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Ten tips on how to care for your CKD patients in episodes of extreme heat

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ABSTRACT

Climate change is responsible for ≈75% of extreme heat events throughout the world. Heat events are associated with an increased risk for acute kidney injury, which contributes to the development of chronic kidney disease (CKD) and cardiovascular events. Patients with CKD are especially vulnerable to heat stress for a variety of reasons. A disproportionate percentage of patients with CKD live in poverty; experience homelessness, mental illness or disabilities; work outside or are elderly, all demographics that overlap with populations most susceptible to episodes of extreme heat. Therefore, it is reasonable to conclude that exposure to episodes of extreme heat can lead to the progression of CKD and increases morbidity and mortality. Given these concerns, clinicians must be prepared to promptly recognize complications of heat in CKD patients and to help patients appropriately acclimate. We propose the following tips for clinicians to effectively care for their CKD patients during extreme heat days.

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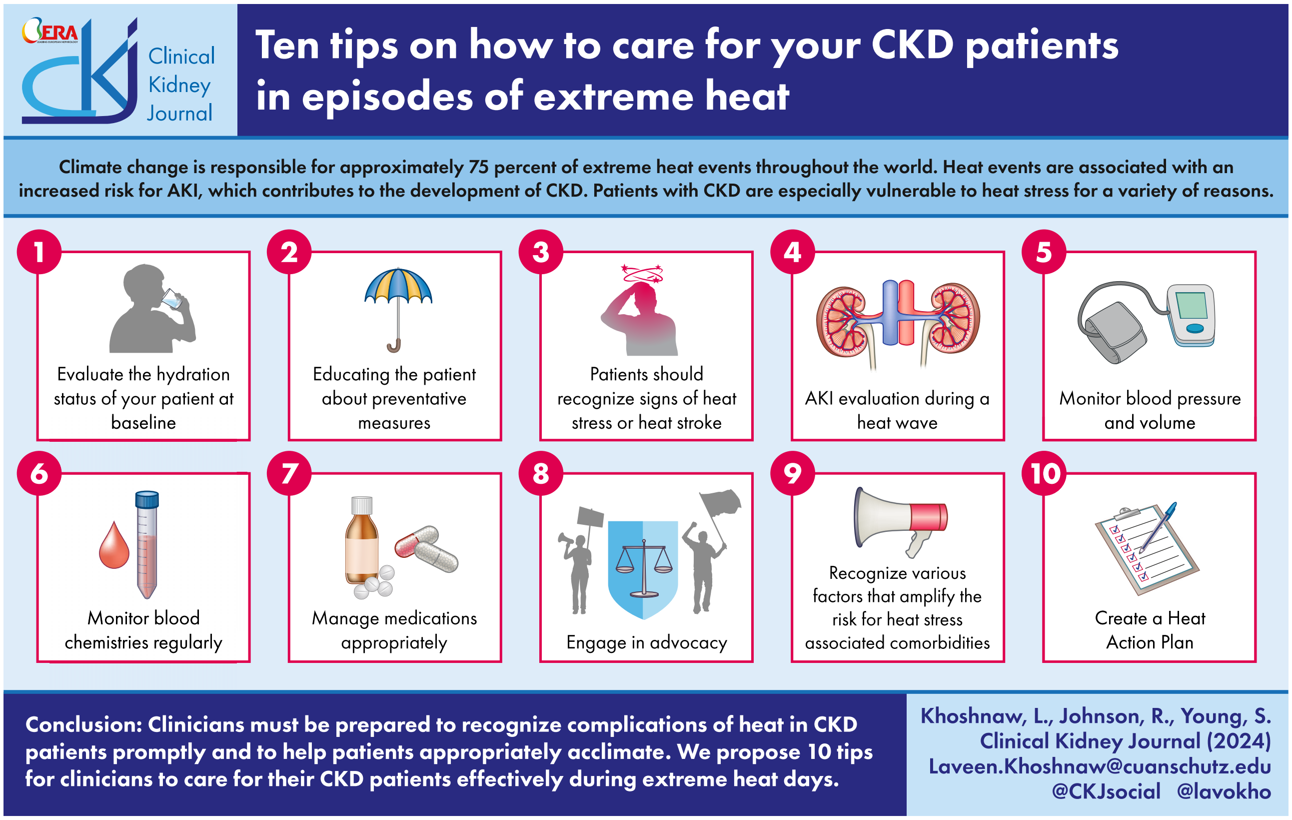
GRAPHICAL ABSTRACT



