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Assessment of Some Biochemical Parameters in Chronic Kidney Disease (CKD) Patients Attending Usmanu Danfodiyo University Teaching Hospital, Sokoto

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In chronic kidney disease (CKD), inadequate and poor nutrition are important factors affecting the outcome and recovery from disease and injury. This study was designed to assess some nutritional indices of patients with CKD attending UDUTH Sokoto. Seventy-five CKD patients and twenty-five controls were recruited, parameters such as serum albumin, total protein, haemoglobin, vitamin D, zinc and copper were determined using standard methods. A significant ($P<0.05$) increase in Urea, Creatinine, Uric acid, phosphate, copper and potassium was observed in the patients as compared to the controls. There was a significant ($P< 0.05$) decrease in sodium, chloride, calcium, bicarbonate and albumin of the patients when compared to the control. However, there was no significant difference ($p>0.05$) observed in the total protein levels of the patients and the control. Therefore, these assessed parameters plays important role for the screening of malnutrition in CKD patents and if this is established, nutritional intervention can be made to these patients to minimize or prevent the advances of malnutrition problems in the CKD patients.

Keywords: Malnutrition, Chronic kidney disease, Biochemical markers.

1. Introduction

Chronic kidney disease (CKD), characterized by a gradual and irrespirable loss of renal function, is a worldwide public health problem (WHO, 2002). It is a structural or functional abnormalities of the kidney with or without decrease in glomerular filtration rate (GFR) of not less than three months and also kidney damage of glomerular filtration rate < 60mL/min/1.73m2 of the body surface area (BSA) for not less than three months with or without kidney damage (NKF/KDOQI 2008).

CKD is one of the world's public health problem rising globally (Makusidi *et al*., 2014). About 10% of the world population are affected by CKD with a high population of sub Saharan Africans that are mainly young adults between 20-50 years (Gama *et al*., 2012). CKD is asymptomatic in its early stage thereby causing a delay in early detection and the missing opportunities in preventing the progression of the disease to end stage renal disease (ESRD) that requires renal replacement therapy (Ngwobia *et al*., 2018). Renal replacement therapy (RRT) which include kidney transplant and dialysis that is available as hemodialysis and peritoneal dialysis are unaffordable or unavailable (Makusidi *et al*., 2014).

In Nigeria, studies conducted in small several hospitals and small scale communities reported a high burden of CKD across the country (Oluyombo *et al*., 2017). This high burden of ESRD is a result of poor healthcare services, poverty, ignorance, inadequate manpower and infrastructures (Ngwobia *et al*., 2018). Patients with CKD present a variety of metabolic and nutritional abnormalities (Heimburger *et al.,* 2000). An early diagnosis and proper treatment of these conditions, including dietary interventions, can slow down the progression of disease symptoms (kovesdy,2013) and may also ameliorate disease complications such as hyperkalemia and acidosis (Debrito *et al*.,2009). Metabolic and nutritional abnormalities arise in CKD from both pathophysiological and induced causes such as polypharmacy, uremic toxicity, altered metabolism and dietary restriction.

Additionally, patients with renal disease are also more vulnerable to malnutrition due to several factors which include poor appetite, various comorbidities, inflammation and infection. With the start of dialysis, some of these abnormalities are improved, but others remain or worsen, while new factors such as amino acid catabolism and inappropriate dialyzer's may likely contribute to malnutrition and protein-energy wasting (PEW) in this patients (Carrero *et al*., 2013). Increased risk of malnutrition may lead to increase in morbidity and mortality among CKD patients (Stenvinkel *et al*., 2002).

Assessing the nutritional status of CKD patients is of great benefit on their clinical condition and will aid in minimizing conditions that contributes to metabolic complications. However, there is no specific marker of nutritional status in CKD patients. Anthropometric parameters such as height, weight, BMI, skin fold thickness, lean body mass and fat mass assessment are commonly used because they are easy and affordable. However, these parameters lack specificity and reliability (Sarkar *et al*., 2005). The biochemical markers such as serum albumin, total protein, prealbumin, Total cholesterol, LDL cholesterol, HDL cholesterol and hemoglobin are early detectors of malnutrition in CKD (Okwonu *et al*., 2017). However, these parameters can have limitations due to the nature of CKD. Example hypoalbuminemia may be due to acute inflammation therefore, may not be a true marker of nutritional status in CKD. They are also expensive, rarely available and impractical for routine use (Jerin, 2003).

