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Pathophysiological changes of the urinary system as a result of unintentional cold injury (scientific and literary review)

V.V. Yekhalov, orcid: 0000-0001-5373-3820, e-mail: sesualiy@gmail.com
O.V. Kravets, orcid: 0000-0003-1340-3290, e-mail: 535951@ukr.net
V.P. Stus, orcid: 0000-0002-4539-8126, e-mail: viktor.stus@gmail.com
S.I. Barannik, orcid: 0000-0002-4089-6126, e-mail: barannikc@ukr.net
M.Yu. Polion, orcid: 0000-0001-9307-1411, e-mail: polion.mykyta@gmail.com
S.L. Chekanov, orcid: 0000-0002-5025-7109, e-mail: chekanovsl@gmail.com

Dnipro State Medical University

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SUMMARY

Acute kidney injury (AKI) is observed in more than 40% of patients with cold injury, and the functional state of the kidneys during unintentional hypothermia and frostbite is a prognostic factor for survival. In thышscientific and literary review, based on information from available sources, the authors for the first time tried to reveal the dynamics of morpho-functional damage to the urinary system, depending on the degree of general random hypothermia of the body. Separately, pathophysiological changes in kidney function in the pre-reactive, post-reactive and recovery-period are considered. The concepts of the "deadly triad", "calcium paradox", "warming acidosis" and modern biomarkers for determining early acute renal failure are given. The authors hope that the collected material will be useful for practitioners in assisting victims with accidental general hypothermia.

Cold damages in the structure of injuries in a peacetime is from 1 to 10% [1, 2, 3, 4, 5, 6]. Currently, the rate of population decline in Ukraine is one of the highest in Europe and is 0.9–1.1% per year. A significant proportion of depopulation factors depend on low living standards, deteriorating health and adverse environmental factors. These include acute cold injury [7].

Emergencies with life-threatening kidney damage most often occur in acute accidental general or local hypothermia or hypothermic damage to other organs and systems [8]. Acute renal impairment

(ARI) is observed in more than 40% of patients with cold injury, and renal function in unintentional hypothermia and frostbite is a prognostic factor for survival [9, 10].

There are isolated reports of acute hypothermic functional disorders of the urinary system with hypothermia in the current available medical literature. In our review we managed to highlight the main patterns of development of GPN depending on the degree and period of cold damage of a generalized short message by Jeican I.I. (2014).

In it's development, cold trauma occurs in four periods: cold stress, pre-reactive (before warming up), reactive (after warming up) and recovery period [11, 12, 13].

Cold stress, no hypothermia (TSO *> 35.0 °C).

Note *: TSO is the central body temperature.

Cold stress is an acute or chronic psychophysiological reaction of the body caused by human exposure to climatic conditions in which the body's heat transfer is bigger or equal to the level of heat balance maintained by significant physiological stress, which can not always be compensated. Hypothermia can dull or slow down the normal hemodynamic response to stress, which, however, allows to maintain adequate renal circulation in these conditions [14, 15, 16, 17, 18, 19].

Pre-reactive period.

Modern classification of stages of general hypothermia of the Swiss Society of Mountain Medicine:

I. Mild hypothermia (adynamic phase, mild hypothermia) – the patient is anxious and trembling (TSO = 32–35 °C).

II. Moderate hypothermia (stupor phase, moderate hypothermia) – the patient is sleepy and does not tremble (TSO = 28–32 °C).

III. Severe hypothermia (convulsive-comatose phase, severe hypothermia) – a patient without consciousness, but with the presence of vital signs (TSO = 24–28 °C).

IV. Deep hypothermia (terminal phase, extremely severe hypothermia) – minimal signs of life or circulatory arrest (TSO = 24–13.7 °C).

V. Death due to irreversible hypothermia (TSO <13.7 °C) (<9 °C ?) [13, 20, 21, 22].

Mild hypothermia (TSO 32–35 °C).

