Continuing Medical Education

Acute Kidney Injury

A Frequently Underestimated Problem in Perioperative Medicine

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Summary

Background: Surgical patients are getting older with increasing comorbidity. Acute kidney injury (AKI) is a commonly underestimated perioperative complication. 2–18% of hospitalized patients and 22–57% of patients in the intensive care unit develop AKI. Even though it has a major impact on patients' outcomes, it goes unrecognized in 57–75.6% of cases.

Methods: This review is based on pertinent papers retrieved by a selective search in PubMed and the Cochrane Library employing the searching terms “acute kidney injury,” “biomarker,” “perioperative,” “renal function,” and “KDIGO.”

Results: The pathophysiology of AKI is complex. Conventional biomarkers are either not specific enough (urine output) or not sensitive enough (serum creatinine) for timely diagnosis. In view of the pathophysiology of the condition and the limited treatment options for it, the early detection of subclinical AKI (kidney damage without functional impairment) would seem to be a reasonable first step toward the prevention of worsening or permanent renal injury. New biomarkers of damage enable the early initiation of nephroprotective interventions. According to the “Kidney Disease: Improving Global Outcomes” (KDIGO) statement, a multimodal treatment approach is needed, including, among other things, optimization of hemodynamics and the discontinuation of nephrotoxic drugs.

Conclusion: It is essential to identify patients at risk and sensitize the treating personnel to the implementation of the guidelines. The incorporation of new biomarkers into routine clinical practice is also reasonable and necessary. Future clinical trials must show in what form these biomarkers should be used (singly or collectively).

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The treatment of elderly multimorbid patients is assuming greater importance in surgical medicine. Avoidance of complications is important in the perioperative phase, and in this context the magnitude and significance of acute kidney injury (AKI) are often underestimated (1). Between 2% and 18% of all patients admitted to the hospital (2, 3) develop AKI during their hospital stay, as do 22% to 57% of intensive care patients (4, 5). This leads to an increase of 3% to 30% in mortality, more work for medical staff, and additional costs (6–8). The mortality among patients who become dependent on dialysis during their hospital stay is drastically increased and lies between 14% and 41% (9). Despite all this, AKI seems not to receive adequate recognition or attention: in Germany and elsewhere, 57% to 75.6% of cases go unde-

tected, undiagnosed, and/or undocumented (10–13). As well as leading to significant underestimation of prevalence, this implies a lack of knowledge and awareness. AKI is an independent risk factor for the occurrence of cardiovascular/cerebrovascular complications and the development of chronic renal insufficiency (4, 14, 15). It is therefore morally, medically, and economically imperative to broaden the awareness of AKI.

Methods

The databases PubMed and Cochrane Library were selectively searched for records featuring the terms “acute kidney injury,” “biomarker,” “perioperative,” “renal function,” or “KDIGO,” with the emphasis on studies published in the past 15 years.

Prevalence

Between 2% and 18% of patients admitted to the hospital and 22% to 57% of intensive care patients develop AKI during their hospital stay.

Independent risk factor

Acute kidney injury is an independent risk factor for the occurrence of cardiovascular/cerebrovascular complications and the development of chronic renal insufficiency.

TABLE

KDIGO: AKI classification and criteria

Severity	Serum creatinine concentration	Urine excretion
1	1.5- to 1.9-fold within 7 d or ≥ 0.3 mg/dL within 48 h	<0.5 mL/kg/h over a period >6 h
2	2.0- to 2.9-fold	<0.5 mL/kg/h for >12 h
3	≥ 3-fold or serum creatinine ≥ 4 mg/dL with an acute increase ≥ 0.5 mg/dL	<0.3 mL/kg/h for >24 h or anuria >12 h

AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes

Learning goals

After finishing this article, the reader should be:

- Aware that an unrecognized and untreated AKI and its consequences are significant
- Familiar with the multimodal approach for prevention and treatment of AKI
- Thoroughly acquainted with the complex pathomechanism of AKI.

Definition

Acute kidney injury is diagnosed and classified according to established criteria (the three-stage KDIGO [Kidney Disease: Improving Global Outcome] classification of severity) (16). The KDIGO staging is based on the determination of two readily available parameters, serum creatinine concentration and urine output (*Table*). For more detail, please see the recently published review article by Alscher et al. (17).

