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HEAT EXPOSURE AND KIDNEY HEALTH: DEHYDRATION, ACUTE KIDNEY INJURY, AND PUBLIC HEALTH STRATEGIES IN A WARMING CLIMATE

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ABSTRACT

Background: Heat exposure is increasingly recognized as an environmental and occupational health concern with clinically relevant consequences for kidney health. The kidneys are particularly vulnerable to heat-related physiological stress because dehydration, reduced effective circulating volume, renal hypoperfusion, and tubular stress may contribute to acute kidney injury, especially in susceptible individuals.
Methods: This narrative review synthesizes peer-reviewed articles and public health documents identified through PubMed/MEDLINE, PMC, and institutional sources, including the World Health Organization and occupational health agencies. Priority was given to recent reviews, epidemiological studies, mechanistic papers, occupational health studies, and public health guidance relevant to heat-related kidney injury and prevention.
Results: Evidence indicates that high ambient temperature is associated with kidney-related morbidity, including acute kidney injury. Risk is particularly relevant among older adults, patients with chronic diseases, people using medications affecting fluid balance or renal perfusion, outdoor and manual workers, and physically active individuals. Repeated heat stress and dehydration may also contribute to chronic kidney disease risk in occupationally exposed populations, although causality and the relative contribution of environmental, occupational, and social determinants remain under investigation.
Conclusions: Heat-related kidney injury should be approached as a partially preventable clinical and public health problem. Prevention should extend beyond individual hydration advice and include clinical risk identification, medication awareness, occupational heat protection, access to cooling and safe drinking water, heat-health warning systems, and equity-oriented strategies for populations with limited control over heat exposure.

KEYWORDS
Heat Exposure, Dehydration, Acute Kidney Injury, Chronic Kidney Disease, Occupational Heat Stress, Public Health

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1. Introduction

Heat exposure is increasingly recognized as an important public health and occupational health concern, particularly during heat waves and among vulnerable populations. The World Health Organization identifies extreme heat as a major and growing health risk that can exacerbate chronic diseases and affect organs including the heart and kidneys (World Health Organization, 2026). Rising baseline temperatures and more frequent or intense heat events are expected to increase the health burden of heat exposure, including renal complications. Recent nephrology-focused reviews emphasize that heat has emerged as one of the most relevant climate-related threats to kidney health, especially in populations with limited adaptive capacity or occupational exposure (Barraclough, 2026).

The kidneys are particularly sensitive to heat-related physiological stress because they play a central role in fluid, electrolyte, and blood pressure regulation. During heat exposure, thermoregulatory responses increase cutaneous blood flow and sweating, which may reduce effective circulating volume and renal perfusion, especially when fluid losses are not adequately replaced (Chapman, Johnson, et al., 2021). Heat stress may also contribute to kidney injury through dehydration, hyperosmolality, vasopressin activation, oxidative stress, inflammation, and, in physically active individuals, rhabdomyolysis (Barraclough, 2026).

Acute kidney injury is the most clinically evident renal complication associated with heat exposure. In a nationwide case-crossover study from England including 1,354,675 AKI electronic alert episodes, high ambient temperature was associated with increased odds of AKI, with an odds ratio of 1.623 for a day with 32°C compared with 17°C; a 7-day heatwave in July 2021 was associated with a 28.6% increase in AKI counts (Hajat et al., 2024). These findings support the view that heat-related AKI is not limited to tropical climates or occupational hotspots but may also be relevant in temperate healthcare systems.

Repeated exposure to heat stress and dehydration may also contribute to CKD risk, particularly among workers performing physically demanding tasks in hot environments. However, the causal role of heat stress in chronic kidney disease of non-traditional origin remains under investigation, and risk is likely modified by workload, hydration status, socioeconomic conditions, nephrotoxic exposures, and access to prevention measures (Nerbass et al., 2017). Reviews of occupational heat exposure describe heat-related tubular injury as being exacerbated by dehydration, longer work duration, higher core temperature, and muscle-damaging exercise (Chapman, Hess, et al., 2021).

From a clinical perspective, heat-related kidney injury is important because it may present outside specialized nephrology settings. Patients may seek care in emergency departments, primary care clinics, occupational health services, or during routine follow-up for chronic diseases. The early clinical picture may be non-specific and include fatigue, dizziness, reduced urine output, muscle symptoms, orthostatic complaints, or laboratory findings such as dehydration, electrolyte disturbances, and rising creatinine. Therefore, heat exposure should not be treated only as an environmental background factor. It may represent a clinically relevant exposure that modifies the risk of AKI in susceptible individuals, particularly during heat waves or periods of prolonged physical work in hot conditions.

