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Review article

Food strategies of renal atrophy based on Avicenna and conventional medicine

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ABSTRACT

Kidneys have an important role in the body. Any damage to kidney role can damage many organs of the body. Traditional Persian Medicine (TPM) or Iranian traditional medicine (ITM) is an ancient temperamental medicine with many literatures about kidney diseases and Avicenna (980–1025 AD) describes kidney diseases in details. This is a review study by searching of the most important clinical and pharmaceutical TPM textbooks such as The Canon of Medicine by Avicenna and scientific data banks using keywords such as "Hozal-e-Kolye", renal atrophy, tubular atrophy, kidney, chronic kidney disease, and end stage renal disease. This paper found that "Hozal-e-Kolye" in TPM texts is the same tubular atrophy in conventional medicine due to some similar symptoms between them. Lifestyle modification and use of proposed foodstuffs can be considered as a complementary medicine in addition to conventional treatments to manage these patients. TPM scholars prescribed some foodstuffs such as camel milk, sheep's milk and *Ficus carica* for this disease as a complementary management. This study aimed to explain HK (the same tubular atrophy considering their similar symptoms) and introduce some foodstuffs. It seems using of foodstuffs affecting tubular atrophy based on TPM literatures can has a role as a supplemental method in company with conventional medicine management.

1. Introduction

Chronic kidney disease (CKD) can lead to end stage renal disease (ESRD).[1] Renal atrophy is one of the kidney diseases occurring due to shrinkage of the kidney in which the nephrons are lost and tubular atrophy is the loss of parenchymal renal cells characterizing in CKD.[2] Tubular atrophy is a hallmark of chronic kidney disease.[3] The prevalence of CKD is increasing and estimated to be 8–16% worldwide.[4,5] Treatment of CKD considering the stages of the disease is various and the relationship between nutrition and kidney disease has a main effect on outcomes.[6] Using complementary medicine (CAM) in treatment of chronic disease is growing in the past 10 years in the U.S.[7] The management of CKD based on herbal traditional medicine is recommended as a preventive and therapeutic strategy[8] and medicinal plants with kidney protective activities is prescribed.[9]

Traditional Persian Medicine (TPM) is an ancient temperamental medicine with a history of over one thousand years. Temperament is made of a normal interaction between four basic elements, named hot, cold, wet, and dry elements. And dystemperament occurs when the whole body or an organ's temperament changes.[10] The kidney dystemperament occurs when the kidney temperament is changed and its function is disturbed. These conditions can lead to "Hozal-e-Kolye" (HK). HK in TPM occurs when the kidney becomes thin means its fat gets low or eliminates.[11–14] This study aimed to explain HK (the same tubular atrophy

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considering their similar symptoms) and introduce some foodstuffs as a complementary food management based on TPM.

2. Materials & methods

This is a review study by searching of the most important clinical and pharmaceutical TPM textbooks (That is not derived from the other books) such as *The Canon of Medicine* by *"Avicenna"* (10th and 11th centuries), *Exir-e-Azam* by *Chishti* (19th century), *Tuhfat Al-momenin* by *Tonkaboni* (17th century) and *Makhzan-Al-advie* by *Aqili* (18th century), with the keyword of "Hozal-e-Koleye" (the same tubular atrophy). Then, all of the foodstuffs extracted from this study were searched to find the related activity concerning the kidney function improvement by searching in scientific data banks such as Medline with these keywords: renal atrophy, tubular atrophy, kidney, chronic kidney disease, end stage renal disease. Finally, the results were inserted into a table.

