International Journal of Research in Medical Sciences
Addanki RJD et al. Int J Res Med Sci. 2023 Oct;11(10):3945-3946
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20233068

Letter to the Editor

Role of silica nanoparticles in pathogenesis of Mesoamerican nephropathy

Sir,

The authors have done an excellent job in shedding light on Mesoamerican nephropathy (MeN), a lesser-known variant of chronic kidney disease that affects agricultural workers in rural areas especially regions with hot climates like Central America, etc. The inclusion of social factors in the pathogenesis and highlighting the risks posed by nephrotoxic drugs and heat stress leading to subclinical rhabdomyolysis and uricosuria are crucial contributions to the understanding of MeN.[1]

Furthermore, distinctive preventive techniques, such as measuring body weight to assess dehydration status have been emphasised.[1] Early diagnosis and prompt referrals can significantly improve outcomes for patients with MeN.[1]

We would like to mention another possible cause of MeN among sugarcane field workers-the potential nephrotoxic effects of amorphous silica nanoparticles (SiNP) produced as a result of burning sugarcane. Several studies have demonstrated that inhaled SiNP can translocate into the systemic circulation which is the starting point in the pathogenesis SiNP mediated MeN.[2]

Here are some of different mechanisms by which silica can cause renal damage after entering systemic circulation (Figure 1): Silica generates reactive oxygen species (ROS) within cells, leading to oxidative stress and cell damage of renal tubular epithelium.[3] Uptake of SiNP by renal proximal tubular cells, leads to damage of host cell membranes and lysosomes within. Accumulation of these damaged lysosomes, potentially causes renal cell injury. In addition, impaired autophagy in autophagy-deficient proximal tubules worsen kidney damage.[4] Mitochondrial dysfunction and disruption of metabolic activity in human proximal tubular cells.[5] Impairment of vascular homeostasis and induction of systemic inflammation, contributing to kidney injury.[6] Persistent accumulation of amorphous siNP in kidneys, causes chronic inflammation and fibrosis.[7]

To summarise, SiNP exposure can cause apoptosis of tubular cells, tubular atrophy, interstitial fibrosis, and irreversible renal damage (chronic kidney disease) which has been proven in a study conducted by Mascarenhas et al.[8]

Results in an animal study conducted by Sasai et al adds weight to the argument by presenting compelling evidence of the potential role of SiNP in causing kidney injury. The study demonstrates histological similarities between the kidney injury induced by SiNP and chronic kidney disease of unknown etiology (CKDu). The observed slow disease progression and significant tubulointerstitial involvement in the animal model closely resemble the characteristics of CKDu. Furthermore, the fact that kidney injury persisted even after stopping silica administration reinforces the notion that silica could be a significant contributor to the development of CKDu.[9] They have also demonstrated ability of silica isolated from sugarcane ash to induce kidney disease, this provides substantial support for the hypothesis that SiNP play a role in CKDu, thereby adding credibility and strength to the argument.[9]

In conclusion, we believe that adding the nephrotoxic effects of amorphous SiNP from burning sugarcane to the causation of MeN would significantly enhance its overall understanding and implications.