This narrative review aims to summarize current evidence on the relationship between heat exposure, dehydration, acute kidney injury, and repeated heat-related renal stress, with emphasis on vulnerable populations and kidney-protective public health strategies in a warming climate.

2. Methods

This article was designed as a narrative review rather than a systematic review or meta-analysis. The aim was to synthesize clinically and public-health-relevant evidence on the relationship between heat exposure, dehydration, acute kidney injury, chronic kidney disease risk, and prevention strategies in occupational and community settings.

Relevant literature was identified through searches of PubMed/MEDLINE, PubMed Central, and institutional sources, including documents from the World Health Organization and occupational health agencies. The search focused on combinations of the following terms: “heat exposure,” “heat stress,” “dehydration,” “acute kidney injury,” “kidney injury,” “chronic kidney disease,” “CKD of unknown etiology,” “occupational heat stress,” “agricultural workers,” “construction workers,” “heat wave,” and “public health prevention.” Priority was given to recent peer-reviewed reviews, epidemiological studies, mechanistic and physiological papers, occupational health studies, and public health guidance documents. Older sources were included when they provided foundational evidence or definitions.

The synthesis was structured around clinically relevant themes: physiological mechanisms linking heat exposure with renal stress, evidence for heat-associated AKI, the possible contribution of repeated heat stress to chronic kidney disease risk, vulnerable populations, and preventive strategies. Because the purpose of the review was to provide an integrative clinical and public health overview, no formal risk-of-bias assessment or quantitative pooling was performed. Therefore, the findings should be interpreted as a narrative synthesis of current evidence rather than as a systematic estimate of effect size.

3. Heat Exposure and Renal Physiology

The physiological response to heat exposure is primarily aimed at maintaining core body temperature. Cutaneous vasodilation increases heat dissipation through the skin, while sweating enables evaporative cooling. However, these thermoregulatory mechanisms may also increase water and electrolyte loss. When fluid replacement is insufficient, effective circulating volume may decrease, increasing renal physiological stress because the kidneys are central to water, electrolyte, and blood pressure regulation (Chapman, Johnson, et al., 2021).

Reduced effective circulating volume may decrease renal perfusion, particularly during prolonged heat exposure, dehydration, or physical exertion. In response, the kidneys activate water- and sodium-conserving mechanisms, including neurohormonal pathways that help maintain blood pressure and fluid balance. These adaptations are protective in the short term, but prolonged or repeated activation during heat stress may contribute to renal hypoperfusion, tubular stress, and susceptibility to acute kidney injury (Chapman, Johnson, et al., 2021). Barraclough describes heat-related kidney injury as involving reduced renal perfusion, volume depletion, vasopressin activation, oxidative stress, inflammation, and profibrotic signalling, particularly when heat exposure is recurrent or excessive (Barraclough, 2026).

Additional mechanisms may further increase renal vulnerability in specific contexts. Physical exertion in hot environments may contribute to muscle injury and rhabdomyolysis, while severe or prolonged heat stress may promote oxidative stress, inflammatory activation, and electrolyte disturbances (Barraclough, 2026). In a controlled study of prolonged wet-bulb heat exposure in healthy young men, higher wet-bulb temperature was associated with greater increases in urinary kidney injury biomarkers, renal oxidative stress markers, and plasma IL-17a, supporting the biological plausibility of oxidative and inflammatory pathways in heat-related kidney injury (Hess et al., 2022). These mechanisms help explain why dehydration, exertion, and prolonged exposure may act together to increase the risk of heat-related renal dysfunction.

4. Heat Exposure and Acute Kidney Injury

Acute kidney injury is defined by KDIGO as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours, an increase in serum creatinine to ≥1.5 times baseline within the previous 7 days, or urine volume <0.5 mL/kg/h for 6 hours (Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012). This definition is useful in the context of heat exposure because heat-related renal dysfunction may develop rapidly and may be missed if renal risk is not considered in susceptible patients after significant heat illness, dehydration, or reduced urine output.

Heat-related AKI is most plausibly linked to dehydration, reduced effective circulating volume, renal hypoperfusion, and tubular stress during prolonged heat exposure. These mechanisms may be intensified by physical exertion, inadequate fluid replacement, electrolyte disturbances, and rhabdomyolysis in exertional heat illness (Chapman, Johnson, et al., 2021). In this context, heat-related AKI should be understood as a

potentially preventable complication of environmental or occupational heat stress, rather than only as an incidental consequence of extreme weather.

