Ukrainian Journal of Nephrology and Dialysis
Scientific and Practical, Medical Journal

**Founders:**
- State Institution «Institute of Nephrology NAMS of Ukraine»
- National Kidney Foundation of Ukraine

**ISSN 2304-0238;**
**eISSN 2616-7352**

**Journal homepage: https://ukrjnd.com.ua**

## Clinical case reports

doi: 10.31450/ukrjnd.4(68).2020.03

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# Acute kidney injury and sinus bradycardia associated with near-drowning

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Citation: Kemeç Z, Gürel A. Acute kidney injury and sinus bradycardia associated with near-drowning. Ukr J Nephr Dial. 2020;4(68):18-22. doi: 10.31450/ukrjnd.4(68).2020.03

**Article history:**
*Received May 29, 2020*
*Received in revised form July 18, 2020*
*Accepted August 5, 2020*

**Abstract.** *Acute kidney injury (AKI) occurs in different situations and may have a variable prognosis due to underlying cause, clinical setting and comorbidity. Near-drowning is known to lead to bradycardic rhythms which can lead to hypoxia because of hypoperfusion. AKI has a high risk of mortality and morbidity. However, sequelae of sinus bradycardia are related to its underlying etiology. Urinary, cardiovascular and respiratory disorders are more frequently seen after near-drowning. Near-drowning related AKI and sinus bradycardia are not reported together in the literature. We aimed to emphasize these complications in near-drowning patients.*

**Key words:** *Near-drowning, acute kidney injury, sinus bradycardia.*

**Conflict of interest statement:** all the authors declared no competing interests.



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УДК: 616.61-008.6-036.11:616.12-008.314-092

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# Гостре пошкодження нирок та синусова брадикардія асоційовані з нефатальним утопленням

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**Резюме.** *Гостре пошкодження нирок (ГПН) може ускладнювати різні клінічні ситуації та мати різний прогноз залежно від основної причини, клінічних умов та супутньої патології. Відомо, що утоплення призводить до брадикардії, яка може бути причиною гіпоксії через гіпоперфузію. ГПН має високий ризик смертності. Порушення серцево-судинної системи дихання та сечовиділення часто спостерігаються після нефатального утоплення, але у науковій літературі майже не існує повідомлень з цієї проблеми. У цій роботі ми продемонстрували клінічний випадок ГПН та синусової брадикардії, асоційованих з нефатальним утопленням.*

**Ключові слова:** *нефатальне утоплення, гостре пошкодження нирок, синусова брадикардія.*

**Introduction.** The risk factors for acute kidney injury (AKI) include many different and variable factors, which are critical as they are associated with numerous reported cases of high mortality and morbidity worldwide. Although the typical flow diagram splits into prerenal, renal, and postrenal subgroups. Some of the clinical entities may not be defined within a specific subset, which is the case for AKI associated with near-drowning [1]. Only a small number of such cases have been reported. The case-control study of 30 hospitalized near-drowning victims has shown that 50 % of the patients had acute renal impairment [2]. This might in part be due to poor recognition of this condition.

It is clearly known that inadequate ventilation leads to hypoxia, and hypoxia leads to bradycardic rhythms; likewise, it is known that near-drowning leads to bradycardic rhythms, which can lead to hypoxia because of a lack of perfusion. Sinus bradycardia may be defined as a sinus rhythm with a resting heart rate of 60 beats per minute or less. However, few patients actually become symptomatic until their heart rate drops to less than 50 beats per minute. The action potential responsible for this rhythm arises from the sinus node and causes a P wave on the surface electrocardiography (ECG) that is normal in terms of both amplitude and vector. The presence of sinus bradycardia in itself does not cause a change in the QRS complex and T wave [3].

Here we present a rare cause of AKI and sinus bradycardia in a middle-aged male due to near-drowning. We have been unable to find together documented instances of these complications in the literature.

**Case report.** A 40-year-old male without significant past medical history was admitted with nausea, chest pain, and lumbar pain to our Nephrology Outpatient Clinic. He had a history of near-drowning in the Black Sea while he was swimming with his son. The duration of immersion was about 10–15 minutes. Our patient was resuscitated on the beach and aspirated sea-water was cleared from the airways. He was taken to the hospital and discharged as his kidney function tests were normal. He contacted us after 16 hours. We admitted the patient for inpatient stay at our Nephrology Clinic. He underwent 12 days of uneventful observation. His admission creatinine (Cre) level was high (4.83 mg/dL). During the physical examination, he was conscious and cooperative. Blood pressure was 110/70 mmHg and heart rate was 57/min, respiratory rate was 20/min, weight 65 kg, height 169 cm and body temperature was 36.4°C. Urine output was >400 mL/day. Biochemical tests on admission are summarized in Table 1.