Peripheral vasoconstriction leads to an increase in central blood volume, which in turn causes "cold" diuresis [23, 24]. It begins 10–20 minutes after the onset of cold and could be so obsessive that it leads to involuntary urination [25, 26]. The starting mechanism of this process is a significant release of catecholamines [11, 25], which leads to increased perfusion pressure in the renal arteries. This increases the hydrostatic gradient against which sodium is transported, which leads to a decrease in it's resorption. Cold restricts the secretion of ADH with the subsequent development of cold diuresis, which is very sensitive to antidiuretic hormone [27]. Peritubular capillary resorption capacity is a crucial factor in transepithelial sodium transport. Sodium is excreted in the urine with subsequent loss of fluid, as the ability of the distal tubules to reabsorb water is lost [24, 28, 29, 30, 31,32]. Hyponatremia due to osmolar diuresis is quite common, especially in chronic hypothermia [33]. The oxidative activity of the tubules decreases [23, 24]. If hypothermia develops rapidly, the stress response may contribute to hyperglycemia, which may also contribute an osmotic component to diuresis [29].

The influence of cold causes signs of dyshidria, which become more pronounced the lower the body temperature drops [25, 26]. This process leads to progressive hemoconcentration and a decrease in blood volume [23, 24, 27, 33]. There is an increase in the number of erythrocytes due to thickening of the blood due to dehydration, reduction of total water in the body due to several mechanisms, including cold-induced diuresis, sweating, respiratory water loss, thirst, etc. [33, 34]. The hematocrit level increases by 2% with a decrease in the central temperature for every 1p C [35, 36]. Decreased circulating blood volume exacerbates functional oligoanuria [37, 38].

Changes in the ratio of endogenous creatine in urine and it's concentration in plasma, as well as changes in chloride excretion indicate that cold diuresis is due to reduced reabsorption of water in the tubules, and increased urination at low temperatures is regulated by posterior pituitary hormone [27]. Significant changes in the concentration of urea and creatinine in the blood have not been identified [39].

Moderate hypothermia (TSO 32–28 °C).

During the cooling the exposed areas of the skin of the extremities reflexively develops spasm of the vessels of the kidneys, accompanied by albuminuria, oliguria, tension of the bladder [32]. During the experiment on laboratory animals, it was found that morphological changes in the kidneys under the influence of low temperatures are an increase in the number of mesangiocytes with pyknotic nuclei, the number of defective nephrocytes of proximal and distal tubules with pyknotic nuclei. These changes can be regarded as a progressive decrease in the functional activity of the tubular apparatus of the kidneys [40]. With moderate hypothermia, the glomerular filtration rate decreases, as does cardiac output and bleeding rate [41]. At a body temperature of 28–30 °C, heart rate, renal circulation and glomerular filtration rate are reduced by 50%. Severe hypothermia leads to the development of renal

dysfunction and insufficiency [19, 23, 24, 30, 32]. There is also a further decrease in tubular function and reduced renal glucose clearance [29, 42]. Cold proteinuria of healthy individuals after general hypothermia is caused by reflex circulatory disorders in the glomeruli [43]. Prerenal insufficiency resulting from decreased renal blood flow is considered to be the main cause of HPP caused by hypothermia, although it's pathogenesis has not been definitively studied [19]. Cells are forced to support the synthesis of adenosine triphosphate (ATP) by anaerobic glycolysis using glycogen and extracellular glucose stores. This leads to local tissue acidosis. ATP depletion seriously disrupts tubular homeostasis, increasing the load on the cytoplasm with calcium, which activates proteases, phospholipases and caspases, hypoxanthine and reactive oxygen species (ROS) accumulate intracellularly. Hypoxia, glucose depletion, acidosis and ROS promote apoptosis and active necrosis. An early structural manifestation of ischemia is the loss of cell polarity with decreased reabsorption of sodium and water from the lumen of the renal tubules. Due to the decrease in sodium reabsorption, the distal segments of the tubule are activated to provide signals that cause narrowing of afferent vessels (tubuloglomerular feedback) [44]. Urea and creatinine levels are often elevated due to the inability of chilled kidneys to excrete nitrogen [33]. Disorders of microcirculation lead to significant changes in the body and are manifested by a violation of the main function of blood circulation – metabolism between cells and blood. Endomesoperiarteritis and vascular phlebitis with endothelial hyperplasia and thickening of the vascular lining lead to obliteration of their lumen. This period of cryosurgery can be characterized as the formation of manifestations of cold shock, so this is a condition where blockage of microcirculation and impaired hemostasis lead to decreased renal perfusion [16, 17].