Epidemiological studies increasingly support an association between high ambient temperature and kidney-related morbidity. A systematic review and meta-analysis of 11 studies found that high temperatures were associated with a 30% increase in kidney disease morbidity, supporting a broader relationship between elevated temperature and renal outcomes (Lee et al., 2019).

More specifically, high ambient temperature has been associated with increased AKI risk. In a nationwide case-crossover study from England including 1,354,675 AKI electronic alert episodes, high ambient temperature was associated with increased odds of AKI, with an odds ratio of 1.623 for a day with 32°C compared with 17°C; a 7-day heatwave in July 2021 was associated with a 28.6% increase in AKI counts (Hajat et al., 2024). These findings are particularly important because they demonstrate a measurable association between ambient heat and AKI in a temperate healthcare system, not only in tropical regions or occupational hotspots.

Individual susceptibility to heat-related AKI depends on age, baseline kidney function, comorbidities, medication use, hydration status, access to cooling, and occupational or recreational exposure. In a matched case-control study among older adults in Ontario, Canada, heat periods were associated with a modestly higher risk of hospital encounter with AKI (McTavish et al., 2018). This suggests that preventive strategies should focus not only on severe heatstroke, but also on earlier recognition of dehydration and renal risk in vulnerable populations during periods of high temperature.

5. Repeated Heat Stress and Chronic Kidney Disease Risk

Chronic kidney disease is defined by KDIGO as abnormalities of kidney structure or function, present for at least three months, with implications for health (Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2024). In the context of heat exposure, the transition from acute or subclinical renal stress to chronic kidney disease should be described cautiously, because CKD is multifactorial and usually develops over time.

Repeated episodes of dehydration, renal hypoperfusion, and subclinical or overt acute kidney injury may contribute to CKD risk, particularly when heat exposure is prolonged, recurrent, and combined with physical exertion. This pathway has been proposed mainly in occupational settings, where workers may experience repeated cycles of heat stress, inadequate recovery, and incomplete rehydration (Chapman, Hess, et al., 2021; Chapman, Johnson, et al., 2021). Barraclough describes recurrent or excessive activation of dehydration-related pathways as potentially maladaptive, promoting oxidative stress, inflammation, hypertension, profibrotic signalling, tubulointerstitial fibrosis, and glomerulosclerosis over time (Barraclough, 2026).

The relationship between occupational heat exposure and CKD has been most widely discussed in the context of chronic kidney disease of non-traditional origin. This condition has been reported in several occupational and geographic “hotspots”, particularly among agricultural and manual workers exposed to high temperatures, intense physical activity, and limited opportunities for rest and rehydration (Nerbass et al., 2017). However, the causal contribution of heat stress remains uncertain, and CKD of non-traditional origin should not be interpreted as a disease caused by heat alone. Current evidence supports a multifactorial interpretation, in which dehydration, workload, core body temperature, work duration, payment systems, socioeconomic vulnerability, pesticide exposure, water quality, access to clean water, and labour conditions may all modify risk (Barraclough, 2026; Nerbass et al., 2017).

Therefore, repeated heat stress may be considered a plausible contributor to CKD risk in susceptible and occupationally exposed populations, but the relative importance of heat compared with other environmental, occupational, and social determinants remains under investigation (Barraclough, 2026; Nerbass et al., 2017). This distinction is clinically important: heat-related AKI may be an acute and potentially preventable event, whereas CKD risk reflects cumulative exposure, repeated injury, incomplete recovery, and broader working and living conditions. Accordingly, the heat-stress hypothesis should be interpreted as one plausible pathway within a broader multifactorial framework that may also include workload intensity, socioeconomic vulnerability, limited access to healthcare, nephrotoxic exposures, infections, medication use, and barriers to adequate rest and hydration.

6. Vulnerable Populations

Heat-related kidney injury does not affect all individuals equally. Susceptibility depends on physiological reserve, baseline kidney function, comorbidities, medication use, hydration status, type of activity, occupational exposure, and access to cooling, rest, and fluids. Therefore, vulnerability should be understood not only as an individual clinical characteristic, but also as a consequence of environmental, occupational, and social conditions that influence heat exposure and the ability to respond to it (Barraclough, 2026; Nerbass et al., 2017). Identifying vulnerable populations is clinically important because heat-related renal stress may be preventable if risk is recognized before severe dehydration or acute kidney injury develops (Chapman, Johnson, et al., 2021).