3. Results

3.1. CKD in conventional medicine

CKD occurs when the impaired kidney function persists for three months or more. In this disorder, there is a decrease kidney function based on the presence of kidney damage. Glomerular filtration rate (GFR) has a central role in the pathophysiology of CKD complications. There are five stages in CKD classification on the basis of GFR: stage1 (more than 90 ml/min/1.73 m^2), stage2 (60–89 ml/min/1.73 m^2), stage3 (30–59 ml/min/1.73 m^2), stage4 (15–29 ml/min/1.73 m^2), stage5 (less than 15 ml/min/1.73 m^2). Proteinuria has an important role in the pathogenesis CKD progression. When GFR is less than 15 ml/min/1.73 m^2, kidney failure occurs and it needs dialysis or transplantation for treatment.[15]

Manifestations of CKD include: fluid and electrolyte imbalance (impaired ability to excrete leading to sensitive hypertension and edema due to reduce GFR), acid base abnormalities, carbohydrate intolerance, calcium and phosphate abnormalities and metabolic bone disease, hematologic abnormalities, gastrointestinal abnormalities, dermatological abnormalities, neuromuscular abnormalities. These patients are at risk of cardiovascular diseases.[16] Also, there is a nocturnal polyuria in renal insufficiency that is a precocious symptom in CKD.[17]

3.2. Phosphorus and tubular defect

The main homeostasis of phosphorus occurs in the kidney and the small intestine. A large part of phosphate from eating is excreted in the urine. There is a hypophosphatemia in the renal tubular defect.[18] 80% of the phosphorus reabsorption occur in the proximal tubule of the kidney, but in a tubular defect such as tubular atrophy this process doesn't occur so in this condition there is a hypophosphatemia and phosphaturia.[19] The urine color is white because of phosphaturia.[20]

3.3. Renal atrophy in conventional medicine

3.3.1. Renal atrophy

Renal atrophy caused by many diseases such as acute or chronic pyelonephritis and obstruction of the urinary tract, the systemic atherosclerosis, metabolic syndrome, sickle cell disease,[21] atherosclerotic renal artery stenosis,[22] after hereditary renal cell carcinoma surgery,[23] xanthogranulomatous pyelonephritis (Cortical renal atrophy),[24] posttraumatic (injury).[25] Etiology of unilateral renal atrophy includes hydronephrosis, tumor, tuberculosis, Calculouse, chronic pyelonephritis,[26,27] congenital hypoplastic kidney, renal infarction, radiation, renal artery stenosis,[26] partial nephrectomy.[23] The classic signs of renal atrophy in modern medicine include high blood pressure, low calcium, acidosis, anorexia, malnutrition (serious deficiency minerals and vitamins),[28] elevations in the serum creatinine concentration.[29] Acute kidney injury (AKI) can lead to renal atrophy by incomplete tubular repair, tubulointerstitial inflammation, and interstitial fibrosis.[30] Insufficient blood flow of kidney, can result in the renal atrophy too.[31]

3.3.2. Renal tubular disorder

Renal tubules are very important in the body homeostasis. The proximal tubules play a main role in the transport of phosphate, glucose, amino acid, bicarbonate and sodium. The dysfunctions of these tubules are primary or secondary. When these tubules are injured, some disorders occur, such as hypophosphatemia, and aminoaciduria. Common symptoms of most renal tubulopathies include polyuria and also growth failure and resistant rickets (in children). If tubular dysfunction occurs, phosphaturia will be present.[32] Phosphaturia cause white urine.[20] (Table 1).

3.3.3. Etiologies of tubular atrophy

After allografts, persistent glomerulonephritis and proteinuria, antiglomerular basement membrane disease, medullary cystic kidney disease type I (a mutation in the mucin 1 gene), chronic tubulointerstitial diseases, allergic interstitial nephritis, granulomatous interstitial nephritis, vesicoureteral reflux and reflux nephropathy, lithium salts, the calcineurin inhibitor (CNI) immunosuppressive agent's cyclosporine and tacrolimus, prolonged and severe hypokalemic nephropathy, diphtheria toxin.[33]