The progression of destructive processes is associated with increased permeability of capillary sides due to the action of vasoactive substances, primarily histamine and serotonin, released during cell damage. Histamine increases the permeability of capillaries, causes sharp swelling of their sides and narrowing of their lumen. Serotonin has the ability to damage the endothelium and provoke thrombosis. Later, vascular thrombosis progresses in the affected areas due to activation of the hemostasis system and inhibition of fibrinolysis. Significant role is played by microcirculation disorders caused not only by local exposure to cold, but also by changes in the rheological properties of blood and water-salt metabolism [10, 38]. Vascular narrowing, endothelial damage and thromboembolism contribute to the development of vascular insufficiency and ischemia. Inflammatory mediators such as prostaglandins may also be important [23]. Metabolic acidosis is caused by ketogenesis in the liver, lactate formation during tremor, peripheral circulatory disorders with subsequent tissue hypoxia and decreased cardiac output [33]. As hypothermia progresses, sodium levels tend to decrease and potassium levels increase, probably due to a decrease in the enzymatic activity of the sodium-potassium pump [32, 45]. Prerenal insufficiency can turn into renal, as circulatory disorders in the kidneys lead to ischemia and necrosis. On average renal failure begins after 2 hours of cold ischemia [37].

Severe hypothermia (TSO B 28 °C).

Morphological manifestations of the adaptive response of the structural elements of the kidneys are amplified. This manifested in the earlier and stronger development of dystrophic changes in tubular cells [39]. Proliferative-dystrophic changes of the renal tubules were detected in 12.6% of cases of cold injuries [46]. Hypothermia causes preglomerular vasoconstriction, which leads to a decrease in glomerular filtration rate and renal blood flow [31]. Renal circulation is reduced by 50%. At lower temperatures, the tubular capacity for the secretion of H + ions is reduced, which causes the renal contribution to the progression of metabolic acidosis. Anuria occurs at temperatures below 27.2 °C. Severe hypothermia leads to acute tubular necrosis and acute renal failure. Occlusion of the bladder is due to deep inhibition of the CNS during cooling and disruption of innervation, as a result of which it loses the ability to contract [29, 47, 48]. The biopsy showed ischemic kidney damage, which is thought to pass in the warming phase after a period of relative protection at lower temperatures. There is spotty and focal loss of tubular cells, epithelial necrosis, interstitial edema and restriction of arterial lumen by severe diffuse thickening of the intima. Vascular narrowing and ischemia lead to the release of endothelin, which increases more than fivefold in the early stages of the clinical course, associated with endothelial edema and the development of acute renal failure. The clinical course corresponds to acute renal failure associated with acute tubular necrosis [29, 49]. Drops in blood pressure and hypokalemia lead to a sharp decrease in glomerular filtration rate and damage to the renal tubules [29, 50]. Metabolic acidosis is a sign of severe hypothermia and may be due to the formation of lactic acid due to insufficient tissue perfusion [29, 51].

Elevated levels of cryofibrinogen can increase dramatically when exposed to cold, disrupting microcirculation and possibly leading to widespread

tissue microinfarctions [29]. Cryoglobulinemia is characterized by the appearance in the circulatory system of cold-precipitating cryoglobulins with their deposition in the walls of small vessels (capillaries, arterioles and venules) and symptoms of systemic vasculitis [52]. Elevated serum potassium in a hypothermic patient usually indicates that the hypothermia was preceded by hypoxia. Thus, it is a marker of lysis and cell death [13]. Erythrocytes and leukocytes (1–5 in n/s), epithelial cells (5–10 in n/s), granular cylinders (5–10 in n/s) are determined in the urine sediment [26]. Spontaneous urination is observed in 92.9% of victims [34].