6.1. Older adults

Older adults are particularly vulnerable to heat-related kidney injury because aging is associated with reduced physiological reserve, impaired thermoregulation, lower thirst perception, higher medication burden, and a higher prevalence of chronic diseases. During heat waves, older individuals may develop dehydration and electrolyte disturbances before symptoms are recognized, especially if they live alone, have limited access to cooling, or have cognitive or mobility limitations (Chapman, Johnson, et al., 2021). Large studies of heat wave-related hospital admissions among older adults have identified increased risks of fluid and electrolyte disorders, renal failure, and acute kidney failure, supporting the clinical relevance of renal monitoring in this population (Bobb et al., 2014; Hopp et al., 2018). In a matched case-control study from Ontario, Canada, heat periods were associated with a modestly higher risk of hospital encounter with AKI among older adults, supporting the relevance of heat-related renal risk even in northern climates (McTavish et al., 2018). This suggests that prevention should include early hydration support, heat alerts, access to cooling, medication awareness, and active monitoring of older adults during periods of high temperature.

6.2. Patients with chronic kidney disease

Patients with chronic kidney disease have reduced renal reserve and may be less able to compensate for dehydration, electrolyte shifts, reduced effective circulating volume, and decreased renal perfusion during heat exposure (Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2024). This vulnerability is clinically relevant because heat stress may increase sweating and insensible fluid losses, while dehydration-related activation of vasopressin and the renin–angiotensin–aldosterone system may further reduce cortical renal blood flow and increase tubular stress (Barraclough, 2026). In patients with pre-existing CKD, even a transient episode of volume depletion may therefore precipitate acute deterioration of kidney function, particularly when heat exposure is combined with vomiting, diarrhea, excessive sweating, poor oral intake, or nephrotoxic medication use.

Preventive advice in CKD should not be reduced to a simple recommendation to increase fluid intake. Some patients with CKD, heart failure, advanced age, or cardiovascular comorbidity may require individualized fluid and medication guidance because both dehydration and fluid overload can be clinically harmful (Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2024). Heat-related counselling should therefore focus on avoiding dehydration, recognizing warning symptoms such as dizziness, reduced urine output, orthostatic symptoms, confusion, or persistent weakness, reviewing nephrotoxic exposures such as non-steroidal anti-inflammatory drugs, and seeking medical assessment after significant heat illness or sustained reduction in urine output.

6.3. Patients with diabetes, hypertension, and cardiovascular disease

Patients with diabetes, hypertension, and cardiovascular disease may be at increased risk during heat exposure because these conditions can reduce cardiovascular and renal adaptive capacity, particularly during dehydration or acute illness (Barraclough, 2026; World Health Organization, 2026). Heat-related extracellular fluid volume loss can activate vasopressin and the renin–angiotensin–aldosterone system, reduce cortical renal blood flow, and promote inflammatory and oxidative pathways involved in AKI development (Goldfarb & Patel, 2024). These mechanisms are clinically relevant in patients with limited cardiovascular or renal reserve, especially when heat exposure is combined with poor oral intake, excessive sweating, hypotension, or intercurrent illness.

This group is also frequently exposed to medication-related vulnerability, because antihypertensive, diuretic, cardiometabolic, and nephroactive drugs may affect blood pressure regulation, renal perfusion, fluid balance, or electrolyte handling during heat stress (Centers for Disease Control and Prevention, 2025). In practical terms, patients with diabetes, hypertension, or cardiovascular disease may benefit from anticipatory education before heat waves, including advice on avoiding dehydration, limiting excessive heat exposure, recognizing hypotension or dehydration symptoms, avoiding unnecessary nephrotoxic drugs such as non-steroidal anti-inflammatory drugs, and seeking timely medical contact if symptoms or reduced urine output develop.

6.4. Medication-related vulnerability

Medication use is an important and clinically modifiable contributor to heat-related kidney risk. Diuretics may increase fluid and electrolyte losses, while ACE inhibitors and angiotensin receptor blockers may impair compensatory renal hemodynamic responses during volume depletion (Centers for Disease Control and Prevention, 2025; Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012). Non-steroidal anti-inflammatory drugs may further reduce renal perfusion by inhibiting prostaglandin-mediated afferent arteriolar vasodilation, particularly in dehydrated patients (Centers for Disease Control and Prevention, 2025; Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012). These drug-related mechanisms are especially relevant in older adults and in patients with CKD, diabetes, hypertension, or cardiovascular disease, who often use several medications affecting blood pressure, renal perfusion, or electrolyte balance.

Sodium-glucose cotransporter 2 inhibitors should not be framed as harmful in themselves, because they provide important cardiometabolic and nephroprotective benefits in appropriate patients. However, their osmotic diuretic effect makes patient education about dehydration, acute illness, and clinician-guided temporary medication management particularly important (Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012). Medication-related vulnerability should therefore be framed as a reason for individualized counselling and "sick day" education, not as a reason to avoid evidence-based cardiometabolic or nephroprotective therapy.