3.4. HK in Traditional Persian Medicine

TPM scholars believed that any organ of the body has a typical temperament. In an ideal healthy state, the individual function is very good and dystemperament occurs when the whole body or an organ's temperament changes. In other words, it grows up in the imbalance of the quality or quantity of humors including phlegm, bile, blood and black bile.[13] The kidney dystemperament occurs when the kidney temperament is changed and its function is disturbed. This kidney dystemperament can lead to "Hozal-e-Kolye" (HK). HK in TPM occurs when the kidney becomes thin (its fat gets low or eliminates), hot or cold.[13,14] In high kidney temperature, the kidney fat is lost because of the warmness and in low kidney temperature, the kidney equalizer is disturbed. TPM scholars believed that HK occurred by several reasons such as dystemperament of the kidney, evacuation (in TPM, evacuation means excretion of many fluids from the body, such as excessive hemorrhage, diarrhea, severe vomiting, expelling excess semen and excess usage of purgative or diuretic drugs resulting severe dehydration). Symptoms of HK include white urine, polyuria, weight loss, permanent low back pain and low libido.[11–14] (Table 1). Some

Table 1
Comparing symptoms of HK and tubular atrophy.

Symptoms of tubular disorders in conventional medicine	Ref	Symptoms of HK in TPM	Ref
Polyuria	17	Polyuria	11–14
White urine (because of phosphaturia)	20	White urine	
Growth failure	32	Weight loss	
Resistant rickets	32	Permanent low Back pain	
Hypophosphatemia in the laboratory test	18	Low libido	
		There was not laboratory test at Avicenna's time	

TPM scholars believed that eye weakness and headache are caused due to the kidney fat loss in HK process. It was written in the other book that one of the symptoms of HK is mild pain in the back of head.[34,35]

3.5. Food strategies in CKD in conventional medicine

Nutrition has a main role in the treatment of CKD. Nowadays in America, diet and lifestyle behaviors are important. There are food strategies recommending in CKD such as:

- Intake of protein and energy (Considering the risk of malnutrition in CKD) is recommended. However, protein-restricted diets are used to decrease uremic symptoms.[36]
- A calorie of at least 30–35 kcal/kg is recommended.
- Adequate Intake of vitamin supplements (vitamin C, Thiamin, Riboflavin, Niacin, Folate, Pyridoxine, Cobalamin, Biotin, and Pantothenic acid) is recommended.
- Use of vitamin D to prevent bone loss is needed.[36]
- Low phosphorus intake (ideally 700 mg/day) especially in ESRD is needed. However, adequate protein intake must be maintained.[37]
- Low fat diets including nonhydrogenated and unsaturated fats are recommended to prevent and treat the progression of cardio vascular disease (CVD) in these patients. Intake of whole grains, fruits, vegetables, and omega-3 fatty acids to correct dyslipidemias.[36]
- Control of hypertension with DASH plans (Dietary Approaches to Stop Hypertension) including lower in fat and sodium and high in potassium, magnesium, calcium, fiber and antioxidant is necessary.[36]
- Considering the risk of diabetes, diabetic regimen is needed.[36]
- Allergen foods such as gluten, nuts, dairy foods, citrus foods such as oranges and grapefruit, cantaloupe, honeydew, berries, chocolate, shellfish, eggs, and sulfites should be restricted.[36]

3.6. Food strategies in HK in Traditional Persian Medicine

The principle of treatment of any disease is the elimination of its main causes and modifying the lifestyle.[13] The management of HK is based on the etiologies too. Avicenna and the other TPM scholars prescribed fattening foods for patients with HK (Table 2):

3.6.1. Herbal-derived compounds

Herbal drugs mentioned in TPM literature include *Ficus carica*, *Dava-Al-Taranjabin* (milk + Manna of Alhagi), safflower, raisin, banana, chickpea, kidney bean, rice flour, flixweed, sesame oil, coconut and almond.[11,12,14]

3.6.2. Animal-derived compounds

Animal-derived compounds prescribed in TPM to fatten the thin kidney include poultry meat, lamb meat, camel milk, sheep's milk and the other food materials like honey, egg.[11,12,14]