AKI is a broad clinical syndrome that embraces various etiologies. It affects both renal structure and renal function, and is characterized by abrupt deterioration of the latter. AKI thus includes renal failure but encompasses more than that. Kidney injury unaccompanied by loss of function (subclinical AKI) may also occur, and AKI may be reversible (*Figure 1*) (16).

Physiology and pathophysiology

In the situation of normotension, perfusion of the kidney is maintained by renal autoregulation. The goal is to ensure a sufficient glomerular filtration rate by means of “redistribution” despite fluctuations in renal blood flow.

Definition

The diagnosis of acute kidney injury is based on determination of two readily available parameters, serum creatinine concentration and urine output.

In the perioperative period various factors can act to reduce the renal blood flow, thus leading to renal hypotension and consequently an insufficient supply of oxygen (*Figure 2*). At this point two potent renal vasoconstrictors can be mentioned: antidiuretic hormone (ADH), which is released in greater quantities at times of stress, and angiotensin II (18, 19). Regional changes in renal blood flow arise, damaging the endothelium and triggering an inflammatory reaction (20). Disruption of the microcirculation (20, 21) and occlusion of small-caliber vessels lead to local microischemia.

Owing to its low oxygenation and the nature of its vascular architecture, the outer medulla of the kidney is very sensitive to changes in perfusion and oxygen supply (22). Furthermore, the blood supply is compromised by locally occurring edema. The imbalance between production of and demand for oxygen and nutrients harms the tubular epithelial cells. Cytokines are released, again attracting inflammatory cells and damaging tubular cells. A vicious circle arises and harmful waste products accumulate. In parallel, repair mechanisms are initiated (21). Mild kidney injury can be compensated and noticeable loss of function avoided. If this renal reserve is exhausted, long-term kidney disease may result.

Urine and serum creatinine: functional biomarkers

Kidney function is evaluated by determination of the amount of urine excreted and the serum concentration of creatinine. Urine and serum creatinine serve as functional biomarkers, but are of limited value. Around 400–500 mL of urine has to be excreted each day to eliminate nitrogen-containing waste products. Perioperative factors (*Figure 2*) often lead to oliguria (urine <0.5 mL/kg/h). One way of determining a patient’s precise glomerular filtration rate would be to measure the amount of urine excreted each hour (or 24 h). This approach is, however, cumbersome, time-consuming, and error-prone (e.g., distortion by medication), especially in patients with pre-existing AKI. Other factors also contribute to oliguria, such as stress-related release of ADH, which inhibits the excretion of free water. In the past, a perioperative decrease in urine output was frequently treated with volume substitution, which resulted in hypervolemia and tissue edema (see “Hemodynamics” below). Because the kidney itself is surrounded by a tissue capsule, edema can contribute to elevated pressure within the capsule and thus to reduced organ perfusion. A general increase in abdominal pressure, e.g., during laparoscopic surgery or in the

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presence of abdominal compartment syndrome, also lowers the amount of urine produced. Studies have demonstrated that an decreased urine output is associated with a poorer outcome of treatment in both adults (23) and children (24). However, a decreased amount of urine does not necessarily indicate a renal function disorder. (Short) episodes of oliguria are not always associated with AKI as defined in the current KDIGO criteria (25–27).

Perioperative urine output is affected by many different factors and thus possesses only low specificity. Transient or less pronounced changes go undetected, although the kidney may already have been injured.

Limitations also apply to serum creatinine. It is the end product of the creatine phosphate metabolic pathway, which primarily serves to supply energy to muscle, liver, and brain. The concentration of creatinine in serum is determined principally by muscle mass, nutrition, activity, and hemodilution. Cephalosporins and barbiturates can also increase the serum concentration. The serum creatinine concentration and the glomerular filtration rate have an exponentially antiproportional relationship to one another: even only slight elevations of serum creatinine cause a reduction of $\geq 50\%$ in renal function, which is associated with a poorer prognosis (28, 29). The relationship is hyperbolic: even when 50% of functional kidney tissue is lost, there is “only” a doubling of serum creatinine concentration, which thus possesses low sensitivity. Moreover, the concentration gives no indication of the renal reserve (30).