6.5. Outdoor and manual workers

Outdoor and manual workers represent a key vulnerable group from both clinical and public health perspectives. Agricultural, construction, landscaping, and other physically demanding occupations combine environmental heat, metabolic heat production, sweating, limited rest breaks, and sometimes restricted access to water or shade. Outdoor workers are consistently identified among populations at increased risk of heat-related kidney injury, particularly when heat exposure is combined with dehydration and physically demanding work (Goldfarb & Patel, 2024; Nerbass et al., 2017). This risk is not determined by ambient temperature alone, but also by work organization, access to water and shaded rest areas, acclimatization, socioeconomic vulnerability, and the degree of worker control over pace and breaks.

Field studies support the clinical relevance of this exposure. Among Florida agricultural workers, approximately 53% were dehydrated before the work shift and 81% after the shift, while 33% experienced AKI on at least one workday; the odds of AKI increased by 37% for each 5°F increase in heat index (Mix et al., 2018). In California agricultural workers, heavier workload and piece-rate work were associated with increased odds of AKI, suggesting that work organization and payment systems may influence renal risk, not only ambient temperature (Moyce et al., 2020). In a pilot study among Florida construction workers, dehydration was common and AKI was observed in 38% of participants after a work shift (Chicas et al., 2025). These findings support the need for occupational strategies such as water, rest, shade, acclimatization, workload modification, and protection of workers who may have limited control over working conditions. An integrative review of U.S. agricultural workers similarly reported that AKI incidence after a single work shift reached up to 43%, and that occupational heat stress and piece-rate compensation were associated with increased odds of AKI, further supporting the importance of work-organization factors in kidney risk (Zhao et al., 2025).

6.6. Athletes and physically active individuals

Athletes and physically active individuals may develop heat-related renal stress when prolonged or intense exertion is performed in hot and humid conditions. Exercise increases metabolic heat production and sweating, and when fluid losses are not adequately replaced, the combined effects of heat stress and dehydration may reduce renal perfusion and increase tubular stress (Chapman, Johnson, et al., 2021). This risk is not limited to professional athletes and may also occur during endurance events, military training, outdoor recreational activity, and unaccustomed high-intensity exercise in hot weather.

In most healthy individuals, transient renal stress after exercise is likely to resolve with rest, cooling, and rehydration. However, severe exertional heat illness may be complicated by electrolyte disturbances, marked dehydration, and muscle injury. In this setting, rhabdomyolysis can further increase AKI risk through myoglobin-related tubular injury, particularly when symptoms such as collapse, confusion, dark urine, severe muscle pain, or reduced urine output occur (Chapman, Johnson, et al., 2021). Therefore, the clinical relevance of this group lies mainly in early recognition of exertional heat illness and identification of individuals who require medical assessment rather than simple self-care.

Preventive strategies should include heat acclimatization, avoidance of peak heat, planned hydration, rest and cooling breaks, and adjustment of exercise intensity during heat waves (Masoud et al., 2024). These recommendations are especially important for endurance athletes, military trainees, outdoor workers who exercise outside work, and people performing unaccustomed strenuous activity in hot environments. This group illustrates that heat-related kidney risk may arise not only from chronic occupational exposure, but also from episodic high-intensity exertion when environmental heat, dehydration, and delayed recognition of symptoms overlap.

The main vulnerable groups and practical preventive priorities are summarized in Table 1.

Table 1. Groups at increased risk of heat-related kidney injury and practical preventive focus.