Keywords: AKI, CKD, climate change, extreme heat, hydration

INTRODUCTION

Climate change is responsible for ≈75% of extreme heat events throughout the world. The Centers for Disease Control defines extreme heat events as 2–3 days of weather that is much hotter than average for a particular time and place. These heat events are associated with increased risk for heat stroke, hospitalization and mortality [1]. Heat events are also associated with an increased risk for acute kidney injury (AKI) [2, 3], which contributes to the development of chronic kidney disease (CKD) and cardiovascular events [4, 5]. Indeed, recurrent heat stress is considered a contributing factor in CKD of unknown aetiology that is occurring in rural agricultural communities in Central America, Mexico and southern Asia [6, 7].

Patients with CKD [defined as kidney damage or a decrease in glomerular filtration rate (GFR) <60 ml/min/1.73 m^2 persisting for at least 3 months) are especially vulnerable to heat stress for a variety of reasons (Table 1). First, a disproportionate percentage of patients with CKD live in poverty, experience homelessness, have mental illness or disabilities, work outside or are older [6, 8, 9], all demographics that overlap with populations most susceptible to episodes of extreme heat [10]. Many patients with CKD live in the inner portions of major cities in which temperatures are high (due to the 'heat island' effect) and air conditioning may not be available. Structures such as buildings and asphalt roads absorb heat from the sun and reflect it more than natural formations like forests or bodies of water [11].

Patients with CKD often have lost their ability to concentrate their urine and are therefore more likely to be at risk for dehydration. Patients with CKD are also often on diuretics or may be diabetic, which further increases their risk for dehydration and AKI [6, 8, 9], and when episodes of AKI do occur, the risk for progression of their CKD to end-stage renal disease (ESRD) is increased [12]. When exposed to heat stress, AKI may occur via multiple mechanisms (Fig. 1).

Given these concerns, clinicians must be prepared to promptly recognize complications of heat in CKD patients and to help patients appropriately acclimate. We propose the following tips for clinicians to effectively care for their CKD patients during extreme heat days.

CLINICAL TIPS FOR THE MANAGEMENT OF PATIENTS WITH CKD AT RISK FOR HEAT STRESS

Evaluate the hydration status of your patient at baseline

The Institute of Medicine has suggested that adequate water intake in the healthy adult should be ≈2.6 l for men and 1.8 l for women [13]. Hydration status of a CKD patient should be assessed regularly during CKD follow-up so patients know what their standard water consumption should be and they are counselled on how to adjust it in an episode of extreme heat. As

Table 1: Common comorbidities in patients with CKD that may enhance the risk for AKI.

Comorbidity	Effect to consider
Chronic diarrhoea	Excessive GI losses lead to increased dehydration
Kidney stones	May need increased hydration
Psychiatric disorders	Heat episodes may exacerbate psychiatric conditions
Mobility disorders	Patients without support may be unable to access water or a cooling centre
Cognitive disorders	Patients may forget or be unsure of how to access water
Social isolation	Patients without support may neglect to prepare for episodes of extreme heat
Experiencing homelessness	Patients may not have the resources to avoid heat exposure
Loss of urinary concentration	Increased risk for AKI associated with volume depletion
Diabetes	Increased risk for AKI associated with volume depletion
Use of diuretics and antihypertensive agents	Increased risk for volume depletion or hypotension. Blockade of the RAS may increase the risk for AKI in subjects who are dehydrated or volume depleted