Particularly in elderly and muscle-deficient patients, the serum creatinine concentration can be near normal despite a marked reduction in the glomerular filtration rate. In this situation, determination of serum creatinine may lead to significant overestimation of renal function (31). The rise in serum creatinine level and the decrease in urine output are not early enough indicators, and their sensitivity and specificity are too low for timely diagnosis of AKI (32). On pathophysiological grounds, however, it can be assumed that early intervention is a crucial factor for success of treatment. This justifies the search for new biomarkers that not only show deterioration of renal function but also permit earlier identification of structural damage to the kidney (32).

New injury markers

Investigations have demonstrated that various new renal biomarkers show the presence of kidney injury (33–35). The goal is to identify and treat subclinical

Biomarker “urine output”

Perioperative urine output is affected by many different factors and thus possesses only low specificity. Transient or less pronounced changes go undetected, although the kidney may already have been injured.

FIGURE 1

Schematic depiction of the relationships between acute kidney disease/disorder (AKD), acute kidney injury (AKI), subclinical AKI (sAKI), and chronic kidney disease (CKD)

AKI. Studies have emphasized that many new biomarkers are indeed superior to the conventional markers in early diagnosis, determination of prognosis, and estimation of long-term mortality. Perioperative increases in concentration were significantly associated with the occurrence of postoperative AKI (36–40, e1–e3). For example, the product of TIMP-2 and IGFBP7 predicted moderate to severe AKI with 92% accuracy. In contrast to the aforementioned glomerular processes, the new injury markers are released in the tubules—the most likely site of injury. They permit conclusions regarding various injury mechanisms and aspects of renal function (36–38). These markers include NGAL, TIMP-2, IGFBP7, and cystatin C, all of which can be determined more and more economically via routine laboratory procedures and point-of-care systems. A more detailed account would exceed the scope of this article.

The concept of angina renalis, subclinical AKI, and biomarker-guided treatment

Sensitive biomarkers such as troponin have helped to improve the early detection and treatment of ischemic myocardial insults. The year 2010 saw the introduction of the concept of “angina renalis.” In contrast to acute coronary syndrome, AKI is asymptomatic, more

New injury markers

Studies have emphasized that many new biomarkers are superior to the conventional markers in early diagnosis, determination of prognosis, and estimation of long-term mortality.

FIGURE 2

Flow chart of the pathophysiology of acute kidney injury

complex in origin, and diagnosis using conventional functional markers is delayed, postponing the initiation of treatment. For these reasons, the two clinical presentations are difficult to compare. The concept of angina renalis is intended to achieve early renal risk stratifi-

cation with the aid of biomarkers. This is the context in which the term “subclinical AKI” was coined to describe a biomarker-positive yet (still) serum creatinine-normal status—kidney injury without functional impairment (e4) (Figure 1). Biomarker-positive

The concept of “angina renalis”

The “angina renalis” concept is intended to achieve early renal risk stratification with the aid of biomarkers. This is the context in which the term “subclinical AKI” was coined.

Further risk stratification

In patients with pre-existing AKI, the furosemide stress test can be carried out to estimate the severity of the AKI. This is useful for further stratification of the risk.

patients seem already to be at higher risk of complications (renal replacement therapy: odds ratio [OR] 16.4, 95% confidence interval [3.6; 76.9], $p = 0.001$; hospital mortality: OR 2.8 [1.9; 4.1], $p = 0.001$) (e5, e6). Although an increase in the biomarkers predicts AKI and a subsequent rise in serum creatinine concentration, the (current) situation is that, by definition, AKI can be diagnosed only on the basis of changes in the serum concentration of creatinine or in the amount of urine produced. According to a consensus definition published in 2017, AKI can also be identified on the basis of a biomarker increase without a change in serum creatinine (e7). The early release of biomarkers in the injured renal tubuli could open a time window to prevent further damage and thus avoid impairment of renal function (Figure 3) (e8). The additional implementation of clinical risk scores (e.g., the renal risk index) and consideration of the clinical context increase the informational worth and improve the negative predictive value (e9–e11). In patients with pre-existing AKI, the furosemide stress test can be carried out to estimate the severity of the AKI. This is useful for further stratification of the risk (AKI progression, need for renal replacement therapy, mortality) (e12, e13).