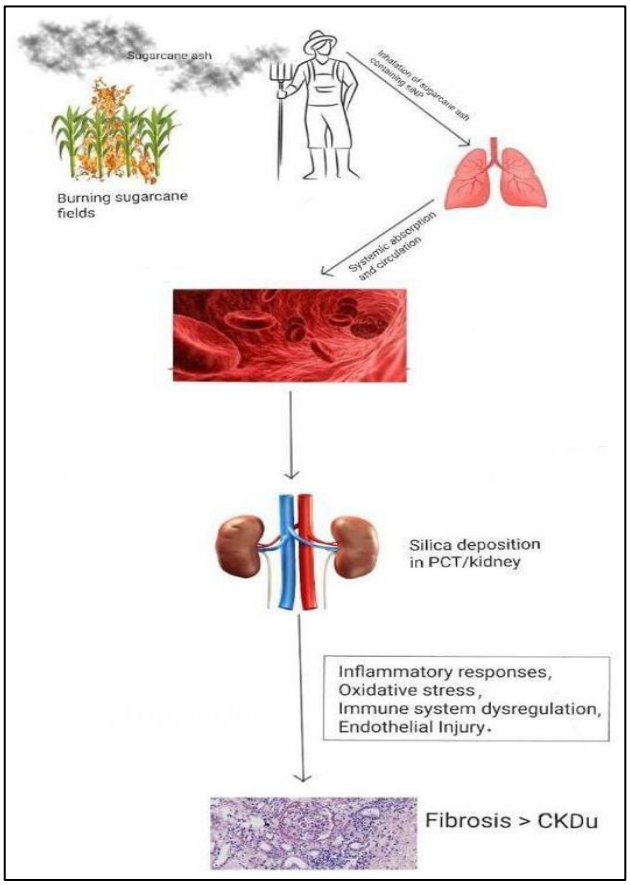

Figure 1: Pathogenesis of SiNP leading to CKDu.

REFERENCES

1. Oza YR, Mannava AS, Tousif MJ, Shah JK, Gondalia SV, Mevawala HB et al. Mesoamerican nephropathy: a narrative review. Int J Res Med Sci. 2023;11:3100-4.
2. Nemmar A, Vanbilloen H, Hoylaerts MF, Hoet PH, Verbruggen A, Nemery B. Passage of intratracheally instilled ultrafine particles from the lung into the systemic circulation in hamster. Am J Respiratory Crit Care Med. 2001;164(9):1665-8.
3. Mahmoud AM, Desouky EM, Hozayen WG, Bin-Jumah M, El-Nahass ES, Soliman HA et al. Mesoporous Silica Nanoparticles Trigger Liver and Kidney Injury and Fibrosis Via Altering TLR4/NF-κB, JAK2/STAT3 and Nrf2/HO-1 Signalling in Rats. Biomolecules. 2019;9(10):528.
4. Maejima I, Takahashi A, Omori H, Kimura T, Takabatake Y, Saitoh T et al. Autophagy sequesters damaged lysosomes to control lysosomal biogenesis and kidney injury. EMBO J. 2013;32(17):2336-47.
5. Stem AD, Rogers KL, Roede JR, Roncal-Jimenez CA, Johnson RJ, Brown JM. Sugarcane ash and sugarcane ash-derived silica nanoparticles alter cellular metabolism in human proximal tubular kidney cells. Environmental Pollution (Barking, Essex : 1987). 2023;332:121951.
6. Nemmar A, Albarwani S, Beegam S, Yuvaraju P, Yasin J, Attoub S, Ali BH. Amorphous silica nanoparticles impair vascular homeostasis and induce systemic inflammation. Int J Nanomed. 2014;9:2779-89.
7. Iavicoli I, Fontana L, Nordberg G. The effects of nanoparticles on the renal system. Crit Rev Toxicol. 2016;46(6):490-560.
8. Mascarenhas S, Mutnuri S, Ganguly A. Deleterious role of trace elements-Silica and lead in the development of chronic kidney disease. Chemosphere. 2017;177:239-49.
9. Sasai F, Rogers KL, Orlicky DJ, Stem A, Schaeffer J, Garcia G et al. Inhaled silica nanoparticles cause chronic kidney disease in rats. Am J Physiology. 2022;323(1):F48-58.

Raja Narendra Divakar Addanki*,
Anjali Srikanth Mannava

Department of Medicine, All India Institute of Medical Sciences, Raipur, Chhattisgarh, India

***Correspondence to**
Dr. Raja Narendra Divakar Addanki,
E-mail: rajanarendradivakaraddanki@gmail.com

Cite this article as: Addanki RND, Mannava AS. Role of silica nanoparticles in pathogenesis of Mesoamerican nephropathy. Int J Res Med Sci 2023;11:3945-6.