Compared with other methods of nutritional assessment (Anthropometric, Clinical and Dietary evaluation methods), biochemical tests provide the most objective and quantitative data on nutritional status. These tests often can detect nutrient deficits long before anthropometric measures are altered and clinical signs and symptoms appear. Some of this test are useful indicators of recent nutrient intake and can be used in conjunction with dietary methods. Biochemical tests are a valuable adjunct in assessing and managing nutritional status. However, a variety of pathologic conditions, use of certain medications and problems in sample handling can affect test result and they are not specific. Additionally, no single test 3 is sufficient enough to monitor nutritional status therefore biochemical tests must be used in conjunction with dietary evaluation, anthropometric measures and clinical methods.

2. Materials and Methods

2.1 Study area

The study was carried out at Usmanu Danfodiyo University Teaching Hospital (UDUTH), Sokoto. This is a tertiary referral Centre with a bed capacity of 850 and a catchment population of about 20 million covering Sokoto, Kebbi, Zamfara, Niger and Katsina States of Nigeria. The hospital runs medical outpatient clinics managed by the Internal Medicine Department. It has established Nephrology unit and Chemical pathology department.

2.2 Chemicals and reagents

All chemicals and reagents used were of analytical grades.

2.3 Inclusion criteria

i. CKD patients
ii. Adult age ≥ 18 years
iii. Those that consented

2.4 Exclusion criteria

i. CKD patients with malignancy
ii. 2.CKD patients with chronic inflammatory conditions (Tuberculosis, SLE, Rheumatoid arthritis)
iii. CKD patients on parenteral supplementation
iv. CKD patients with Nephrotic syndrome
v. HIV Positive patients with CKD

2.5 Study protocol

Informed consent was obtained from each participant. Demographic, clinical and laboratory data of interest would be collected by the investigator using a structured open ended proforma.

2.6 Ethical approval.

Ethical approval was sought and obtained at Usmanu Danfodiyo University Teaching Hospital Sokoto.

2.7 Blood sample collection.

Five mls of blood were drawn from patient with accurate diagnosis of CKD in Medical outpatient department (MOPD), Dialysis centre and/or admitted into Medical wards of Usmanu Danfodiyo University Teaching Hospital, Sokoto. Control were also recruited from the same hospital community mainly patient's relatives, hospital staff and students. The samples were allowed to clot and centrifuged for 15 min at 2000 rpm and serum stored immediately at -80˚C until analysis.

2.8 Methodology for eGFR calculation

Formula used for calculating the GFR was the MDRD Formula (Levey *et al*., 1999). The formula

was derived from the Modification of Diet in Renal Disease Trial.

GFR = 1.863 × serum creatinine (µmol/L) × age × 1.212 (if patient is black) × 0.742 (female)

2.9 Determination of some biochemical parameters

a. Serum uric acid was determined using uricase tops method (Jung and Parekh, 1970)
b. Serum calcium was determined using calcium – cresolphthaline complexone colorimetric method (Young, 1991)
c. Serum phosphate was determined using colorimetric method.
d. Serum total protein was determined using direct biuret method (Ouweland and Church, 2007).
e. Serum albumin was determined using bromo cresol green methodology (Doumasa *et al.,* 1971).
f. Serum creatinine was determined using jaffe's method (Barthels and Bohmer, 1971).
g. Serum urea was determined using urease berthelot method (Young, 1997).
h. Serum electrolytes (sodium and potassium) were measured using flame photometry (Chees brough, 1991).
i. Chloride ion was determined as described in Cheesbrough (1991).
j. Serum bicarbonate was determined as described in Van skyle *et al*. (2013).
k. Serum zinc was determined as described in Johnsen and Eliasson (1987).
l. Serum copper was determined using Dibrom PAESA method (Abe *et al*., 1989).
m. Serum vitamin d was determined using ELISA method (Holick 2009).
n. Serum haemoglobin was determined as described in Dancier and Lewis (1991).
o. Haemoglobin (Hb) contents of the sample was estimated by Cyanomethaemoglobin method (Dancier and Lewis 1991).