Vulnerable group	Main risk factors	Practical preventive focus
Older adults	Reduced thirst perception, impaired thermoregulation, comorbidities, and medication use may increase susceptibility to dehydration and AKI during heat exposure (Chapman, Johnson, et al., 2021; McTavish et al., 2018).	Heat alerts, hydration support, check-ins, access to cooling, and early assessment after significant heat illness.
CKD patients	Reduced renal reserve may limit the ability to compensate for dehydration, electrolyte disturbances, and reduced renal perfusion (Barraclough, 2026; Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2024).	Individualized fluid advice, avoidance of dehydration, and renal function assessment after significant heat illness.
Diabetes, hypertension, cardiovascular disease	Hemodynamic vulnerability and frequent use of medications affecting fluid balance or renal perfusion may increase heat-related renal risk (Barraclough, 2026; Centers for Disease Control and Prevention, 2025; World Health Organization, 2026).	Medication review, anticipatory education before heat waves, and early recognition of hypotension or dehydration.
Medication-related risk	Diuretics, ACE inhibitors/ARBs, NSAIDs, and SGLT2 inhibitors may become clinically relevant during dehydration or acute illness (Centers for Disease Control and Prevention, 2025; Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012).	Sick-day education, avoidance of NSAIDs during dehydration, and individualized counselling rather than routine drug interruption.
Outdoor/manual workers	Environmental heat, metabolic heat production, sweating, limited rest/water/shade, and piece-rate work may increase AKI risk (Chicas et al., 2025; Mix et al., 2018; Moyce et al., 2020).	Water-rest-shade, acclimatization, workload modification, scheduled breaks, and occupational heat protection (Flouris et al., 2024; Masoud et al., 2024).
Athletes/physically active individuals	Exertional heat stress increases sweating, dehydration risk, and in severe cases may contribute to rhabdomyolysis-related AKI (Chapman, Johnson, et al., 2021; Masoud et al., 2024).	Heat acclimatization, hydration, avoiding peak heat, and early evaluation of collapse, dark urine, or severe muscle symptoms.

Note: AKI, acute kidney injury; CKD, chronic kidney disease; CKDnt, chronic kidney disease of non-traditional origin; NSAIDs, non-steroidal anti-inflammatory drugs.

7. Public Health Strategies and Preventive Measures

Heat-related kidney injury should be approached as a partially preventable clinical and public health problem. The main preventive target is the interruption of the pathway from heat exposure to dehydration, reduced renal perfusion, tubular stress, and acute kidney injury. However, prevention should not rely only on individual behaviour, because exposure and adaptive capacity are strongly influenced by age, comorbidities, medication use, working conditions, housing, access to cooling, and socioeconomic resources (Barraclough, 2026; Nerbass et al., 2017). Therefore, effective prevention requires coordinated strategies at the individual, clinical, occupational, community, and policy levels.

7.1. Individual-level strategies

At the individual level, preventive measures should focus on reducing heat strain and avoiding dehydration before kidney injury develops. Practical strategies include avoiding prolonged outdoor activity during peak heat, maintaining adequate fluid intake, using shaded or cooled environments, reducing exertion during heat waves, and recognizing early symptoms such as dizziness, weakness, nausea, confusion, reduced urine output, or heat exhaustion (World Health Organization, 2026). Individuals should also be advised to avoid unnecessary nephrotoxic exposures, particularly non-steroidal anti-inflammatory drugs, during dehydration or acute heat illness (Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012). These recommendations should be framed as risk-reduction measures rather than universal rules, because fluid needs and medication-related risks differ between healthy adults, older adults, patients with chronic kidney disease, and patients with cardiovascular disease.

7.2. Clinical strategies

Clinical strategies should focus on identifying patients who are most likely to develop heat-related renal complications before periods of high temperature. Older adults, patients with chronic kidney disease, diabetes, hypertension, cardiovascular disease, and patients using diuretics, renin–angiotensin system inhibitors, non-steroidal anti-inflammatory drugs, or other medications affecting fluid balance may benefit from anticipatory counselling before heat waves (Centers for Disease Control and Prevention, 2025; World Health Organization, 2026). Medication-related advice should be individualized rather than automatic, because potential changes in dose, timing, or temporary interruption during acute illness depend on the drug class, clinical indication, comorbidities, fluid status, and risk–benefit assessment (Centers for Disease Control and Prevention, 2025).

In selected high-risk patients, clinicians may consider reviewing medication plans, discussing clinician-guided sick-day management during acute illness, and advising earlier medical contact when symptoms of dehydration, hypotension, reduced urine output, confusion, or persistent weakness occur. After significant heat illness, especially in patients with CKD or cardiovascular disease, assessment of serum creatinine, estimated glomerular filtration rate, electrolytes, and creatine kinase when rhabdomyolysis is suspected may help detect AKI or related complications (Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012; Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2024). This clinical approach should not be interpreted as routine laboratory testing for all heat-exposed individuals, but as targeted risk management for patients with reduced renal or cardiovascular reserve.

7.3. Occupational strategies

Occupational prevention is particularly important because outdoor and manual workers may have limited control over heat exposure, workload, rest breaks, hydration, and access to shade or cooling. Reviews of occupational heat exposure emphasize the need for prevention and mitigation strategies that address both environmental heat and work organization (Nerbass et al., 2017). Field studies among agricultural and construction workers show that dehydration and AKI may occur during a single work shift, supporting the need for workplace-level interventions rather than relying only on worker self-management (Chicas et al., 2025; Mix et al., 2018; Moyce et al., 2020).