3.7. Foodstuffs forbidden in CKD in conventional medicine

Eating of some foodstuffs are forbidden in CKD such as: Alfalfa, Aloe, Aristolochic acid, *Artemisia absinthium* (wormwood plant), Autumn crocus, Bayberry, Blue cohosh, Broom, Buckthorn, Capsicum, Cascara, Chaparral, and Chuifongtuokuwan (Black Pearl), Coltsfoot, Comfrey, Dandelion, Ephedra (Ma Huang), Ginger, Gingko, Ginseng, Horse chestnut, Horsetail, Licorice, Lobelia, Mandrake, Mate, Nettle, Noni juice, Panax, Pennyroyal, Periwinkle, Pokeroot, Rhubarb, Sassafras, Senna, St. John's wort, Tung shueh, Vandeliacordifolia, Vervain, Yohimbe.[52]

4. Discussion

According to this study, HK is the same tubular atrophy in the early stages of CKD. There is oliguria in CKD; however, there is polyuria in the early stage of CKD.[16,17] Tubular atrophy is a hallmark of CKD[3] and polyuria is one of its symptoms,[32] so HK is the same tubular atrophy at the beginning of CKD.

The management of tubular atrophy is based on the causes in both CM and TPM. In both of these medicines, the diet is important.[53] In CM, restriction of protein in the diet is discussed. Some researchers believed that restriction in the intake of protein and energy is not necessary to prevent of the risk of malnutrition.[54] In a clinical trial, restricted protein diet was more effective.[55] Some scientists believed that restriction of protein is needed to decrease of uremic symptoms.[56] Although some scientists have proven that a

Table 2
Fattening foods and herbal or animal-derived compounds in tubular atrophy based on TPM resources.

	Common name	Local name[11,13,14]	Scientific name[38]	Family[38]	Fattening cause in conventional medicine	Reference
Herbal-derived compounds	Safflower	Ghortom	*Carthamustinctorius*	Asteraceae	The source of fat	39
	Fig	Tin	*Ficus carica*	Moraceae	Excellent source of mineral, vitamin & carbohydrates	40
	Raisin	Zabib	*Vitis vinifera*	Vitaceae	A rich source of carbohydrate	41
	Banana	Mauz	*Musa* spp	Musaceae	High carbohydrate & energy	42
	Manna of Alhagi with milk	Taranjabin	–	–	High calorie[a]	43
	Chickpea	Hemmas	*Cicer arietinum*	Fabaceae	A good source of carbohydrate & protein	44
	Kidney bean	Loubia	*Phaseolus vulgaris*	Fabaceae	High carbohydrate & protein	45
	Rice flour	Ard-e-brenj	*Oryza sativa*	Poaceae	High carbohydrate	45
	Flixweed	Bazr al Katan	*Descurainia sophia*	Brassicaceae	Stomach strengthening, appetizer	46
	Sesame oil	Dohn al Samsem	*Sesamumin dicum*	Pedaliaceae	High fat	45
	Coconut	Nargil	*Cocos nucifera*	Arecaceae	High calorie	47
	Almond	Louz	*Prunus dulcis* L	Rosaceae	High calorie	48
Animal-derived compounds	Camel milk	Laban al Baghar	–	–	Rich in protein	49
	Sheep's milk	Laban al Ghanam	–	–	High protein	50
	Poultry meat	Lahm al Bot	–	–	High protein	45
	Lamb meat	Lahm al Hamalan	Bos Taurus – *Ovis aries*		High protein	45
	Honey	Asal	Honey		The carbohydrate-rich meal	51
	Egg	Bayz	*Gallus domesticus*	–	High quality protein, vitamin B, minerals	49

[a] Dava al taranjabin is a compound of manna of alhagi (high calorie) with milk (high protein).

restricted protein diet supplemented with keto analogues (a diet of essential amino acids such as phenylalanine and Valine) can delay the progression of CKD without malnutrition.[57] In TPM, correction of the kidney dystemperament by some foodstuffs prescribed in addition to fattening foodstuffs, including some nuts and seeds, meats, and natural drugs is done.[13] In CM some chemical drugs prescribe instead of that, in addition to recovering of the complications and comorbidities. Nutrition therapy in CM is recommended for prevention of malnutrition and decreasing the progression of CKD. In CM, some foodstuffs should be avoided as mentioned in the results. Albeit neither of them is mentioned in TPM as a prescribed food strategy.[36]