The new biomarkers represent an important means of detecting AKI and planning the appropriate treatment. They should therefore be implemented in routine clinical practice, provided the local health care system has the necessary resources—ideally together with the KDIGO bundle.

Treatment options in the perioperative phase

It was long assumed that AKI was neither preventable nor treatable. The findings of more recent studies led to a paradigm shift.

The KDIGO bundle

The KDIGO bundle is a multimodal approach and recommends prophylactic measures to be carried out in patients at high risk of AKI. These comprise avoidance of nephrotoxic substances, optimization of volume status and perfusion pressure, maintenance of normoglycemia, regular monitoring of serum creatinine and urine excretion, and, if required, extension of hemodynamic monitoring (Box) (e14).

Two recently published randomized controlled clinical trials illustrated that biomarker-guided implementation of the KDIGO bundle, compared with standard treatment, led to a significant reduction in the occurrence of AKI in both heart surgery patients and general surgery patients (Prevention of Acute

FIGURE 3

Schematic model of acute kidney injury

In subclinical acute kidney injury (sAKI) there is a biomarker-positive status without loss of function. If the damage progresses, the glomerular filtration rate (GFR) decreases further and acute kidney injury (AKI) arises. While the GFR goes down, the concentrations of the biomarkers increase

Kidney Injury [PrevAKI]: 55.1% versus 71.1%, $p = 0.004$; Biomarker-guided Intervention to Prevent Acute Kidney Injury [BigpAK]: 27.1% versus 48.0%, $p = 0.03$) (e15, e16).

In this context, one must also consider the treating medical personnel. Investigations into the implementation of guideline recommendations showed clearly that although the measures ought to be known, they were properly applied in only 24% of cases. As one complex example, the sepsis bundle achieved an acceptance rate of only 34%, while the glucose monitoring component reached acceptance of 45% (e17). Another study on the implementation of recommendations with regard to AKI found a similarly poor rate: the AKI care bundle was completed in only 26% of patients within 24 hours (e18). Steps must definitively be taken in respect of training, information, and exemplary action.

The KDIGO bundle

The KDIGO bundle comprises avoidance of nephrotoxic substances, optimization of volume status and perfusion pressure, maintenance of normoglycemia, and regular monitoring of serum creatinine and urine output.

Hemodynamics

Renal underperfusion with the consequent shortage of oxygen and inflammatory reactions are thought to be two of the main factors responsible for the development of acute kidney injury.

BOX

KDIGO bundle recommendations by severity of AKI

● High risk

- Discontinue all nephrotoxic medications, if possible
- Optimize volume status and perfusion pressure
- Consider hemodynamic monitoring
- Monitor serum creatinine and urine output
- Avoid hyperglycemia
- Do not use contrast medium

● Severity

KDIGO-AKI 1

- Consider non-invasive diagnostics
- Consider invasive diagnostics

KDIGO-AKI 2

- Consider dose adjustment of medications
- Consider renal replacement therapy
- Consider management in intensive care unit

KDIGO-AKI 3

- Avoid subclavian catheterization

Treatment measures become more important from top to bottom
AKI, acute kidney injury; KDIGO, Kidney Disease: Improving Global Outcomes

Hemodynamics

Renal underperfusion with the consequent underoxygenation and inflammatory reactions are thought to be two of the main factors responsible for the development of AKI. AKI is associated with intraoperative hypotension (e19, e20). Moreover, persistent hypotensive episodes lead to impaired autoregulation (e21).