IV. Deep hypothermia (extremely severe hypothermia) – minimal signs of life or circulatory arrest (TSO <24 °C).

General hypothermia leads to clinically significant metabolic disorders in the form of decompensated metabolic acidosis, hypercapnia, hypokalemia, cytolytic syndrome with hyperenzymemia, increased urea and blood creatinine [53]. Hyperkalemia is a marker of acidosis, lowering the pH <6.5 and the development of hyperkalemia were predictors of death in general hypothermia [30, 38, 54]. In the bladder remnants of residual urine, it loses the ability to empty urine. There is a tonic contraction of cremasters, recorded throughout the general cooling. This contributed to the preservation of the gonads from the effects of cold in the ancestors of humans who had an unoccupied vaginal sprout by pulling them into the abdominal cavity [47, 48].

V. Death due to irreversible hypothermia (TSO <13.7 °C).

There is a complete morpho-functional failure of the kidneys. Proliferative-dystrophic changes of the testicular tubules were observed in 28.8% of cases of cold death [46].

Reactive period.

"No one died until he became warm and dead" [33, 45, 55, 56, 57, 58].

"Rewarming shock" is a fatal circulatory disorder that is common in victims of accidental hypothermia caused by reheating and vasodilation in response to active warming, characterized by decreased cardiac output and a sudden drop in blood pressure through the peripheral vasodilation [26, 46]. Attempts to warm up quickly can cause shock, that is why it accelerate the increase in cardiovascular load and collapse,as peripheral vasodilation leads to adecrease in internal body temperature after a sudden return of cold blood from the extremities [13]. Because of these complications, patients may deteriorate briefly before improvement begins [59]. Secondary shock or collapse is observed during reheating, which is also associated with active external warming and peripheral vasodilation.Further cooling of the heart and return to the circulation of anaerobic metabolism products increases the risk of arrhythmias [30, 36, 45, 60]. At the same time, there is another complex of systemic disorders – trophic disorders, which are based on deep disorders of the internal organs, especially dystrophic changes in the kidneys [16, 17]. Warm therapy may cause acute tubular necrosis due to apoptosis. Granular casts and epithelial cells, which are often found in urine sediment, confirm the presence of acute tubular necrosis. ARF caused by hypothermia can be considered as a kidney injury caused by ischemia and hypovolemia [26]. Persistent oliguria, which persists after removal of the patient from shock, should be wary of the possibility of developing acute renal failure. When the later hypokalemia is replaced by hyperkalemia [50].

Ischemic-reperfusion injuries are caused by prior transient or prolonged renal hypoperfusion. Interruption of blood flow causes an imbalance between the supply and demand of O2, resulting in glucose depletion, acidosis and ROS production, which contributes to cell death. In addition, tubular epithelial and vascular endothelial cells release proinflammatory cytokines (interstitial inflammation), which are also responsible for endothelial cell edema with corresponding prolonged ischemia and delayed reperfusion (interstitial microvasculopathy). During reperfusion, pH reduction induces Ca2 + cell overload and increases the formation of free radicals, which contributes to the triggering of the inflammatory cascade and cell damage [61, 62]. Reperfusion of ischemic kidneys and heart can cause secondary damage to their vascular system with the support of similar mechanisms, causing a "double blow" to attack free radicals. There is a "calcium paradox" of reperfusion tissue damage in which sodium is exchanged for calcium, which leads to cell damage [61, 62].

"Deadly triad" of victims of cold injuries – hypothermia, coagulopathy and acidosis. The presence of hypothermia and acidosis, which lead to coagulopathy,and the relationship between these three conditions can lead to 90% mortality among victims of severe cold injuries [21, 45, 65]. "Warming acidosis" occurs when lactic acid from the periphery returns to the central bloodstream [18, 30, 36, 59, 60]. The pH paradox may arise during the revascularization phase: if reperfusion of ischemic cells occurs in the presence of acidotic pH apoptosis is inhibited. On the contrary, an increase in intracellular pH during reperfusion causes cell destruction [44]. Non-gaseous acidosis is naturally accompanied by a decrease in urine volume. This is due to a significant reduction in blood supply to the kidneys due to inhibition of hemodynamics, narrowing of renal vessels. Possible lowering of blood

pressure and drop in myocardial activity also adversely affect the blood supply to the kidneys. Renal vasospasm reduces urine production due to impaired glomerular perfusion [66]. The main causes of ischemia: hypercatecholaminemia (observed in acidosis) and hypersensitivity of adrenoceptors of peripheral arterioles. Prolonged ischemiaof tissues and organs is manifested by multiorgan dysfunction. However, as a rule, the signs of renal ischemia dominate: when the pH is lowered, the renal circulation and the volume of glomerular filtration are suppressed [17,67]. Reducing the pH of urine to 4.5 1 / -unit. almost stops glomerular filtration [37]. Histopathological examination of biopsy specimens showed the presence of tubulopathy and interstitial changes, indicating acute renal failure, which was caused by acute tubular necrosis associated with vasoconstriction [18, 26]. Rhabdomyolysis, which sometimes complicates general and local cold trauma, also contributes to the development of metabolic acidosis [68].