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*Table 1*

**Dynamics of the patient laboratory data**

| Date | One day[x] | Two day | Four day | Five day | Six day | Twelve day | seventy-five day[y] | Reference range |
|---|---|---|---|---|---|---|---|---|
| Glucose | 105 | 115 | | | 124 | | | 70 – 105 mg / dL |
| Urea | 88.1 | 92.1 | 76.9 | 60.1 | 53.8 | 27.6 | 35.5 | 8 – 50 mg / dL |
| Cre | 4.83 | 5.86 | 4.76 | 3.49 | 2.74 | 1.47 | 1.19 | 0.05 – 1.3 mg / dL |
| Uric acid | 15 | 13.5 | 9.5 | 8.4 | 8 | | | 02.5 – 8 mg / dL |
| Na | 139 | 140 | 140 | | | | | 132-150 mmol / L |
| K | 4 | 4.7 | 4.6 | | | | | 3 – 5.5 mmol/L |
| Cl | 102 | 106 | | | | | | 90-115 mmol/L |
| Ca | 10.3 | 9.5 | 9.4 | | | | | 8.3 – 10.6 mg / dL |
| LDH | 263 | | | | | | | 40-310 U/L |
| CK | 131 | 110 | 675 | 357 | | | | 15-175 U/L |
| MYG | | | | 128.2 | | | | 16-74 ng/mL |
| P | 5.5 | 4.4 | 4.4 | | | | | 2 – 6 mg / dL |
| WBC | 14,2 | | | | | | | 3,5 – 11x109/ L |
| CRP | 28.1 | 26.4 | 14.4 | | 11.2 | 0.4 | | 0-10 mg / L |
| pH | 7.34 | 7.36 | 7.4 | | | | | 7.35-7.45 |
| HCO3 | 18.2 | 16.1 | 22.1 | | | | | 22-26 mEq/L |

[x]Time of increased serum creatinine levels.

[y]Time of creatinine serum levels has returned to normal.

**Abbreviations;** Cre: creatinine, Na: sodium, K: potassium, Cl: chloride, Ca: calcium, LDH: lactate dehydrogenase, CK: creatine kinase, MYG: myoglobin, P: phosphorus, WBC: white blood cell, CRP: C-reactive protein, HCO3: bicarbonate

**Note:** Since some of the data were not analyzed on certain days, related cells of the table were left blank.

Urinalysis revealed a density of 1008, pH: 6, protein +++, leukocytes: 5/HPF and erythrocytes: 14/HPF, without eosinophils. Daily urinary protein excretion was 576 mg in the 24-hour urine sample. Creatinine kinase (CK) level was 131 IU/L on admission (second day after near-drowning) and it increased to 675 IU/L and 357 IU/L on the third and fourth days after admission. On the 6th day, myoglobin was slightly elevated. He had chest pain on the 4th day of hospitalization. Cardiac markers were normal. The electrocardiography showed sinus bradycardia (Fig. 1).

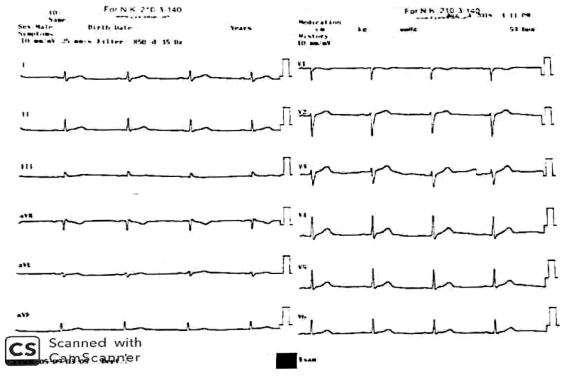


Fig. 1. The heart rate of 57/bpm in the patient.

Thyroid hormones were in physiological reference intervals. Chest X-ray and renal ultrasonography were within the normal range. Boundary metabolic acidosis, hypovolemia, azotemia were found. 250 cc/h of intravenous fluid was administered. The patient was complaining of lumbar pain and dysuria with high C-reactive protein (CRP) levels. Urine culture was taken; an empirical antibiotic was started. Urine culture was negative. He was followed up daily for Cre levels and urine output. There was no need for hemodialysis. Cre levels started to decrease without renal replacement therapy (see Table 1). He was discharged from the hospital on Day 12. Ten weeks after near-drowning, Cre was in the normal range and ECG showed normal sinus rhythm (Fig. 2). During his outpatient follow-up, renal functions remained normal.

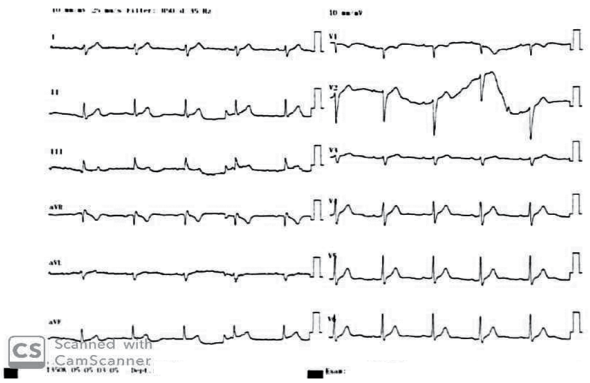


Fig. 2. The heart rate of 68/bpm in the patient.