mentioned, patients with CKD may not concentrate or dilute their urine as effectively and hence may be at risk for both hyponatraemia or hypernatraemia. Hypernatraemia is a risk factor for CKD, hypertension and an array of metabolic conditions [14–17]. Recent studies suggest that a serum sodium in the upper limits of the normal range confers metabolic risk, a condition referred to as underhydration [16, 17]. It is hypothesized that underhydration leads to chronic stimulation of vasopressin [18].

One way to assess hydration status is to look for signs of underhydration, in which patients tend to show elevations in urine osmolality (>500 mOsm/l), mild hypernatraemia or elevations in plasma copeptin (a biomarker for vasopressin) [18, 19]. For the CKD patient, serum sodium may be a better measurement than urine osmolality since the latter may be affected by medications [sodium–glucose co-transporter-2 (SGLT2) inhibitors, diuretics and lithium] or impaired urinary concentration from diabetes or CKD. An ideal serum sodium is ≈138–142 mmol/l [17]. Higher levels are associated with progression of kidney disease and worse outcomes [20].

In patients with CKD, the relation between water intake and the progression of kidney failure is U-shaped; both high and low intake may be consequential in CKD. Patients consuming <0.5 l or >2.0 l were associated with a higher risk of kidney failure, while those who maintained 1.0–1.5 l were unaffected [21]. During heatwaves, water intake must be balanced with the rate of water loss as the body attempts to cool, so patients should feel comfortable increasing their water intake above their baseline prescription. These changes may significantly differ from one CKD patient to another. For instance, a patient with CKD and heart failure who struggles with hyponatraemia may only be able to increase their fluid intake minimally without becoming hyponatraemic. In contrast, a patient with CKD and nephrogenic diabetes insipidus may need to increase their intake by several litres during an episode of extreme heat to prevent hypernatraemia.

A major pitfall is the risk of hyponatraemia in patients who overwhelm their water-excreting capacity, which is limited due to their decreased GFR [22]. Patients must be cautioned about the potential to overwhelm the kidney's ability to excrete water in CKD. Patients should weigh daily, and daily weights should remain stable. Counsel patients to monitor their blood pressure (BP) and heart rate, recognize signs of dehydration, including

