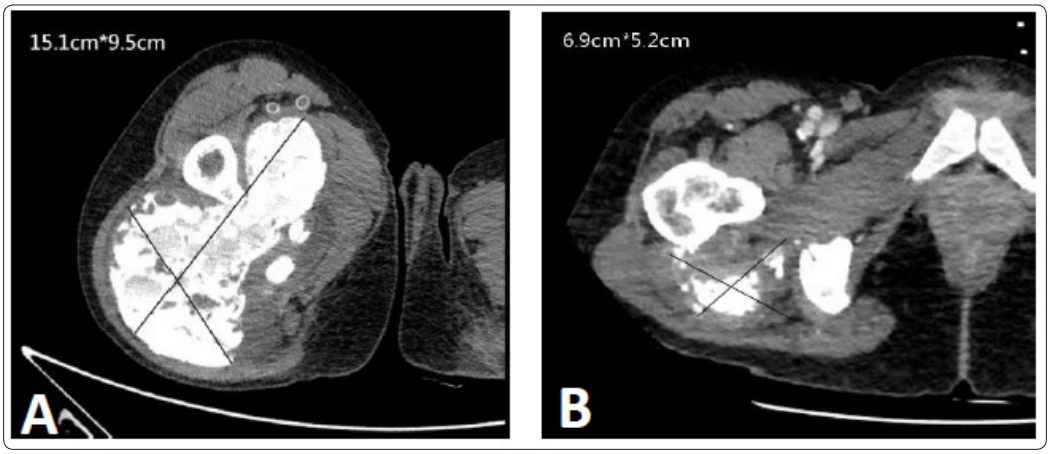

Figure 2: CT scan of calcification lesion before and after treatment. **A**. Before intense hemodialysis treatment. **B**. After intense hemodialysis treatment.

Discussion

CKD-MBD is one of the most important complications of CKD, especially in patients with end-stage renal disease (ESRD). CKD-MBD is a clinical syndrome including series of laboratory abnormalities, bone lesions, vascular and soft tissue calcification, which significantly increases the risk of cardiovascular events and mortality [1, 2]. The incidence of vascular calcification in dialysis patients is very high, and hemodialysis patients are much more likely to develop vascular calcification than peritoneal dialysis patients [3]. This is also an important reason for the high morbidity and mortality rate of cardiovascular diseases in dialysis patients [4]. Soft tissue calcification was mainly found in the shoulder, hip, elbow, wrist, phalangeal, and digital joints which leads joint swelling, pain, and movement limitation [5-8]. There are many reasons for ectopic calcification, including hyperphosphatemia, increased calcium phosphorus product, secondary hyperparathyroidism, abnormal function of fibroblast growth factor 23, improper use of active vitamin D, use of calcium phosphorus binding agent, and increased calcium load caused by dialysate [9-13]. It is reported that low phosphorus diet, calcium free phosphate binder, active vitamin D, and calcium like agent have certain therapeutic effect on vascular calcification [14]. However, the treatments of soft tissue calcification are rarely reported, except for a few clinical cases, which are mainly about surgical resection of mass, parathyroidectomy, increasing dialysis efficiency and dialysis time, low calcium dialysate, high-dose of cinacarbose treatment, kidney transplantation, and et al [15-18].