Perioperative hemodynamic monitoring, by whatever method, results in reduced occurrence of AKI (e22–e25). In a large retrospective cohort study of 5127 non-cardiosurgical patients, AKI was found whenever the intraoperative mean arterial pressure was <60 mm Hg for ≥ 20 min or <55 mm Hg for ≥ 10 min (adjusted OR 2.34 [1.35; 4.05], $p < 0.05$ (e19). Hypotensive phases should therefore be kept as

brief as possible (e19). Nevertheless, there is no universal optimal blood pressure, as the blood pressure depends on the patient's medical history and current status. The INPRESS study (Intraoperative Norepinephrine to Control Arterial Pressure) investigated whether a personalized blood pressure regimen influenced the postoperative function of various organ systems in patients with an elevated risk of AKI. Patients whose systolic blood pressure was kept within 10% above or below the systolic resting pressure had a lower risk of postoperative organ dysfunction (relative risk 0.73 [0.56; 0.94], $p = 0.02$; absolute risk difference –14% [–25; –2]). Renal function disorders occurred less often in the individualized group (32.7%) than in the standard control group (49%; absolute risk difference –16% [–27; –5], adjusted relative risk 0.70 [0.53; 0.92], $p = 0.01$) (e26). Arterial hypotension is a common consequence of hypovolemia. In a recently published study, 3000 abdominal surgery patients were randomized into a liberal volume group and a restricted volume group. Those in the restrictive arm of the study were more likely to develop AKI after their operation (8.6% versus 5.0%, $p < 0.001$) and more likely to need renal replacement (0.9% versus 0.3%, $p = 0.048$) (e27). By contrast, numerous studies have found that volume overload worsens renal function and increases overall mortality (e28, e29). In this context, distant organ damage deserves mention: AKI can also have extrarenal effects, e.g., hyperpermeability, that would aggravate volume overload (e.g., pulmonary edema) (e30–e32). Therefore it seems reasonable to take an intermediate approach to ensure a balanced volume status (Figure 4).

Just as important is the choice of substance for substitution. Isotonic saline solution 0.9% contains an unphysiologically high amount of chloride, leading to hyperchloremic acidosis and to renal vasoconstriction with reduced renal blood flow and a decreased glomerular filtration rate (e33–e36) as well as elevated incidence of AKI (e37). A chloride-restricted infusion regimen is associated with reduced incidence of moderate and severe AKI (e38–e40). Balanced crystalloid solutions should therefore be preferred (e41).

Most guidelines recommend norepinephrine as the first-line blood pressure-supporting medication. Although it is not yet known which vasopressor drug exerts the greatest protective effect against the development of AKI, norepinephrine raises both the global and medullary blood pressure and thus increases diuresis.

Effect of hemodynamic monitoring

Perioperative hemodynamic monitoring, by whatever method, results in reduced occurrence of AKI.

Recent findings

In a recently published study, 3000 abdominal surgery patients were randomized into a liberal volume group and a restricted volume group. Those in the restrictive arm of the study were more likely to develop AKI after their operation and more likely to need renal replacement.

Diminished cardiac ejection fraction and right ventricular dysfunction should be considered in this context. If the right ventricle is compromised, venous outflow is decreased and blood backs up all the way to the kidney. Together with development of interstitial capsular edema there is increased organ resistance, potentially leading to a significant reduction in renal blood flow and thus to a higher risk of AKI (e42). This may be indicated by elevated central venous pressure, a “drainage marker” that may be associated not only with an increased risk of kidney injury but also with greater severity of kidney injury (e43–e45).

In this context, maintenance of the cardiac index makes sense (e46). From the pathophysiological viewpoint the administration of vasopressors would be indicated in these phases, with or without inotropics and accompanied by extended hemodynamic monitoring. If nothing else, renal oxygen deficiency may be caused by a lack of oxygen carriers. Preoperative anemia with hemoglobin <8 mg/dL is associated with an up to four-fold risk of AKI (e47, e48). It must be remembered that blood transfusions constitute an independent risk for AKI (e49–e51).

Pharmacological recommendations/treatment

There is currently no pharmacological option for the prevention and treatment of AKI. For some years, dexmedetomidine, a highly selective α_2 -agonist with pleiotropic effects (e.g., sedative, analgesic, and anxiolytic effects, decreased endogenous norepinephrine release, improvement of hemodynamic stability, and the balancing of myocardial oxygen supply and demand), has been gaining in popularity. In heart surgery, administration of dexmedetomidine led to a significant reduction in the occurrence of AKI in patients whose renal function was normal or only slightly impaired before operation (e52–e54). The incidence of AKI in the first 48 h after surgery was significantly lower in the intervention group than in the control group (14% [n = 14] versus 33% [n = 33]) (e55). Dexmedetomidine seems to bring about a dose-dependent reduction of the increase in neutrophil gelatinase-associated lipocalin (e56), and may therefore have a nephroprotective action. However, further randomized controlled trials are required before dexmedetomidine can be definitively recommended for use in treatment.