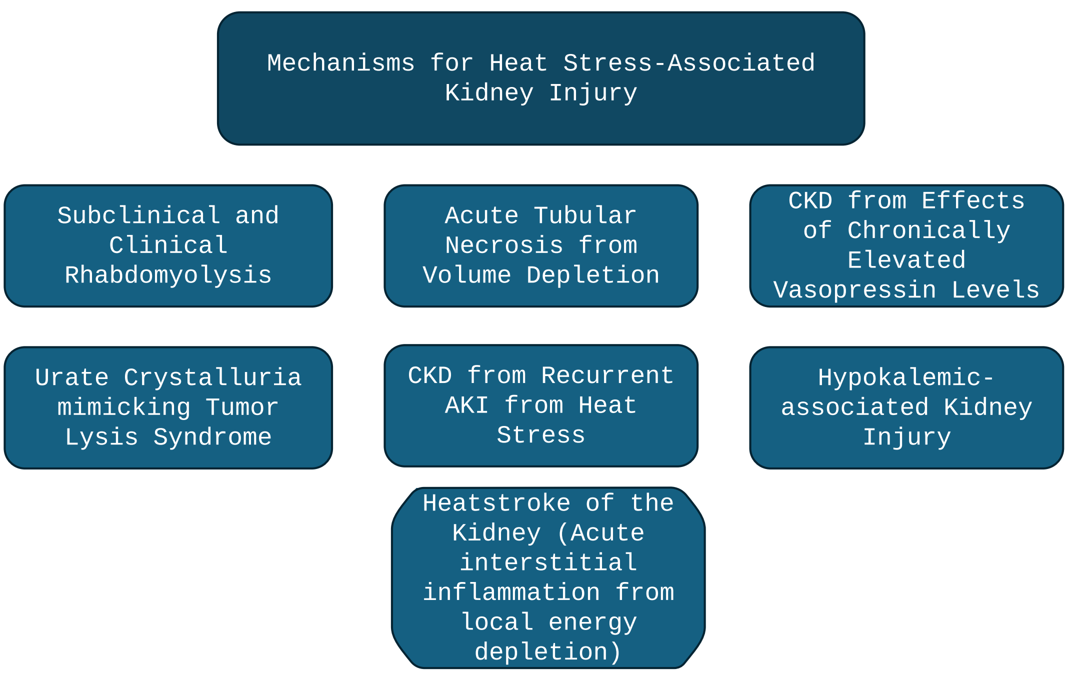


Figure 1: Mechanisms for heat stress–associated kidney injury.