3. Results and Discussion

3.1 Results

3.1.1 Biochemical parameters.

Urea, creatinine, uric acid and potassium ion were elevated in CKD patient when compared to that of the controls while sodium, calcium and chloride levels were low as compared to that of the control as shown in Table 1.

Table 1: Biochemical parameters

Parameters	CKD (n=75)	Control (n=25)
Urea (mmol/L)	17.31±1.426[a]	4.78±0.141[b]
Creatinine (mg/dl)	18.21±1.126[a]	0.732±0.081[b]
Uric Acid (mg/dl)	6.424±0.337[a]	4.076±0.218[b]
Na^+(mmol/L)	130.44±1.240[a]	134.12±1.239[b]
K^+ (mmol/L)	4.928±0.144[a]	4.42±0.092[b]
Cl^- (mmol/L	92.66±1.043[a]	97.48±0.907[b]
Ca^+ (mmol/L)	2.072±0.076[a]	2.348±0.014[b]
HCO^-_3 (mmol/L)	18.48±0.592[a]	25.64±0.908[b]

Values are mean ± SEM. Mean value with different superscript letters in rows are significantly different from the control ($p<0.05$).

Key: CKD – Chronic kidney disease, Na^+ Sodium, K^+-Potassium ion, Cl-Chloride, Ca-Calcium, HCO^-_3-Bicarbonate

3.1.2 Nutritional parameters

Table 2 shows how markers of nutritional status in the table reflect the nutritional status of CKD patients when compared to that of the controls.

Table 2: Nutritional parameter

Parameters	CKD (n=75)	Control
Albumin (g/dl)	*3.394±0.104[a]*	4.436±0.061[b]
Total Protein (g/dl)	*7.008±0.175[a]*	7.172±0.151[a]
Vitamin D (ng/dl)	*35.819±2.399[a]*	52.410±3.677[b]
PO_4^{-3} (mmol/L)	*2.486±0.116[a]*	1.760±0.039[b]
Haemoglobin (g/dl)	*11.381±0.550[a]*	15.544±0.837[b]
Zn^+ (µg/dl)	*53.78±1.960[a]*	96.08±3.550[b]
Cu (µmol/l)	*151.133±5.681[a]*	130.56±5.015[b]
Calcium (mmol/L)	*2.257±0.064[a]*	2.261±0.019[b]
Albumin/Globulin Ratio (g/dl)	*1.124±0.112[a]*	1.867±0.159[b]

Values are mean ± SEM. Mean value with different superscript letters in rows are significantly different from the control ($p<0.05$).

Key: **PO_4^{-3} –** phosphate, Zn^+ -Zinc, Cu- Copper.

3.2 Discussion

3.2.1 Urea and Creatinine

The high levels of urea and creatinine observed in this study might be due to the decline in the glomerular filtration rate in the subjects as a result of reduced renal clearance and decline in fluid and electrolyte balance. A study conducted in Dyal college Pakistan reported elevated levels of urea and creatinine Anee *et al*., (2011) and Emem *et al*., (2008) also reported high level of

serum creatinine in patients as compared to the controls which are all in line with my study. Elevated creatinine level signifies impaired kidney function or disease and therefore creatinine levels will rise in blood due to poor clearance of creatinine by the kidneys, body size, activity level and medications. Most of the clients for this study happens to have other primary cause of the disease and are on medications as shown in their questionnaire and proper monitoring and treatment is required as this factors may interfere with the results. When the kidneys are unable to remove urea from the blood, blood urea nitrogen increases. Other factors like heart failure, dehydration or diet high in protein can increase urea level and protein catabolism. The levels of urea and creatinine rises as the kidney disease progresses and the elevation of the creatinine and urea indicates renal dysfunction (Cotran *et al*., 2005).

Uric acid level observed was still within the normal range as compared with the controls. In CKD uric acid are not get rid by the kidney and form crystals might cause other complications like kidney stones, arthritis, and gout which lead to severe pain, nausea and vomiting.