In this case, the patient had long-term hyperphosphatemia, hyperparathyroidism, increased calcium phosphorus product, long-term use of calcium containing phosphate binder and active vitamin D, and inadequate hemodialysis (4 hours a time, twice a week). The multiple artery calcified plaques and huge ectopic calcification of the soft tissue around the right hip joint may be related to the above mentioned reasons. The initial symptom was severe abdominal pain, and hemodialysis seemed to be its only trigger factor. The pain usually started within 30 minutes after the initiation of hemodialysis, without decrease of blood pressure drop or blood sugar. It could be relieved by antispasmodics and analgesics within 30 minutes. There is no such report in Chinese journal or in international journal, except for a few similar cases. One case was reported by Rossi et al., which presented a diabetic hemodialysis patient who had severe abdominal pain caused by non-occlusive mesenteric ischemic intestinal necrosis. The patient received intestinal resection of the necrotic segment. During the operation, extensive calcification of superior mesenteric artery was found. The possible reasons for the intestinal necrosis were extensive superior mesenteric artery calcification and reduction of blood flow caused by hypotension during dialysis [19]. Another case was reported by Ying et al. The patient also suffered abdominal pain during hemodialysis, and was considered as non-occlusive mesenteric ischemia. The etiology was also extensive abdominal artery calcification accompanied with dialysis hypotension. After reducing ultrafiltration volume, increasing the dosage of low molecular weight heparin, and prostaglandin, the pain was basically relieved [20]. In the present case, after exclusion dialysis hypotension, hypoglycemia, low temperature of dialysis fluid, allergic reaction to dialysis pipeline, imbalance reaction, psychological factors, etc., combined with CTA results, the main reason of abdominal pain was considered as extensive calcification of abdominal artery. With multiple calcified plaques of abdominal aorta, celiac trunk, mesenteric artery and its branches, reduced blood flow during hemodialysis caused ischemia of mesenteric arties and induced abdominal pain. It is also proved by the alleviation of abdominal pain with antispasmodics. Therefore, the treatments were mainly focused on ectopic calcification. We increased the time of hemodialysis, combined with a variety of hemodialysis modes (high flux hemodialysis, hemodiafiltration, and hemoperfusion) to enhance the clearance of uremic toxins (phosphorus, PTH, etc.). Also we take other therapeutic alterations, such as we changed the calcium phosphorus binding agent into non calcium phosphorus binding agent lanthanum carbonate, stopped active vitamin D, gave small doses of cinacacet and benazepril prostaglandin sodium (because of economic reasons, patients could not afford a larger dose of treatment), kept warm of abdomen, and gave other symptomatic treatment. 20 days later, joint swelling and pain, and the frequency of abdominal pain were significantly reduced. One month after treatment, the levels of serum calcium, calcium phosphorus product, and parathyroid hormone levels were significantly decreased, while there were no significant changes in serum phosphorus. The reason of hyperphosphatemia may due to the inadequate dosage of lanthanum carbonate (500 mg/kg, twice to three times a day, for the consideration of side effect of digestive system and economic reasons). The patient stopped taking lanthanum carbonate and changed to calcium acetate in the middle of the treatment, and the rest of the treatment remained unchanged. After the alteration of treatment, the serum calcium phosphorus and calcium phosphorus product increased significantly, while parathyroid hormone level still maintained a downward trend, and the patient got further

improvement in the symptom. After sviram dephosphorization treatment, serum calcium, serum phosphorus, calcium phosphorus product, and parathyroid hormone levels increased significantly. After 11 months of treatment, the abdominal pain was completely relieved, and the heterotopic calcification mass around the right hip joint was significantly reduced. With the pain remission, she got back her daily activity ability (shown in Fig.1).

Interestingly, we found that calcium acetate and vitamin D could maintain serum calcium, serum phosphorus, parathyroid hormone at acceptable levels before the intensified dialysis treatment, while the calcification of blood vessel and soft tissue were still in progress, which is directly related to the modality and mortality of cardiovascular diseases. In many countries, calcium phosphate binding agent and active vitamin D are the most classic and economic treatment, and are widely applied worldwide. This also reminds us that medical workers need to raise awareness, not only pay attention to blood phosphorus, blood calcium, and parathyroid hormone levels, but also pay more attention to the calcification lesions of the cardiovascular system.

Conclusion

In this case, we describe a maintenance hemodialysis patient with severe abdominal pain caused by extensive arterial calcification and accompanied by huge calcified nodules in the right hip joint. By increasing the efficiency and duration of hemodialysis, stopping active vitamin D, using non-calcium phosphate binder to reduce calcium phosphate load, and small dose of cinacarbose to maintain calcium phosphate balance and parathyroid hormone level, vascular and soft tissue calcification caused by CKD-MBD could be significantly improved.

Acknowledgements

This work was supported by grants from the National Natural Science Foundation of China (No. 81570657).

Conflict of interest

The authors have no conflict of financial interest.

References

1. Fujii H (2019) [CKD-MBD and cardiovascular disease.]. Clin Calcium 29: 179-184.

2. Reiss AB, Nobuyuki Miyawaki, Jane Moon, Lora J.Kasselman, Iryna Voloshyna, et al. (2018) CKD, arterial calcification, atherosclerosis and bone health: Inter-relationships and controversies. Atherosclerosis 278: 49-59.