In CM, low phosphorus intake is recommended. Of course phosphorus is not typically restricted until hyperphosphatemia is present.[58]

Low sodium intake to control of hypertension and low phosphorus intake for prevention of progressive CKD are recommended in CM.[36] At TPM scientist's time, there were not laboratory tests to control of these cases. Then, they detected diseases by clinical symptoms and observing touching the skin surface of involved organ to find organ's temperament.

It needs to be cared about using of egg yolk and almond. They are not forbidden in tubular atrophy due to low phosphorus and high protein (an adequate phosphorus-to-protein ratio: 24.7 in egg yolk and 22.3 in almond).[37] But increasing in their usage can cause a problem. Also using of some allergen foods as mentioned in this paper should be cared and it needs to be sure that the consumer has not allergy to these foodstuffs.

5. Conclusion

It seems using of foodstuffs affecting tubular atrophy based on TPM literatures can have a role as a supplemental method in company with CM management. However, more studies about these foods and their effects on these cases are needed.

Conflict of interest

The authors declare that there are no conflicts of interest.

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References

1. Hanif M, Javed H, Jallani U, Ranjha NM. Prevalence of end stage renal disease in diabetic obese and hypertensive patients and cardiovascular risk in dialysis patients. *Pak J Pharm Res*. 2016;2:42–48.
2. Moreno JA, Ramos AM, Ortiz A. 22 apoptosis in the kidney. *Apoptosis Physiol Pathol*. 2011:240.
3. Schelling JR. Tubular atrophy in the pathogenesis of chronic kidney disease progression. *Pediatr Nephrol*. 2016;31:693–706.
4. McCarthy JT. A practical approach to the management of patients with chronic renal failure. In: *Mayo Clinic Proceedings*. 1999:269–273.
5. Jha V, Garcia-Garcia G, Iseki K, et al. Chronic kidney disease: global dimension and perspectives. *Lancet*. 2013;382:260–272.
6. Choi P, Stevenson J. Nutrition and kidney disease. In: Harber M, ed. *Practical Nephrology*. London: Springer London; 2014:621–628.
7. Clarke TC, Black LI, Stussman BJ, Barnes PM, Nahin RL. Trends in the use of complementary health approaches among adults: United States, 2002–2012. *Natl Health Stat Rep*. 2015:1.
8. Wijesinghe W, Pilapitiya S, Hettiarchchi P, Wijerathne B, Siribaddana S. Regulation of herbal medicine use based on speculation? A case from Sri Lanka. *J Tradit Complement Med*. 2017;7(2):269–271. http://dx.doi.org/10.1016/j.jtcme.2016.06.009.
9. Tsai W-H, Yang C-C, Li P-C, Chen W-C, Chien C-T. Therapeutic potential of traditional chinese medicine on inflammatory diseases. *J Tradit Complement Med*. 2013;3:142.
10. Feyzabadi Z, Jafari F, Feizabadi PS, Ashayeri H, Esfahani MM, Aval SB. Insomnia in Iranian traditional medicine. *Iran Red Crescent Med J*. 2014;16:e15981.
11. Aghili Alavi Khorasani MH. *Makhzan-al-Adwiah*. Tehran: Sabz Arang and Tehran University of Medical Sciences; 2011.
12. Hakim Mommen M. *Tuhfat-al Momenin*. Qom: Noor vahy; 2010.
13. Ibn Sina H. *Canon of Medicine*. Beirut: Dar Ehya al-tarath al-arabi; 2005.
14. Nazem Jahan MA. *Exire Azam*. Tehran: Iran University of Medicine; 2008.
15. Levey AS, Coresh J. Chronic kidney disease. *Lancet*. 2012;379:165–180.
16. Sudarshan Ballal H, Augostine R, Vishvanath S. Chronic kidney disease. In: *Manual of Nephrology*. Jaypee Brothers, Medical Publishers Pvt. Limited; 2016:128–147.
17. Fukuda M, Motokawa M, Miyagi S, et al. Polynocturia in chronic kidney disease is related to natriuresis rather than to water diuresis. *Nephrol Dial Transplant*. 2006;21:2172–2177.
18. Barratt J, Topham P, Harris KPG. *Oxford Desk Reference*. Nephrology Oxford University Press; 2008.
19. Amanzadeh J, Reilly RF. Hypophosphatemia: an evidence-based approach to its clinical consequences and management. *Nat Clin Pract Nephrol*. 2006;2:136–148.