3.2.2 Electrolytes

The electrolyte abnormalities found in this study were significantly ($p< 0.05$) low level of sodium, chloride and bicarbonate the CKD patients as compared with the controls but they still happen to be within the normal range. Elevated levels of potassium were also observed in the patients as compared to the control despite the elevation the values are within the normal range. In this work the CKD patients have decreased levels of sodium and chloride which slight declines as the disease progresses and might be due appropriate nutrition and positive response to treatment / adequate diet. Metabolic acidosis also reduces hyperkalaemia in dialysis patients with improvement in their nutritional status, increased serum albumin, less protein catabolism, sensitivity of parathyroid hormones and serum calcium/bone turn over (Movilli *et al*., 2009). When the kidneys fail they can no longer remove excess potassium, resulting in reduces renal excretion and excessive or leakages of potassium from intracellular space this in turn causes weakness, numbness, nausea, difficult breathing and chest pain. Low levels of sodium (hyponatremia) might be as a result of insufficient water removal, excessive oral water consumption, illness and medications (Waikar *et al*., 2011). The decrease in sodium can result from relative increase in the amount of body water relative to sodium (Ashish *et al*., 2006).

The hypo bicarbonatemia observed in this study as shown in table 1 might be associated with reduction in blood pH and the low levels of bicarbonate is as a result of reduced capacity of kidney to excrete ammonia and H^+ ions Emem *et al*., (2008). In a study on seropositive patients in Nigeria reported low sodium level and increased potassium level in CKD patients as compared to the controls which is consistent to the present study. The present work is not the same with finding of (Obinna *et al*., 2015) in southeast Nigeria who reported an increased sodium level in patients as compared to controls, potassium level increased in patients with CKD than in controls and high bicarbonate levels in patients as compared to the controls. Low bicarbonate level in CKD patients could be due to adverse effect from protein, muscle and bone metabolism through negative nitrogen balance, protein degradation and decreased albumin synthesis (Meyring *et al*.,2016). Bicarbonate buffering system is the most important amongst blood buffers when the pH of the blood is considered. The regulatory mechanism of ionic charges and osmotic balance determines the level of electrolytes in the blood (Mckinley and Johnson, 2004). Significant alteration in the concentration of serum electrolytes such as sodium, potassium chloride and bicarbonate is an indication of renal impairment/poor renal functions (Corrand, 2012).

3.2.3 Albumin and Total protein

The low levels of serum albumin (hypoalbuminemia) in the present study is in line with the finding of Agaba *et al*., 2004 who reported hypoalbuminemia of 43.2% in CKD patient at university of Jos and also Liman *et al*., 2015 in National Hospital Abuja reported hypoalbuminemia of 24.2% hypoalbuminemia is a common complication in CKD patients caused by a combination of factors which include reduced synthesis and increased degradation of albumin over hydration, proteinuria and losses into dialysate (Agaba *et al*., 2004). Low serum albumin may also suggest infection in the kidneys Yakubu *et al*., (2003). Although inflammation was not assessed in this study, decrease in serum albumin is associated with chronic inflammation and inflammation promotes protein catabolism and decrease anabolism in CKD patients with increase in resting energy expenditure, protein catabolism, increase protein loss and decreased synthesis of albumin leads to Negative nitrogen balance and muscle wasting (Agaba *et al*., 2004). Hypoalbuminemia associated with chronic inflammation induces anorexia leading to malnutrition (Jankowska *et al*.,2017). The abnormalities in taste and effect of medications on taste also contributes to the decrease in nutrient intake. As several patients recruited for this research are under certain drugs like anti diabetic and anti-hypertensive. Therefore, serum albumin is not directly related

to malnutrition but rather inflammation is considered the major factor due to insufficient oncotic pressure that helps balance the movement of water from interstitial surfaces into circulation. in decrease in albumin level. Secondly blood albumin concentration differs with the amount of intravascular fluids therefore over hydration which is common in CKD can affect the actual concentration of albumin by lowering it (Fougue *et al*., 2007). Albumin level happens to be normal even in extreme cases of malnutrition such as marasmus. Total protein levels in CKD patients were low as compared to the control but still within the normal range. The difference between the serum total protein of the patients and that of the control is not much and fall within the normal range. This might be due to the common practice in the management of CKD especially those on haemodialysis to advice the patient on their protein intake and preferably should be of plant sources which may also be low in potassium and this might be the reason for having adequate levels of total protein in these patients.