Some studies have shown that volatile anesthetics possess nephroprotective and anti-inflammatory properties, although they may induce reductions in renal blood flow and glomerular filtration rate (e57, e58). Volatile anesthetics are believed to ameliorate

Central venous pressure as “drainage marker”

Elevated central venous pressure is a “drainage marker” that may be associated not only with an increased risk of kidney injury but also with greater severity of kidney injury.

FIGURE 4

Perioperative complications

U-shaped relationship between perioperative complications and the patient's volume status

the negative effects of renal reperfusion by activation of anti-inflammatory and inhibition of pro-inflammatory mediators. However, the research data are too sparse for a definite recommendation (e59). Several studies have been initiated to compare inhalational and intravenous anesthetics.

It used to be thought that statins reduce the incidence of AKI, but recently published studies do not support this assumption (e60, e61). The data are too sparse for a general recommendation to use statins for the prevention of AKI (e62).

An initially postulated nephroprotective effect of sodium bicarbonate (e63) by alkalization of the urine was not confirmed in subsequent randomized controlled trials (e64, e65).

Renal replacement therapy

The central questions in treating a patient with severe AKI are whether and when renal replacement therapy should be initiated. The current KDIGO guidelines recommend starting renal replacement therapy in the presence of life-threatening complications (absolute

Renal replacement therapy

The current KDIGO guidelines recommend starting renal replacement therapy in the presence of life-threatening complications (absolute indication), including diuretic-resistant volume overload and significant metabolic/electrolyte disorders.

indication), including diuretic-resistant volume overload and significant metabolic/electrolyte disorders (e14). However, the majority of patients develop severe AKI without life-threatening complications, so the “whether and when” decision about renal replacement therapy lies in the hands of the intensivist or nephrologist. If the treatment is started too late, complications (e.g., volume overload) possibly associated with elevated mortality may already have set in; if too early, an invasive procedure may be carried out unnecessarily. The right time to begin renal replacement therapy in AKI patients with no uremic symptoms, electrolyte imbalance, or volume overload is a subject of heated debate—not least owing to the differing definitions of “early” and “late” initiation of treatment across studies (e66). For example, one randomized controlled trial found no difference between early and late renal replacement therapy with regard to 60-day mortality in patients with KDIGO stage 3 AKI. Forty-nine percent of the patients who received renal replacement therapy in the late group did not need it. There were more catheter infections in the early renal replacement therapy group. In contrast, it was shown that 90-day mortality was lower after early than after late commencement of renal replacement therapy in patients with KDIGO stage 2 AKI (e67). Further studies are in progress to supplement the as yet inconclusive data.

Conflict of interest statement

Prof. Zarbock has received lecture fees and reimbursement of travel and accommodation costs from Fresenius, Astute Medical, Bio Merieux, Braun, Baxter, AM Pharma, Ratiopharm, Amomed, and Edwards; payments for conducting commissioned clinical studies from Biotest, Fresenius, La Jolla Pharmaceuticals, Astute Medical, and Astellas; and funding for a research project of his own initiation from Baxter, Fresenius, Astute Medical, Astellas, and Bio Merieux.

Dr. Meersch has received lecture fees and reimbursement of travel and accommodation costs from Astute Medical.

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- **Supplementary material**
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CLINICAL SNAPSHOT

The Rare Occurrence of an Aneurysm of the Inferior Vena Cava

A 23-year-old man was referred with a right retroperitoneal mass and thrombosis of the inferior vena cava and the pelvic veins. The initial diagnostic imaging had shown a non-perfused right paracaval mass, 5.6 × 6 cm in diameter, below the inflow of the renal vein. Suspicion of lymphoma was expressed, with a differential diagnosis of sarcoma. Examination of a percutaneous biopsy sample revealed thrombotic material with a moderately cell-rich lymphoplasmacellular infiltrate. The findings of magnetic resonance imaging elsewhere had initially aroused the suspicion of an aneurysm of the inferior vena cava (Figure). According to the Gradman and Steinberg classification of 1993, an aneurysm at this location is categorized as type IV. Only 54 cases of aneurysm of the inferior vena cava were described up to the end of 2015. In the case described here, the aneurysm was found to have a narrow 1-cm connection and was completely resected without inferior vena cava replacement. Rivaroxaban was given for 6 months before recanalization of the partially thrombosed veins.