20. Aycock RD, Kass DA. Urine: abnormal color. *South Med J*. 2012;105:43–47.
21. Davran R, Helvaci MR, Davarci M. Left renal atrophy. *Int J Clin Exp Med*. 2014;7:1603.
22. Textor SC, Wilcox CS. Renal artery stenosis: a common, treatable cause of renal failure? *Annu Rev Med*. 2001;52:421–442.
23. Herring JC, Enquist EG, Chernoff A, Linehan WM, Choyke PL, Walther MM. Parenchymal sparing surgery in patients with hereditary renal cell carcinoma: 10-year experience. *J Urol*. 2001;165:777–781.
24. Loffroy R, Guiu B, Watfa J, Michel F, Cercueil J, Krausé D. Xanthogranulomatous pyelonephritis in adults: clinical and radiological findings in diffuse and focal forms. *Clin Radiol*. 2007;62:884–890.
25. Clark D, Georgitis J, Ray F. Renal arterial injuries caused by blunt trauma. *Surgery*. 1981;90:87–96.
26. McAfee JG. Radionuclide Imaging in the assessment of primary chronic pyelonephritis 1. *Radiology*. 1979;133:203–206.
27. Emmett JL, Alverez-Ierena JJ, McDonald JR. Atrophic pyelonephritis versus congenital renal hypoplasia. *J Am Med Assoc*. 1952;148:1470–1477.
28. Brull L, Dumont-Ruyters L, Firket J. Rickets and renal infantilism. *Rev Belge Sci Medicales*. 1941;13:129–150.
29. Caps MT, Zierler RE, Polissar NL, et al. Risk of atrophy in kidneys with atherosclerotic renal artery stenosis. *Kidney Int*. 1998;53:735–742.
30. Adachi T, Sugiyama N, Yagita H, Yokoyama T. Renal atrophy after ischemia–reperfusion injury depends on massive tubular apoptosis induced by TNFα in the later phase. *Med Mol Morphol*. 2014;47:213–223.
31. Chen Y-p. Experiences on the integrative medical diagnosis and treatment of acute kidney injury. *Chin J Integr Med*. 2010;16:207–212.
32. Goodyer P. Tubular disorders. In: *Manual of Pediatric Nephrology*. Springer; 2014:231–248.
33. Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J. *Harrison's Principles of Internal Medicine*. 19th ed. United States: McGraw-Hill Companies; 2015.
34. Jorjani EIH. *Zakhireye Khwarazmshahi*. Ghom: Ehyaye Tebe Tabiee; 2012.
35. Shah Arzani Mir Mohammad AM. *Teb-e-Akbari*. Qom: Ehyaye Tebe Tabiee; 2009:1182.
36. Harvey KS. Nutrition and pharmacologic approaches. In: *Nutrition in Kidney Disease*. Springer; 2008:191–226.
37. Kalantar-Zadeh K. Patient education for phosphorus management in chronic kidney disease. *Patient Prefer Adherence*. 2013;7:379–390.
38. Ghahraman AOA. *Matching the Old Medicinal Plant Name with Scientific Terminology*. Tehran: Tehran University Publisher; 2004.
39. Neuringer M, Connor WE, Van Petten C, Barstad L. Dietary omega-3 fatty acid deficiency and visual loss in infant rhesus monkeys. *J Clin Investig*. 1984;73:272.
40. Mawa S, Husain K, Jantan I. *Ficus carica* L. (Moraceae): phytochemistry, traditional uses and biological activities. *Evid Based Complement Altern Med*. 2013;2013.
41. Ghrairi F, Lahouar L, Brahmi F, Ferchichi A, Achour L, Said S. Physicochemical composition of different varieties of raisins (*Vitis vinifera* L.) from Tunisia. *Ind Crops Prod*. 2013;43:73–77.
42. Kumar KS, Bhowmik D. Traditional and medicinal uses of banana. *J Pharmacogn Phytochem*. 2012;1.
43. Thomson I, Asadi karam GR, Salem Z, et al. Biochemical Compounds and Nutritional Roles of the Foods Explained in the Qur'an. *Pak J Nutr*. 2009;8:13–19.
44. Jukanti AK, Gaur PM, Gowda C, Chibbar RN. Nutritional quality and health benefits of chickpea (Cicer arietinum L.): a review. *Br J Nutr*. 2012;108:S11–S26.
45. McDougall J. Nuts come in hard shells—for reasons. In: *McDougall Newsletter*. November 2009.
46. Aghaabasi K, Baghizadeh A. Investigation of genetic diversity in flixweed (*Descurainia sophia*) germplasm from Kerman province using inter-simple sequence repeat (ISSR) and random amplified polymorphic DNA (RAPD) molecular markers. *Afr J Biotechnol*. 2015;11:10056–10062.
47. Kritchevsky D, Weber MM, Klurfeld DM. Dietary fat versus caloric content in initiation and promotion of 7, 12-dimethylbenz (a) anthracene-induced mammary tumorigenesis in rats. *Cancer Res*. 1984;44:3174–3177.