Vascular constriction and renal ischemia, which may occur during the hypothermic phase or after warming, cause ARF [19], usually associated with a direct consequence of hemodynamic insufficiency, hemolysis or radomiolysis [18].

Paroxysmal hemoglobinuria is characterized by the presence of blood hemolysin, which can destroy red blood cells in the presence of complement. The reaction with hypothermia begins in the pre-reactive period, although hemolysis itself occurs later. In the blood of such patients show an increase in the content of procoagulant microparticles, which are released from the membranes of lysed erythrocytes and activate platelets [69]. Under normal conditions, free plasma hemoglobin is completely bound by a type of α2-globulin – haptoglobin. It is able to bind 70–150 mg of hemoglobin per 100 ml of blood, and the haptoglobin-hemoglobin complex is not filtered by the glomeruli. With a low degree of hemoglobinemia that slightly exceeds the hemoglobin-binding capacity of haptoglobin, some of the hemoglobin may pass through the renal filter, where it is reabsorbed at the level of the renal tubules. However, if the amount of free hemoglobin exceeds the amount of haptoglobin required for its binding, or if it is in the blood plasma more than 15–25 mg per 100 ml, it is found in the urine. With hemoglobinuria, the urine becomes "lacquered" brownish-red ("meat slops"). These symptoms are followed by oliguria and anuria [5, 8, 37].

Dysfunction of the tubular apparatus in general and local cold trauma may also be associated with myoglobinuria due to rhabdomyolysis (obstruction of the tubules), which is manifested by acute tubular necrosis [63, 69, 70]. The term "myoglobinuria" distinguishes between two pathological conditions – signs of myoglobin in the urine and pathological syndrome that occurs when striated muscles are affected. The pathogenesis of myoglobinuria is in all cases associated with the release of myoglobin. The latter, due to it's low molecular weight (three times less than hemoglobin), passes quickly through the glomerular filter. In an acidic environment, myoglobin precipitates in the form of conglomerates, which leads to blockage of the ascending part of the Henle loop, and concentrating in the distal nephron, causes the development of acute tubular necrosis. Defeat of renal structures is also facilitated by severe hyperkalemia, which can reach 7 mmol/l, impaired intrarenal hemodynamics, increased tubular reabsorption of water and sodium [8, 70]. Most studies have determined rhabdomyolysis based on creatine phosphokinase values above the upper limit of normal [71]. Cytolytic syndrome is manifested by hypermyoglobinemia, when there are spiky concentrations of CPK (more than 1000 units/l), increased LDH, AST, ALT, life-threatening hyperkalemia (up to 10.7 mmol/l), hyperphosphatemia, metabolic acidosis, increased plasma concentration hyperosmolarity (urea, creatinine, urea nitrogen, uric acid), myoglobinuria, positive NGAL test. Lipocalin – anew biomarker for early diagnosis of ARF: reference values (USA): in plasma – men: 18–196 ng/ml; – women: 35–151 ng/ml; urine: <131.7 ng/ml. A strong correlation was found between the intensity of specific histochemical staining of NGAL and the duration of cold ischemia [72]. Myoglobin, free hemoglobin, massive proteinuria, cylindruria (granular, waxy cylinders) are present in the urine. In the urine sediment – erythrocytes, hematin crystals. The proportion of urine tends to decrease, proteinuria develops [63, 70, 73].

Plasma extravasation is characterized by the development of hemoconcentration and increased blood viscosity, which may lead to the development of disseminated intravascular coagulation [21, 33, 36, 50, 74]. After tissue warming, internal plasma loss occurs, hemodilution develops, aggregation of blood cells leads to thrombosis of arterioles and larger vessels within the first 2–3 days after warming, sharply increases the permeability of cold-injured endothelium [50].