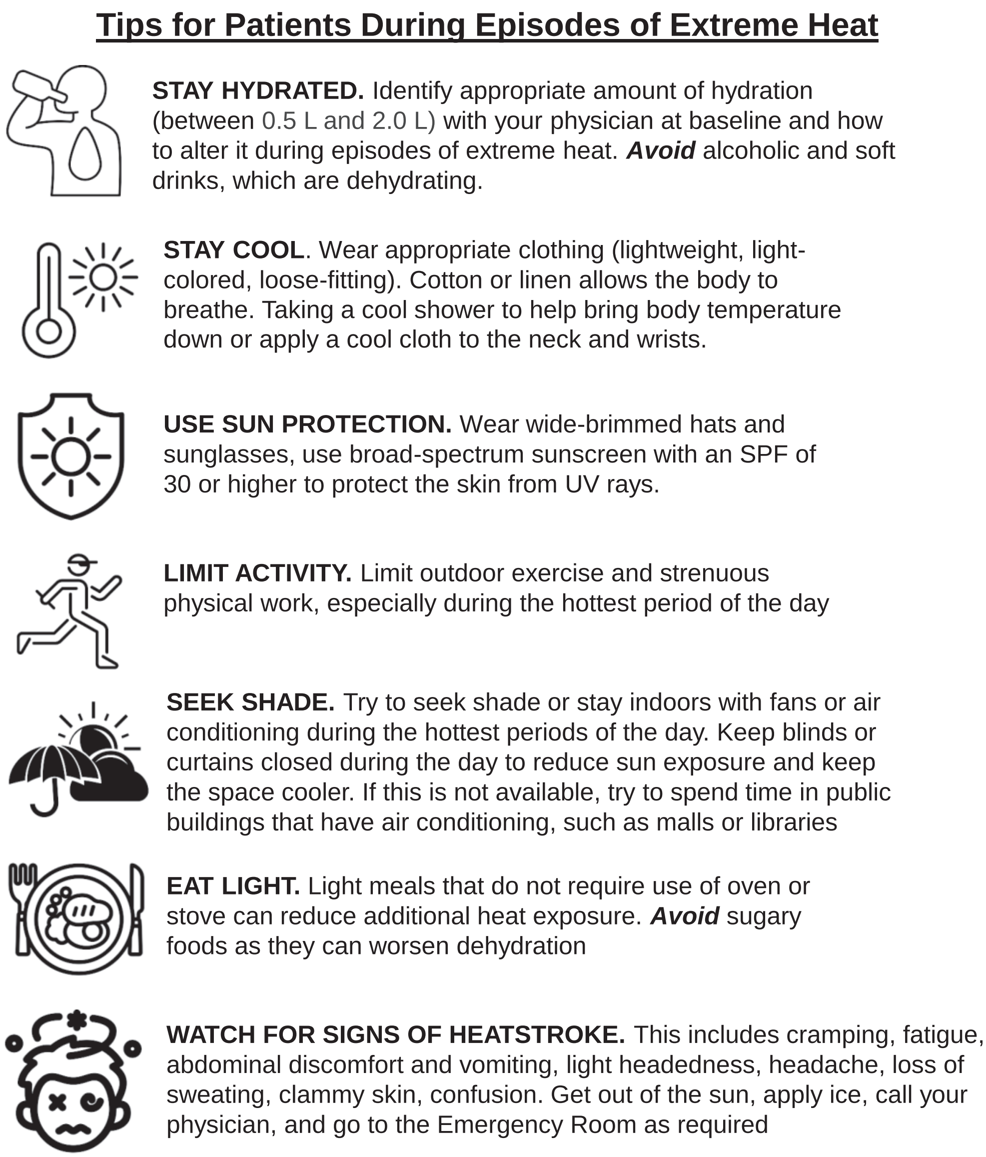


Figure 2: Tips for patients during episodes of extreme heat. Graphics modified from Noun Project Creators (Amethyst Studio, Los Angeles, CA, AU, metami spetiana, Dimiter Petrov, Lukasz Aflo and Soni Sokell).

lightheadedness, fatigue, thirst, muscle cramps and orthostasis, and contact their physicians if in doubt.

Educate the patient about preventive measures

A major goal is to find ways to reduce the risk of overheating. Animal studies show that kidney injury in heat stress is markedly worsened with increasing core body temperature. Raising core body temperature only 1°C was sufficient to induce significant kidney damage [23]. A body temperature >37°C suggests inadequate temperature correction, while a body temperature >39°C is serious and may herald heat stroke [24]. Patients with CKD at risk for elevations in core body temperature from other comorbidities (such as hyperthyroidism) need to be monitored particularly closely.

Education of the patient is key, and basic principles are shown in Fig. 2. Having adequate ways to reduce heat exposure is critical. Investigations have highlighted that air conditioning reduces hospital admissions for AKI [25]. Patients with CKD should minimize their time outside in direct sunlight and heat and seek spaces with central air conditioning to minimize the demand for thermoregulation. Practical ways to ensure that patients stay cool include wearing a hat, seeking shade and staying hydrated to replenish mechanisms of thermoregulation (sweating) [11]. Certain labour, such as agricultural work and mining, poses the risk for occupational heat exposure and should be avoided in patients with CKD [26].

Clinicians should work with their patients to create a Heat Action Plan (HAP) appropriate to their circumstances, which may include cooling centres and avoiding direct outdoor daytime exposure (see Tip 10).

Patients should recognize signs of heat stress and heat stroke

Patients should be able to identify signs of heat stroke, including fever (≥39.4°C), headache, dizziness and nausea (Fig. 2). Altered mental status or decreased sweat production should prompt patients to seek immediate medical attention. Early recognition in a hospital setting (via core body temperature) and rapid cooling should be implemented with the goal of reducing the risk of renal complications. Cold water immersion is recommended as a first-line intervention [24]. Objective clinical data in the hospital setting includes assessment of kidney function tests, creatine phosphokinase, high-sensitivity C-reactive protein and liver function tests.

AKI evaluation during a heatwave

AKI assessment should be conducted as usual by evaluating prerenal, renal, and postrenal causes, with a particular emphasis on volume status. Extreme heat leads to greater loss of both water and salt, which can contribute to prerenal AKI and electrolyte abnormalities. Creatinine and urine output should be monitored in patients with AKI, in accordance with Kidney Disease: Improving Global Outcomes guidelines [27]. Even among well-hydrated patients, there is an increased risk of AKI with heat stress [28].