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A large right paracaval mass around 6 cm in diameter was revealed by axial reconstruction of the magnetic resonance imaging data (reproduced by kind permission of Prof. Dr. M. Lenz, Institute for Radiology, District Hospitals Reutlingen GmbH).

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Only one answer is possible per question. Please select the answer that is most appropriate.

Question 1

Which of the following statements applies most accurately to acute kidney injury (AKI)?

- a) According to current knowledge, AKI exerts no influence on patients' recovery.
- b) The occurrence of AKI leads to a pronounced increase in mortality.
- c) Owing to improved medical care, intensive care patients have only a slight risk of developing AKI.
- d) There is widespread high awareness of the magnitude and importance of AKI.
- e) AKI is not an independent risk factor.

Question 2

For which of these complications is acute kidney injury an independent risk factor?

- a) Oropharyngeal complications
- b) Orthostatic complications
- c) Cerebrovascular complications
- d) Gastrointestinal complications
- e) Pulmonary complications

Question 3

What proportion of intensive care patients develop acute kidney injury during their hospital stay?

- a) 1–37%
- b) 11–47%
- c) 22–57%
- d) 33–67%
- e) 44–77%

Question 4

What parameters are used to evaluate renal function?

- a) Erythrocyte sedimentation rate and protein–creatinine ratio
- b) Creatinine concentration and C-reactive protein
- c) Urine output and creatine phosphate metabolism
- d) Serum creatinine concentration and urine output
- e) Hematocrit level and spontaneous urine

Question 5

What is the advantage of using new injury markers?

- a) They can be used by the patients themselves.
- b) They yield information on renal damage.
- c) They color the urine green and thus enable identification of kidney injury at the bedside.
- d) They are more economical than conventional procedures.
- e) They help to predict wound healing and discharge from the hospital.

Question 6

Which of the following describes “subclinical AKI”?

- a) Acute kidney injury with no further clinical relevance
- b) A biomarker-positive status in the presence of a normal serum creatinine concentration
- c) Acute kidney injury with a moderately reduced serum creatinine concentration
- d) A serum creatinine–positive but biomarker-negative status
- e) Oliguria

Question 7

What factors play a key role in the development of AKI?

- a) Renal oxygen shortage and inflammation
- b) Hypertension and decreased erythrocyte sedimentation rate
- c) Adrenal cortex insufficiency and elevated C-reactive protein
- d) Electrolyte disorders and eosinophilia
- e) Leukopenia and oxygen shortage

Question 8

Vasopressors have a protective effect against the development of acute kidney injury. What must be remembered when norepinephrine is used?

- a) It may not be used in acute hypotension.
- b) Chronic skin disease must be excluded before its use.
- c) Its use causes volume overload, which protects the kidney from being damaged by “running dry.”
- d) It increases global and medullary blood pressure and thus diuresis.
- e) It has no consequences for osmoregulation.

Question 9

What parameters have a decisive influence on serum creatinine concentration?

- a) Peripheral arterial occlusive disease and metabolic syndrome
- b) Antiarrhythmics and antidepressants
- c) Body fat mass and female sex
- d) Statins and acetylsalicylic acid
- e) Muscle mass and hemodilution

Question 10

How high is the mortality among patients who become dependent on dialysis during their hospital stay?

- a) 14–41%
- b) 24–51%
- c) 34–61%
- d) 44–71%
- e) 54–81%

► Participation is possible only via the Internet: cme.aerzteblatt.de

Supplementary material to:

Acute Kidney Injury

A Frequently Underestimated Problem in Perioperative Medicine

by Raphael Weiss, Melanie Meersch, Hermann-Joseph Pavenstädt, and Alexander Zarbock

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