3. Liu ZH, Xue-Qing Yu, Jun-Wei Yang, Ai-Li Jiang, Bi-Cheng Liu, et al. (2018) Prevalence and risk factors for vascular calcification in Chinese patients receiving dialysis: baseline results from a prospective cohort study. Curr Med Res Opin 34: 1491-1500.

4. Cozzolino M, Paola Ciceri, Andrea Galassi, Michela Mangano, Stefano Carugo, et al. (2019) The Key Role of Phosphate on Vascular Calcification. Toxins (Basel) 11: 213.

5. Kim SJ, Marc Goldstein, Tamas Szabo, Andreas Pierratos, et al. (2003) Resolution of massive uremic tumoral calcinosis with daily nocturnal home hemodialysis. Am J Kidney Dis 41: E12.

6. Hung TH, Jui-Kuo Hung, Ing-Lin Chang, Chen tung Yu, Shih-Jen Chen (2007) Tumoral calcinosis of the shoulder. Int Surg 92: 300-303.

7. Razaq S, F Nigar and S Naqvi (2018) Ultrasonographic Diagnosis of Tumoral Calcinosis in End-Stage Renal Disease. J Coll Physicians Surg Pak 28: 640-642.

8. Lindhard K, Bo Broberg, Henrik Groenberg, Henrik Post Hansen (2017) Treatment of a soft tissue calcification in a patient receiving peritoneal dialysis. Hemodial Int 21: E34-E39.

9. Ohnishi M, Teruyo Nakatani, Beate Lanske, M Shawkat Razzaque (2009) In vivo genetic evidence for suppressing vascular and soft-tissue calcification through the reduction of serum phosphate levels, even in the presence of high serum calcium and 1,25-dihydroxyvitamin d levels. Circ Cardiovasc Genet 2: 583-590.

10. Maruyama N, Terumi Higuchi, Michiko Ono, Hidetaka Oguma, Yoshihiro Nakamura, et al. (2019) Correlation between Aortic Calcification Score and Biochemical Parameters in Hemodialysis Patients. Contrib Nephrol 198: 40-51.

11. Chou FF, Jin-Bor Chen, Shun-Cheng Huang, Yi-Chai Chan, Shun-Yu Chi, et al. (2018) Changes in serum FGF23 and Klotho levels and calcification scores of the abdominal aorta after parathyroidectomy for secondary hyperparathyroidism. Am J Surg 218: 609-612.

12. Rodriguez M, et al. (2011) Vitamin D and vascular calcification in chronic kidney disease. Kidney Blood Press Res 34: 261-268.

13. Kobayashi S (2017) Cardiovascular Events in Hemodialysis Patients: Challenging against Vascular Calcification. Ann Vasc Dis 10: 1-7.

14. Nitta K, Tetsuya Ogawa, Norio Hanafusa, Ken Tsuchiya (2019) Recent Advances in the Management of Vascular Calcification in Patients with End-Stage Renal Disease. Contrib Nephrol 198: 62-72.

15. Lindhard K, Bo Broberg, Henrik Groenberg, Henrik Post Hansen (2017) Treatment of a soft tissue calcification in a patient receiving peritoneal dialysis. Hemodial Int 21: E34-E39.

16. Cofan F, S García, A Combalia, JM Campistol, F Oppenheimer, et al. (1999) Uremic tumoral calcinosis in patients receiving longterm hemodialysis therapy. J Rheumatol 26: 379-385.

17. Xie T, Yong Zhang, Xiangling Tang, Mengying Chen, Weiru Zhang (2014) Amelioration of uremic tumoral calcinosis with high-flux hemodialysis treatment. Int Urol Nephrol 46: 1467-1468.

18. Chu HY, Pauling Chu, Yuh-Feng Lin, Heng-Kuang Chou, Shih-Hua Lin (2011) Uremic tumoral calcinosis in patients on peritoneal dialysis: clinical, radiologic, and laboratory features. Perit Dial Int 31: 430-439.

19. Rossi UG, Francesco Petrocelli, Sara Seitun, Carlo Ferro

(2012) Nonocclusive mesenteric ischemia in a dialysis patient with extensive vascular calcification. Am J Kidney Dis 60: 843-846.

20. Qian Y, Xiao-Nong Chen, Hao Shi, Jingyuan Xie, Nan Chen (2015) Refractory Abdominal Pain in a Hemodialysis Patient. Case Rep Nephrol Dial 5: 145-151.