While rhabdomyolysis and myoglobinuric acute renal failure are typical manifestations in exertional heat stroke (EHS), they are also increasingly seen in classic heat stroke (CHS) [29]. CHS results from exposure to high ambient temperatures often coupled with high humidity and occurs in epidemic form during heatwaves. It presents overwhelmingly more in older individuals (due to disrupted thermoregulation) and individuals with pre-existing conditions such as hypertension, diabetes, cardiovascular disease and respiratory disease [30]. EHS typically occurs in younger individuals engaged in vigorous physical activity in hot or temperate environments (such as athletes, military personnel and agricultural workers) [31]. Heat stress–associated labour can result in subclinical or clinical rhabdomyolysis from low-grade muscle trauma and heat [32]. Providers should obtain a urinalysis and assess plasma myoglobin, creatine phosphokinase and aldolase levels [33]. Metabolic (lactic) acidosis is usually seen in exertional forms of heat stroke. Additionally, extremely strenuous physical activity increases systemic inflammation and oxidative stress. Inflammatory cells and inflammation are currently considered important mechanisms for AKI development [34]. CKD patients should be advised not to partake in any exercise during episodes of extreme heat because of the risk of rhabdomyolysis.

Monitor BP and volume

Patients with CKD should monitor their BP with a goal of <140/80 mmHg, and ideally <130/80 mmHg [35]. Generally, BP is lower in the summer (to keep cool and release more heat from the body into the environment) and higher in the winter (to push blood through constricted vessels). During heatwaves, we recommend patients monitor their BP once a day, with additional readings if they are not feeling well. Mild heat stress increases BP through activation of the renin–angiotensin–aldosterone system (RAS) and sympathetic nervous system, but when more severe, BP decreases [24]. Lower BP is a marker of greater mortality in older individuals with advanced CKD [36]. In patients with CKD during episodes of extreme heat, evaluation of BP is critical. Patients may report lightheadedness, weakness, visual changes, diaphoresis or pallor upon positional change. One of the most useful clinical tools is the assessment of orthostatic vitals. Clinicians should use baseline at-home measurements to adjust BP medications and fluid intake accordingly.

Monitor blood chemistries regularly

Evaluation of blood chemistries can give critical insights into the status of the CKD patient, especially in episodes of extreme heat. Patients should have a standing order to obtain serum chemistries if they are feeling unwell during an episode of extreme heat. Creatinine and GFR should be trended from the initial CKD diagnosis and used for comparison to ascertain possible heat stress.

Special attention should be paid to electrolytes. AKI and electrolyte disturbances can still occur in patients who are adequately hydrated [28]. Volume depletion can be associated with either hypokalaemia or hyperkalaemia in CKD. Hypokalaemia may reflect sweating or activation of the RAS and can cause intrarenal vasoconstriction and hypoxia, resulting in chronic tubulointerstitial injury and subsequent exacerbation of CKD [37]. Hyperkalaemia can result in life-threatening cardiac arrhythmias, muscle weakness and paralysis. Hypophosphataemia suggests adenosine triphosphate consumption and risk for rhabdomyolysis [38]. Patients may lose salt in addition to water. This is exacerbated by sweating, diarrhoea and medications. We recommend patients stop their SGLT2 inhibitors and reduce diuretics during heatwaves to avoid volume depletion.

In addition, hyperuricaemia may develop from heat stress, in part because of subclinical or clinical rhabdomyolysis, while urinary acidification may also occur due to lactate production and from the effects of aldosterone acidifying the urine. As a consequence, hyperuricosuria with urate crystal formation can occur, which may cause AKI similar to tumour lysis syndrome. In studies of sugarcane workers at risk for AKI from heat stress, there was an occasional marked increase in uricosuria during the workday [39–41]. This may increase the risk for kidney stones and play a role in the progression of CKD [42, 43].

Re-evaluate patients' prescriptions during heatwaves

Most patients with CKD have comorbidities, including diabetes, hypertension and heart failure. Because of their direct effect on the kidney and electrolytes, medications used for the management of these diseases, such as antihypertensives, SGLT2 inhibitors and diuretics, should be re-evaluated in episodes of extreme heat.