Strategies aimed specifically at reducing AKI risk during physical work in the heat include maintaining hydration, reducing heat strain, supporting heat acclimatization, adjusting workload, and providing structured opportunities for rest and cooling (Masoud et al., 2024). Broader occupational guidance and regulatory documents similarly emphasize reliable access to cool drinking water, scheduled rest breaks, shaded or cooled recovery areas, monitoring for heat illness symptoms, supervisor training, and workplace heat-prevention plans (Flouris et al., 2024; Occupational Safety and Health Administration, Department of Labor, 2024). These measures are clinically relevant because reducing heat strain and dehydration may reduce the immediate risk of AKI and potentially limit repeated episodes of renal stress.

7.4. Community and policy strategies

Community and policy-level strategies are needed because individual advice may be insufficient when people have limited access to cooling, safe drinking water, transport, healthcare, or control over living and working conditions. Heat-health warning systems, public communication campaigns, cooling centers or other accessible cooled public spaces, outreach to older adults living alone, and coordination between healthcare services, occupational health, social care, and local authorities may reduce heat-related morbidity in vulnerable groups (Ebi et al., 2021; World Health Organization, 2026). These interventions are particularly relevant for older adults, patients with chronic diseases, outdoor workers, socially isolated individuals, and people living in housing without adequate cooling.

From a kidney health perspective, community-level interventions shift prevention upstream. Instead of detecting AKI only after heat illness occurs, they aim to reduce heat exposure, dehydration, and delayed recognition of symptoms before renal injury develops. Urban and environmental planning measures, including shade, green spaces, reduction of urban heat islands, cooler public spaces, and access to safe drinking water, may reduce population-level heat strain and therefore indirectly support kidney protection (Ebi et al., 2021; World Health Organization, 2026).

Public health planning should also include occupational exposure, because many heat-related risks occur in people who cannot freely avoid heat during working hours. The International Labour Organization emphasizes that workers should be included in heat action plans, early warning systems, and broader heat-related public health efforts (Flouris et al., 2024). This is important for kidney health because occupational heat exposure may combine environmental heat, physical workload, dehydration, limited rest, and restricted access to shade or cooling.

7.5. Equity considerations

Equity should be considered a central component of heat-related kidney injury prevention. People at highest risk may also have the least ability to modify exposure, including outdoor workers, migrant or informal workers, older adults living alone, people without air conditioning, and patients with limited access to healthcare. Occupational and community prevention strategies should therefore prioritize groups with lower adaptive capacity and limited control over working or living conditions (Barraclough, 2026; Nerbass et al., 2017).

This approach supports a broader public health interpretation of kidney protection: preventing heat-related renal injury requires not only clinical advice, but also safer work environments, accessible cooling, health education, and policies that reduce unequal exposure to hazardous heat.

Overall, prevention of heat-related kidney injury should be understood as a multilevel strategy rather than a single behavioural recommendation. Individual hydration and symptom recognition are important, but they are insufficient without clinical risk identification, medication counselling, occupational heat protection, community heat-response systems, and equity-oriented policies. This multilevel approach is particularly relevant because the populations most vulnerable to heat-related renal injury often have the least control over heat exposure, rest, cooling, and access to healthcare (Barraclough, 2026; Nerbass et al., 2017).

8. Clinical Implications

Heat-related kidney injury has practical clinical implications because early symptoms of dehydration, heat illness, and AKI may be non-specific. Patients after significant heat exposure may present with weakness, dizziness, nausea, confusion, reduced urine output, orthostatic symptoms, or worsening fatigue rather than with clearly “renal” complaints. This is particularly relevant in older adults and patients with chronic kidney disease, diabetes, hypertension, cardiovascular disease, or polypharmacy, in whom renal reserve and compensatory mechanisms may be limited (Chapman, Johnson, et al., 2021; McTavish et al., 2018).

Clinicians should also consider the timing of symptoms in relation to environmental exposure. A recent heat wave, prolonged outdoor work, endurance activity, limited access to fluids, or residence in a poorly cooled environment may provide an important diagnostic clue when evaluating otherwise non-specific deterioration in renal function. This exposure history is especially useful because heat-related AKI may overlap clinically with other common causes of acute kidney dysfunction, including infection, gastrointestinal fluid loss, medication-related hypoperfusion, and exacerbation of pre-existing CKD.

