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From Workplace to Clinical Nephrology: Integrating Occupational Risk into Chronic Kidney Disease Prevention

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INTRODUCTION

Chronic Kidney Disease (CKD) is a major global health concern and one of the fastest-growing causes of mortality worldwide. According to the Global Burden of Disease (GBD) 2021 study, there were approximately 673.7 million prevalent cases of CKD, 19.9 million new cases, 1.53 million deaths, and 44.5 million disability-adjusted life years (DALYs) attributable to CKD in 2021 [1]. These numbers represent striking increases compared to 1990, indicating CKD as not only a clinical but also a societal and economic challenge.

Traditionally, diabetes, hypertension, and chronic glomerulonephritis have been considered the predominant etiologies. However, an increasing body of evidence suggests that occupational exposures also play a substantial role in kidney injury. Workers in industries with exposure to nephrotoxic chemicals, excessive heat, or physically demanding schedules may develop CKD even in the absence of conventional risk factors. The phenomenon of CKD of unknown etiology (CKDu), reported among agricultural workers in Central America and South Asia, provides a striking example. This condition, often occurring in young laborers without diabetes or hypertension, showed how work-related exposures-particularly recurrent heat stress, dehydration, and vigorous physical exertion-can precipitate progressive kidney disease. Such patterns challenge the traditional framework of CKD prevention and demand broader recognition of occupational risk.

DESCRIPTION

Occupational risk factors

A recently published comprehensive review summarized current evidence on occupational risk factors for kidney disease, suggesting heavy metals, organic solvents, heat stress (high environment temperatures), and psychosocial workload as key determinants of renal outcomes [2].

Heavy metals: Long-term exposure to heavy metals, such as lead, cadmium, mercury, and chromium is well documented as nephrotoxic, leading to diverse renal injury like acute tubular damage, proteinuria, and glomeruolsclerosis. Workers in industries such as mining, welding, and battery manufacturing demonstrate earlier declines in renal function and proteinuria compared to unexposed populations. These metals accumulate in renal tissue, especially in the proximal tubules, inducing oxidative stress and tubular injury that may progress to tubulointerstitial fibrosis and tubular atrophy and lead to CKD.

Organic solvents and pesticides: Solvents such as trichloroethylene and certain hydrocarbons, particularly methanol and ethylene glycol and their metabolic byproducts, have been associated with tubular necrosis and chronic glomerular injury. Long-term exposure in industrial settings correlates with higher risks of end-stage renal disease. In agriculture, the widespread use of pesticides and agrochemicals significantly increases CKD risk, with the physical strain of labor further amplifying this vulnerability.

Heat stress and dehydration: Recurrent thermal strain and dehydration, particularly in outdoor labor under high ambient temperatures, can cause repeated subclinical kidney injury. The mechanisms related to heat-induced renal damage are multifaceted and involve a combination of dehydration, physical exertion, and rhabdomyolysis [2]. Chronic dehydration combined with persistent hear stress is thought to lead to repeated episodes of acute renal injury that eventually evolve into CKD as exemplified by Mesoamerican nephropathy among sugarcane workers [3]. Climate change is expected to increase the number of workers exposed to hazardous heat stress, making this an urgent occupational risk factor.

Psychosocial workload: Occupational stress, long working hours, and irregular shift schedules can also indirectly compromise renal health. Chronic activation of stress responses in the workplace can result in elevated blood pressure, increased release of pro-inflammatory cytokines, hormonal imbalances (cortisol and melatonin), and enhanced oxidative stress, which are associated with CKD outbreaks and exacerbations. Evidence suggests that individual engaged in night-shift work, especially women, are at an elevated risk of developing CKD through hormonal imbalances [4]. Recent prospective study suggests that multiple adverse work factors act additively, nearly doubling CKD risk compared to low-risk occupational groups [5].

Clinical and public health implications

The recognition of occupational kidney disease has far-reaching implications for both clinical practice and public health. At the clinical level, nephrologists and primary care physicians must integrate occupational history into routine evaluation. This practice allows for earlier detection of at-risk individuals and timely intervention. If renal function decline may appear even without conventional risk factors, nephrologists should routinely consider occupational exposure when evaluating unexplained CKD. From a societal perspective, the burden of occupation-related CKD extends beyond health outcomes. The disease disproportionately affects economically active populations, creating productivity losses, household financial strain, and rising healthcare costs. Addressing occupational CKD therefore contributes to both health equity and economic stability. Public health systems must establish early-warning and surveillance mechanisms in vulnerable populations, such as agricultural laborers and construction workers. By systematically capturing occupational information in registries and electronic health systems, clinicians and policymakers can identify emerging clusters rapidly and respond with preventive measures. Moreover, international collaboration is essential to address cross-border labor migration and climate-driven shifts in exposure risk.

Future Perspectives

The emergence of occupation-related CKD signals that traditional approaches to kidney disease prevention are no longer sufficient. Moving forward, cross-sectoral research must be prioritized, linking nephrology, occupational medicine, climate science, and labor economics to better define causal pathways and identify biomarkers for early detection. At the same time, occupation and exposure histories should be systematically integrated into national health records and CKD surveillance systems to ensure proactive monitoring.

Given the accelerating impact of climate change, preparedness for heat stress and related risks is critical. Preventive strategies for heat-induced renal damages, such as hydration programs, shaded rest areas, and climate-adapted work schedules, must be embedded within occupational health regulations. Physicians should also play an active role as advocates and leaders, engaging with policymakers and industry stakeholders to secure safer work environments and equitable labor protections.

Educational initiatives are equally important. Incorporating occupational health literacy into medical training will equip future clinicians to respond effectively to exposure-driven kidney disease. Communities and patients must also be empowered with knowledge that enables them to take protective measures for their kidney health.

CONCLUSION

In conclusion, if these strategies are pursued, occupational kidney disease can serve not only as a warning but also as an opportunity to strengthen health systems and promote equity. By addressing upstream determinants of health, mobilizing cross-disciplinary collaboration, and leveraging the trusted voices of physicians, we can change the trajectory of occupational kidney disease and safeguard kidney health for future generations.

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