The decrease in albumin globin ratio (A/G) ratio in CKD patients might be in response to low albumin or increased globulin. According to Stegmayr *et al*., (2014) low level of albumin globulin ratio can be as a result of over production of globulin in cases of myeloma or autoimmune diseases. The A/G ratio decrease in liver disease, decreased albumin production, renal disease and when albumin is lost in urine (Jankowska *et al*., 2017). Renal failure, decreased albumin production and albumin lost in the urine can be the cause of decreased A/G ratio because patient used this studies were confirmed deficient of the parameters.

3.2.4 Haemoglobin

Haemoglobin level in CKD patients was low when compared to the controls and to the normal range. This might be due to erythropoietin deficiency and or low levels of nutrients that are necessary for the red blood cells to produce haemoglobin such as iron, folic acid and vitamin B12 (Brugnara and Eckardt, 2011). The kidneys of patients recruited for this study might be damaged or in diseased state, and do not produce enough erythropoietin, as a result the bone marrow makes fewer red blood cells causing anaemia. Anaemia develops at early stage of CKD and worsens as CKD progresses to end stage as shown in Table 2 and the patients were classified according to stages of CKD using there estimated glomerular filtration rate. Anaemia can be as a result of iron deficiency or folate and vitamin B 12 deficiency. Iron deficiency anaemia happens when an individual does not consume enough iron in the diet which in turns makes red cells appear abnormal and usually pale and small in size. The pallor of the red cells reflects their low haemoglobin content leading to depression, hair loss, pale skin and lack of energy which can all contributes to malnutrition (Zimmerman and Hurell, 2007). Body needs folic acids for the synthesis, repair and methylation of DNA to produce healthy red blood cells also vitamin B12 plays an important role in supplying methyl groups for protein and DNA synthesis. These vitamins are also involved in cell growth and development. Deficiencies of folate and vitamin B12 causes red blood cells become too large or adequate amount of red blood cells not produced. A sore/red tongue, month ulcer and depression are associated to the deficiency of these nutrients and was observed from physical examination of the enrolled patients and are likely causes of malnutrition in this patients. (Fragasso, 2012). Other factors such as blood loss from haemodialysis inflammatory problems and chronic infection can contribute to haemoglobin deficiency causing weakness, fatigue, headache, paleness and cardiovascular problems. The result of this finding is similar to the of Anee *et al.,* (2011) who reported low level of haemoglobin and also the findings of Obinna *et al*., (2014) in Southern Nigeria who reported low levels of haemoglobin and also the findings of Okwuonu *et al*., 2017 in the Southeast Nigeria also reported low levels of haemoglobin. For the reasons mentioned above malnutrition in CKD patients can be associated with low levels of haemoglobin.

3.2.5 Vitamin D

Decreased vitamin D in chronic kidney disease patients is related to inadequate dietary intake of vitamin D, exposure to sunlight and the correlation of this factors with multiple disease that include bone metabolism and calcium haemostasis (Restrepo *et al*., 2016). Only few diet are naturally rich in vitamin D, therefore, vitamin D supplementation and exposure to sunlight is very vital as it aids in reducing hypovitaminosis D. Other factors such as skin colour obesity and age contributes to the CKD patients of this study from physical examination. In obsessed individual that is people with BMI of 30 and even greater needs more vitamin D. older people skin becomes less efficient at synthesizing this vitamin. Thus, age can contribute to vitamin D deficiency and this was observed during the study period. Older adults also tend to limit time in sun and may eat diets with insufficient amounts of vitamin D (Restrepo *et al*., 2016). Also chronic kidney disease patients have inability to convert vitamin D to its active form; as such vitamin D could not serve its vital purpose of balancing the calcium and

phosphate levels in the body which is achieved via absorption of this micro nutrient from food and regulation of parathyroid hormones.

3.2.6 Calcium and phosphate

Hypocalcaemia is associated with deficiency in mineral metabolism in general and specifically in CKD patients which is mainly due to derangement in the homeostasis of calcium phosphate and vitamin D (Chartsrisak *et al*., 2013). Deficiency of calcium can also be due to inadequate intake, poor calcium absorption and excess calcium loss. Poor nutrients intake affects bones and muscles causing rickets in children and osteomalacia in adults. Low level of calcium in serum may also be as a result of tetany. Increasing calcium solubility, increases the rate of the release of calcium from the bones into the extracellular fluids and these all are increasing load reaching the kidneys. This may increase the renal calcium loss and thereby causing prolonged acidosis. Dietary deficiencies with also in adequate calcitriol synthesis by deficient kidneys could also be the causative agent in reduced calcium in CKD patients. Rajbhandari et al., 2017 reported hypocalcaemia of 60.6% in their study which is in line with the present study. Therefore, malnutrition might be due to dietary defects which affect activation of calcitriol, absorption of calcium and hence low levels of calcium.