48. Brufau G, Boatella J, Rafecas M. Nuts: source of energy and macronutrients. *Br J Nutr*. 2006;96:S24–S28.
49. McCord BA. *Vegetarian Cooking for Starters*. Crystal Clarity Publishers; 2002.
50. Bencini R, Pulina G. The quality of sheep milk: a review. *Anim Prod Sci*. 1997;37:485–504.
51. Raben A, Agerholm-Larsen L, Flint A, Holst JJ, Astrup A. Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but not on appetite and energy intake. *Am J Clin Nutr*. 2003;77:91–100.
52. Reilly Lukela J, Van Harrison R, Jimbo M, Mahallati A, Saran R, Sy AZ. Management of chronic kidney disease. In: *Guidelines for Clinical Care Ambulatory*. 2014:1–27. University of Michigan: Clinical alignment & performance excellence. Taubman Medical Library.
53. Harvey KB, Blumenkrantz MJ, Levine SE, Blackburn GL. Nutritional assessment and treatment of chronic renal failure. *Am J Clin Nutr*. 1980;33:1586–1597.
54. Yuvaraj A, Vijayan M, Alex M, Abraham G, Nair S. Effect of high-protein supplemental therapy on subjective global assessment of CKD-5D patients. *Hemodial Int*. 2016;20:56–62.
55. Ihle BU, Becker GJ, Whitworth JA, Charlwood RA, Kincaid-Smith PS. The effect of protein restriction on the progression of renal insufficiency. *N Engl J Med*. 1989;321:1773–1777.
56. Kovesdy CP, Kalantar-Zadeh K. Back to the future: restricted protein intake for conservative management of CKD, triple goals of renoprotection, uremia mitigation, and nutritional health. *Int Urol Nephrol*. 2016;48:725–729.
57. Jiang Z, Zhang X, Yang L, Li Z, Qin W. Effect of restricted protein diet supplemented with Keto analogues in chronic kidney disease: a systematic review and meta-analysis. *Int Urol Nephrol*. 2016;48:409–418.
58. Beto JA, Schury KA, Bansal VK. Strategies to promote adherence to nutritional advice in patients with chronic kidney disease: a narrative review and commentary. *Int J Nephrol Renovascular Dis*. 2016;9:21.