**Discussion.** Near-drowning is accepted as the initial survival of the patient at least 24 hours after the incident [4]. Cardiac arrest, brain edema, massive gastrointestinal hemorrhage, pulmonary edema, adult respiratory distress syndrome, pneumonia, and hypoxic encephalopathy may occur frequently after near-drowning. However, patients can also be asymptomatic. Near-drowning is a rare cause of AKI, but patients who experience this catastrophic situation should be closely followed up for this complication [5]. Unfortunately, the exact pathogenesis of AKI after near-drowning has not been well known. In animal models, hypoxia and reperfusion injury are suspected as the cause of hemodynamic damage. The leading pathophysiological events during drowning or near-drowning are arterial oxygen desaturation and cardiac output decrease. Multiorgan failure, cerebral autoregulation dysfunction and rhabdomyolysis may contribute to AKI [2, 6].

The patient was admitted to our center on the second day of the incident. Although CK and myoglobin levels were slightly high on the fourth day, it was not clear whether AKI was solely dependent on and related to rhabdomyolysis. This may happen without overt clinical signs of vascular collapse. The normalization of renal functions was in nearly 10 weeks, which is similar to the recovery phase of acute tubular necrosis. The clinical course in this patient is entirely consistent with that of acute renal failure due to acute tubular necrosis. The diagnosis is supported by the electrolyte and osmotic composition of urine during the period of renal failure, the absence of any other cause for renal failure in previously healthy young men, and recovery of renal functions after ten to twelve days. The patients were followed up for 3 and 6 months, respectively, and no evidence of residual renal insufficiency was found in either period. Although no histological evidence of acute tubular necrosis was obtained in these patients, the clinical picture was not that of acute glomerular disease, bilateral obstruction, or persistent bilateral renal arterial insufficiency. This lack of evidence for other renal failure causes, taken together with the clinical courses of these patients, seems sufficient to establish the diagnosis of acute tubular necrosis.

In a study including 30 near-drowning patients, it was shown that acidosis on admission was the best predictor for acute renal failure. In our patient, metabolic acidosis was at the upper limit. Also in this retrospective analysis, "isolated acute renal injury" was defined in this group as requiring hemodialysis without overt extrarenal findings, particularly in the respiratory system. The cases with severe isolated AKI after near-drowning are very seldom reported [5]. In another study, Alp A et al present a rare cause of AKI in a middle-aged man with hemodialysis [1]. Our case had AKI. There was no requirement for hemodialysis. Renal functions improved with controlled and effective fluid replacement. We thought that CRP and WBC elevation in our patient may be associated with lung infection as a result of bronchial-alveolar irritation caused by aspirated seawater. He benefited from ampicillin-sulbactam given prophylactically.

Causes of sinus bradycardia include the following: the sick sinus syndrome, medications, a broad variety of other drugs and toxins. Less commonly, the sinus node may be affected as a result of diphtheria, rheumatic fever, or viral myocarditis [3]. Disordered physiological processes that are related to sinus bradycardia is dependent on the etiology of the disorder [7]. Commonly, sinus bradycardia is an incidental finding in otherwise healthy individuals, particularly in young adults, athletes or sleeping patients [8]. Other causes of sinus bradycardia are related to increased vagal tone. Physiologic causes of increased vagal tone include the bradycardia seen in athletes. Pathologic causes include, but are not limited to, inferior wall myocardial infarction, toxic or environmental exposure, electrolyte disorders, infection, sleep apnea, drug effects, hypoglycemia, hypothyroidism, and increased intracranial pressure [3]. Our patient had chest pains. Sinus bradycardia was detected on the ECG. Cardiac enzymes were normal. There was no need for atropine. When chest pain did not respond to paracetamol, 50 mg tramadol was used for control. We concluded that our patient may have chest pain associated with rhabdomyolysis. After discharge, ECG returned to normal sinus rhythm. What was the cause of the bradycardia, hypoperfusion, and hypoxia in this patient? It is well known that inadequate ventilation leads to hypoxia, and hypoxia leads to bradycardic rhythms. Likewise, it is known that hypothermia causes bradycardic rhythms, which in turn leads to hypoxia due to a lack of perfusion [9]. Thyroid hormones, calcium, magnesium and glucose values of our patient were within normal values. There was no drug or alcohol intake. He did not participate in any sports activity. Thus, bradycardia may be due to the interaction of hypoxia and vagal tone.

Our case has a limitation. We did not perform a kidney biopsy for diagnostic purposes. As we mentioned above, we avoided interventions that require invasive intervention as clinical and laboratory findings support acute tubular necrosis

**Conclusions.** In conclusion, in the case of sea-water drowning, physicians should consider AKI and sinus bradycardia. Our case will be the first in this aspect. The effect of sinus bradycardia on progression can be neglected; renal failure can often be reversed with medical treatment and rarely complicated.

**Informed consent:** Informed consent was obtained from the patient included in the study.

**Disclosure statement:** No potential conflict of interest was reported by the authors.

**Authors contribution.**

***Zeki Kemeç*** and ***Ali Gürel*** contributed to the analysis of the results and the writing of the manuscript.

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