High level of phosphate in the CKD patients can reduce synthesis of active form of vitamin D and hence secondary to hyperparathyroidism and this can cause increase breakdown of vitamin D with decreased substrate for calcitrol synthesis (Nigwekar *et al*., 2014). High level of phosphate is majorly as a result of hypocalcaemia particularly in the plasma protein concentration and this is due to the fact that calcium and phosphate precipitation in the tissue can ensue when the phosphate/calcium plasma concentration exceed their solubility product thus causing metastatic calcification. As kidney failure progresses the damaged kidney does not respond to parathyroid hormones, phosphate continues to accumulate as less is filtered by the kidney on reaching to the end stage of the disease the phosphate levels of the patients declines and might be as a result of dietary restriction during dialysis and also the dialysis procedure as a whole .In line with the above statement serum phosphorus levels rises and dietary restriction, phosphate binding drugs and decreased vitamin D supplementation becomes necessary. High levels of phosphate in CKD patients cause joint pain, muscle pain, muscle weakness, itching and redness of eyes and in more severe cases have constipation, vomiting and diarrhoea. Result of this work is similar to the finding of rajbhandari *et al*., 2017 who reported hyperphosphatemia of 63.6% CKD patients in Bir hospital, Kathmandu Nepal.

3.2.7 Zinc and Copper

Low level of zinc in the CKD patients of the present study might be due to increased urinary zinc excretion, decreased intestinal zinc absorption, poor zinc intake, diet associated with attempt to prevent the progression of chronic kidney disease, hypoalbuminemia and medications (coombes *et al*., 2012). Zinc deficiency may also lead to anorexia (Suzuki *et al*, 2011) which is an appetite disorder that in turn causes inadequate brain growth and development depending principally on micronutrients and malnutrition and this disorder was observed from the physical examination of the subjects enrolled for this research. Zinc is an essential component of some protein and enzymes; it participates in the synthesis and breakdown of carbohydrates, lipids, proteins and nucleic acids. Hypoalbuminemia that was observed in this study may also be a contributory cause to zinc deficiency and hence malnutrition. Zinc has shown to also play vital role in polynucleotide transcription and translation as well as gene expression. (Obinna *et al*., 2015). Hypozincemia was observed in the study of Anee *et al*., 2011 in pakistan and that of tetiket *et al*., 1993. This micronutrient deficiencies, contributes to comorbidities such as anaemia, cardiovascular problems and metabolic in balance. The decrease intake of zinc, poor absorption and other causes of hypozincemia put CKD patients at risk of malnutrition (Jankowska *et al*., 2017).

Elevated Serum copper levels in CKD patients as compared to the control the elevation exceed the reference value of copper. The high levels of copper might be associated to dialysate combination and if not controlled over long period of time it can lead to acute or temporary copper poisoning causing nausea, vomiting, diarrhoea, anaemia and kidney damage. Copper is an essential trace element that has positive effects on metabolisms and energy production in the body for growth and development. It is also vital to bone health, nervous system function, iron level and red blood cell production.

A study by Sholman *et al*., (2014) reported copper levels of CKD patients to be normal when compared to the controls that of Rajashri *et al*., (2013) reported low levels of coppers. The above findings contradict the present study. Despite the benefits of copper high levels can lead to acute and temporary copper poisoning with symptoms that are associated with malnutrition in CKD.

4. Conclusion

The biochemical parameters like zinc, vitamin D, Albumin and haemoglobin were low and others like copper and phosphate were elevated. Parameter like total protein and calcium were normal. Even though the level of malnutrition cannot be asserted as the parameters utilized cannot alone provide accurate information of the nutritional status of individual, but theses parameters plays an important role in screening of malnutrition in CKD patients and if this is established, nutritional intervention can be made to these patients to minimize or prevent the advances of malnutrition problems in the CKD patients.

Conflict of Interest

The author declares that there is no conflict of interest.

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