Medication history is clinically important in this context because diuretics, renin–angiotensin system inhibitors, non-steroidal anti-inflammatory drugs, and other drugs affecting fluid balance, renal perfusion, or thermoregulation may increase vulnerability during dehydration or acute illness (Centers for Disease Control and Prevention, 2025; Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work

Group, 2012). This does not imply routine discontinuation of evidence-based therapy, but supports individualized counselling and clinician-guided medication review in high-risk patients.

After significant heat illness, especially in older adults, patients with CKD, cardiovascular disease, or symptoms such as reduced urine output, persistent weakness, confusion, hypotension, or severe muscle pain, clinicians may consider laboratory assessment for kidney-related complications. Serum creatinine, estimated glomerular filtration rate, and electrolytes may help detect AKI or fluid-electrolyte disturbances, while creatine kinase should be considered when exertional heat illness or rhabdomyolysis is suspected (Chapman, Johnson, et al., 2021; Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012). This targeted approach is more appropriate than routine testing of all heat-exposed individuals and is consistent with risk-based prevention.

A practical clinical sequence may be summarized as follows: heat exposure plus dehydration or heat illness symptoms should prompt assessment of individual risk factors, medication use, hydration status, urine output, and signs of exertional injury. In high-risk patients, assessment of renal function and electrolytes should be considered, heat illness should be managed promptly with cooling and rehydration when appropriate, and follow-up should be arranged if AKI, persistent symptoms, or ongoing vulnerability is present. This approach reframes heat-related AKI as a potentially preventable or earlier-recognized complication rather than an unavoidable consequence of extreme weather.

9. Limitations of Current Evidence

Current evidence on heat exposure and kidney health has several limitations. Much of the available literature is observational, and studies differ in how they define heat exposure, heat waves, acute kidney injury, chronic kidney disease, hydration status, and occupational heat stress (Lee et al., 2019). This heterogeneity limits direct comparison between studies and makes it difficult to estimate the precise magnitude of heat-related renal risk across populations and settings.

Causal interpretation is also challenging because heat exposure rarely occurs in isolation. The renal effects of high temperature may be modified by physical exertion, hydration practices, medication use, comorbidities, socioeconomic conditions, access to water and cooling, type of work, and local occupational protections. This is particularly important in the context of chronic kidney disease of non-traditional origin, where heat stress and dehydration are plausible contributors, but the condition is likely multifactorial and may also involve workload, environmental exposures, labour conditions, and social vulnerability (Barraclough, 2026; Nerbass et al., 2017).

Evidence on prevention is clinically promising but still incomplete. Strategies such as hydration, rest breaks, acclimatization, cooling, workload modification, and heat-health warning systems are biologically plausible and supported by occupational and public health guidance, but more prospective and interventional studies are needed to determine which measures most effectively reduce heat-related AKI and long-term kidney risk in specific populations (Flouris et al., 2024; Masoud et al., 2024). Therefore, the conclusions of this review should be interpreted as a clinically oriented synthesis of current evidence rather than as evidence for a single causal pathway or a definitive prevention protocol. Because this was a narrative review, the selection of evidence was not based on a predefined systematic search protocol, and relevant studies may have been missed.

10. Conclusions

Heat exposure is an increasingly relevant environmental and occupational health factor that may affect kidney health through dehydration, reduced effective circulating volume, renal hypoperfusion, tubular stress, and heat-related acute kidney injury. Current evidence supports an association between high ambient temperature and kidney-related morbidity, while large population-based data indicate that heat-related AKI is relevant not only in tropical or occupational hotspots, but also in temperate healthcare systems (Hajat et al., 2024; Lee et al., 2019).

Repeated heat stress and dehydration may contribute to CKD risk in susceptible and occupationally exposed populations, particularly when combined with physical workload, limited recovery, inadequate hydration, and adverse working or socioeconomic conditions. However, causality and the relative contribution of heat compared with other environmental, occupational, and social determinants remain under investigation, especially in chronic kidney disease of non-traditional origin (Barraclough, 2026; Nerbass et al., 2017).

Preventive strategies should therefore move beyond simple advice to increase fluid intake. A kidney-protective approach requires clinical risk identification, medication awareness, early recognition of

dehydration and reduced urine output, occupational heat protection, access to cooling and safe drinking water, and community heat-response planning. This multilevel perspective may help reduce heat-related kidney harm in a warming climate, particularly among older adults, patients with chronic diseases, outdoor and manual workers, and populations with limited control over heat exposure.

Ethics approval: Not required. This narrative review is based exclusively on previously published literature and does not involve direct recruitment of human participants, patient-level data collection, or animal experimentation.

Informed consent: Not applicable. No primary patient data or individual clinical cases were collected for the purpose of this review.

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