Intravascular blood clotting can begin as a result of one of three closely related processes:

1) damage to the vascular endothelium (triggers the internal mechanism of blood clotting);

2) activation of the Hagemann factor, followed by the whole internal path blood clotting that occurs with cold injury due to systemic inflammatory response syndrome;

3) release of thromboplastins with activation of the external blood clotting pathway in direct temperature tissue damage.

Consumption of coagulation factors by DIC leads to their deficiency when they are needed to achieve hemostasis. The main morphological signs of kidney damage in DIC are numerous fibrin clots in small vessels, which cause the development of microinfarctions. In the final stage of DIC against the background of progression of microcirculatory disorders in the organs progresses multiorgan failure and tissue hypoxia (lactic acidosis), there are signs of hemolysis, azotemia ("shock kidney"), there is macrohematuria [76, 77, 78, 79, 80, 81].

Recovery period.

This period is characterized by the presence of hypercatabolism and fever, but exacerbation of pre-existing chronic kidney disease is rare [26]. Toxemia lasting at least two weeks usually results in prolonged asthenia and other severe long-term complications, including chronic renal failure. Immunity suppression often leads to secondary infection, which leads to the development of acute cystitis, pyelonephritis, etc. [26, 82]. Chronic glomerulonephritis is a typical visceral complication of cold injury [52].

Thus, lesions of the urinary system in general and local cold injuries are a frequent complication of unintentional hypothermia, which is accompanied by significant delayed mortality and disability. The authors hope that the collected material will be useful to practitioners in providing assistance to victims of accidental hypothermia.

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РЕФЕРАТ

Патофізіологічні зміни сечовидільної системи внаслідок ненавмисної холодової травми (науково-літературний огляд)

В.В. Єхалов, О.В. Кравець,
В.П. Стусь, С.І. Баранник,
М.Ю. Поліон, С.Л. Чеканов

Гостре пошкодження нирок (ГПН) спостерігається більш ніж у 40% пацієнтів з холодовою травмою, а функціональний стан нирок при ненавмисній гіпотермії та відмороженнях є прогностичним фактором виживання. У своєму науково-літературному огляді на основі інформації доступних джерел автори вперше спромоглися навести динаміку морфо-функціональних ушкоджень сечовидільної системи в залежності від ступеня загального ненавмисного переохолодження організму. Окремо розглянуто патофізіологічні зміни функції нирок у дореактивному, післяреактивному та у періоді відновлення. Наведено поняття про «смертельну тріаду», «кальцієвий парадокс», «зігрівальний ацидоз» та сучасні біомаркери для визначення раннього ГПН. Автори сподіваються, що зібраний матеріал стане в нагоді практичним лікарям при наданні допомоги постраждалим з випадковою загальною гіпотермією.

Ключові слова: холодова хвороба, загальне переохолодження організму, стадії, гостре пошкодження нирок.

РЕФЕРАТ

Патофизиологические изменения мочевыводящей системы вследствие непреднамеренной холодовой травмы (научно-литературный обзор)

В.В. Ехалов, О.В. Кравец,
В.П. Стусь, С.И. Баранник,
Н.Ю. Полион, С.Л. Чеканов

Острое повреждение почек (ОПН) наблюдается более чем у 40% пациентов с холодовой травмой, а функциональное состояние почек при непреднамеренной гипотермии и отморожениях является прогностическим фактором выживаемости. В своём научно-литературном обзоре на основе информации доступных источников авторы впервые попытались раскрыть динамику морфо-функциональных повреждений мочевыделительной системы в зависимости от степени общего случайного переохлаждения организма. Отдельно рассмотрены патофизиологические изменения функции почек в дореактивном, постреактивном и в периоде восстановления. Приведены понятия о «смертельной триаде», «кальциевом парадоксе», «согревающем ацидозе» и современных биомаркерах для определения раннего ОПН. Авторы надеются, что собранный материал будет полезен для практических врачей при оказании помощи пострадавшим со случайной общей гипотермией.

Ключевые слова: холодовая болезнь, общее переохлаждение организма, стадии, острое повреждение почек.