Other therapeutic drugs can be risk factors for heat-related illness and subsequent renal involvement due to their ability to inhibit thermoregulation in various ways (e.g. altered sweat production, dehydration, increased heat production, impaired thirst recognition or inhibited heat loss). In episodes of extreme heat, consider adjusting medications including psychotropics (such as neuroleptics, anxiolytics, antidepressants, barbiturates and anticholinergics) and antihistamines [44]. An important caveat when adjusting psychiatric medications is that acute psychiatric illness is exacerbated in episodes of extreme heat [45, 46]. Collaboration with mental health providers is critical. Polypharmacy in older adults is also a risk factor for heat-related illness due to disruption in thermoregulation [10]. Patients should always contact their physician prior to adjusting medications.

Engage in advocacy

Clinicians are often respected in communities, and this can provide opportunities for advocating for their patients both at an individual and community level. Generating awareness of the health issues related to global warming can aid in the education of the public and the importance and need for prioritization at

Figure 3: Heatwave action plan.

the social and political level. Public awareness regarding the vulnerability of CKD patients in such circumstances remains limited, underscoring the critical role clinicians play. Sharing their expertise with governmental bodies, public health officials, major dialysis organizations and other community stakeholders is essential. For example, clinicians can work with public health officials to ensure heatwave warnings are reaching vulnerable populations. Moreover, clinicians should actively engage government officials and emphasize the necessity for cooling centres, advocating for their establishment in areas with identified need.

Recognize various factors that can amplify the risk for heat stress–associated comorbidities

Patients with CKD often have several comorbidities. Clinicians must be mindful of how these comorbidities increase the risk of AKI, hospitalizations and mortality during extreme weather events. For example, in patients with chronic gastrointestinal conditions and CKD, there may be chronic losses that amplify the effects of extreme heat. Patients who are disabled or have a lack of social support may struggle in accessing water to stay hydrated or accessing a cooling centre, putting them at higher risk of morbidity and mortality during these events (see Table 1).

Create a HAP

As global temperatures continue to rise, we recommend establishing a structured plan between healthcare providers and patients to navigate periods of extreme heat effectively.

Drawing from successful initiatives such as Seizure Action Plans (SAPs) and Asthma Action Plans (AAPs), which have increased patient confidence and are expected to reduce healthcare utilization, we advocate for the implementation of a HAP [47–49] (Fig. 3). Much like SAPs and AAPs, a HAP offers personalized guidance to patients during heatwaves, aiming to mitigate the risk of AKI. Engaging patients in the collaborative development of a HAP facilitates education and yields a tangible document outlining key clinical indicators, recommended adjustments and when to seek medical assistance.

CONCLUSION

Extreme heat episodes are a growing phenomenon for which clinicians must be prepared. As global temperatures continue to trend upward, patient exposure to episodes of extreme heat will warrant dedicated care, especially in patients with CKD. Clinicians must gather data in these patients, with both objective and subjective pieces of information regarding the patient's body temperature, BP and hydration status. Laboratory measures of kidney health should be evaluated through the lens of heat stress. Finally, non-medical management, prevention and advocacy is vital to ensuring patients remain healthy through episodes of extreme heat. We call for the creation and implementation of HAPs as a method to educate patients and prepare them for these global changes.

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AUTHORS' CONTRIBUTIONS

S. Young, R. Johnson, and L. Khoshnaw conceptualized the study; R. Johnson was responsible for the software; S. Young provided supervision; L. Khoshnaw and S. Young were responsible for the visualization; L. Khoshnaw wrote the original draft; and S. Young, R. Johnson, and L. Khoshnaw reviewed and edited the manuscript.

DATA AVAILABILITY STATEMENT

The data underlying this article are available in the article itself.

CONFLICT OF INTEREST STATEMENT

None declared.

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