Int Urol Nephrol (2017) 49:461–466
DOI 10.1007/s11255-016-1451-4

NEPHROLOGY – REVIEW

Obesity, acute kidney injury and outcome of critical illness

Helmut Schiffl[1,3] · **Susanne M. Lang**[2]

Received: 20 June 2016 / Accepted: 28 October 2016 / Published online: 7 November 2016

Abstract Acute kidney injury is a heterogeneous clinical syndrome encompassing a spectrum of risk factors and acute insults, occurring in multiple settings and affecting both short-term and long-term outcomes. Obesity has become an epidemic. The available literature suggests that AKI is common in critically ill surgical or medical obese patients and that obesity is a novel risk factor for this acute renal syndrome. The pathophysiology of obesity-associated AKI is not completely understood. Obesity-related factors combined with the burden of other comorbidities in elderly obese patients may interact with known precipitating factors such as hypotension, nephrotoxins or sepsis and increase the susceptibility of this population to AKI. Whether or not obesity may counterintuitively be protective and associated with better survival of critically ill patients with AKI ("reverse epidemiology") is a subject for debate. Further investigations exploring the role of novel biomarkers and optimal management are needed urgently.

Keywords Obesity · Acute kidney injury · Critically illness · Outcome

✉ Helmut Schiffl
h-schiffl@t-online.de

[1] SRH University of Applied Sciences for Health, Gera, Germany

[2] Medizinische Klinik II, SRH Wald-Klinikum Gera, Gera, Germany

[3] Department of Internal Medicine IV, University Hospital Munich, University of Munich, Ziemssenstr. 1, 80336 Munich, Germany

Introduction

Abnormal or excessive body fat accumulation presents a serious health hazard with increasing incidence [1]. The WHO defines obesity as a BMI equal or greater than 30 kg/m^2. The WHO classification of severity of obesity (Table 1) [2] is the most widely used score in clinical trials. This is in part due to its relative simplicity. However, the validity of BMI as an ideal score of obesity is questionable, because it does not account for differences in body composition (i.e., nonfat solid mass, extra- and intracellular water mass, visceral vs. subcutaneous adipose mass), and the cutoff values of BMI used to define a patient as obese can be different for different ethnic populations. The waist-to-hip ratio, sagittal abdominal diameter and radiology imaging studies reflect body composition better, and they have been found to have stronger associations with clinical outcomes than BMI [1, 3, 4]. However, only a limited number of studies in the critical care setting have used these non-BMI measures.

Overweight and obesity pose major risk factors for fatal non-communicable diseases, including diabetes mellitus, cardiovascular disorders, some cancers and chronic kidney disease (CKD). These medical conditions go along with the compromised quality of life, lifelong disability, premature deaths and burgeoning health care costs. Once considered a problem only in high-income countries, overweight and obesity are now dramatically on the rise in low- and middle-income countries as well [1].

Obesity: a novel risk factor for acute kidney injury in critically ill patients

With the wide spread obesity "epidemic," intensive care units (ICU) have to treat an increasing number of obese critically ill patients. Today, 20–30% of the ICU patients

Table 1 WHO classification of overweight and obesity by BMI

Body mass index	Classification	Old definition
18.5–24.9	Normal weight	
25.0–29.9	Overweight	
30.0–34.9	Class I obesity	
35.0–39.9	Class II obesity	Severe obesity
40–49.9	Class III obesity	Morbid obesity

fall into the category "obese" and 7% into the category "morbidly obese" [5–8].

In spite of the increasingly recognized fact that obese patients are more likely to suffer from AKI, the incidence of AKI in critically ill obese patients is largely unknown. There is a consistent signal that the incidence of ICU AKI increases with BMI.

Recently, a secondary analysis of a single-center cohort including 15,470 critically ill patients found that AKI incidence rates ranged from 18.6% in normal weight, to 20.6% in overweight, 22.5% in class I obesity, 24.3% in class II obesity and 24.0% in class III obesity. In this analysis, each increase in BMI by 5 kg/m^2 was associated with a 10% risk for more severe AKI [9]. In another analysis of 445 patients undergoing cardiac surgery, each increase in BMI by 5 kg/m^2 was associated with a 26.5% risk for AKI [10]. In patients with ARDS, the incidence of AKI is even higher. The retrospective study of a cohort of 751 patients with ARDS found AKI (defined and graded by the RIFLE classification system) in 61.9%. The incidence increased significantly with increasing BMI categories [11]. A multicenter analysis of 53 institutions in Austria assessed the impact of BMI on the development of AKI in ICU patients. The risk of AKI necessitating renal replacement therapy showed a U-shaped distribution with the lowest risk (5.4%) in patients with normal BMI and the highest (11.8%) in the morbidly obese group [12].

A higher incidence of AKI in obesity has been described in surgical patients undergoing orthopedic [13, 14], cardiac [10, 13, 15–18], bariatric [19–24] or trauma surgery [25] or in medical patients with ARDS [11] or pancreatitis. The incidence of AKI following bariatric surgery varied between 2.8 and 17.5% depending on the definition of AKI, type of surgery and the population studied. It is noteworthy that the reported rates of AKI in morbidly obese patients are significantly higher in elective bariatric surgery than in normal weight patients undergoing elective orthopedic surgery [26].

Obesity not only is associated with a higher incidence of AKI, but has been identified as an independent risk factor for AKI in trauma [25], ARDS [11], general ICU populations [12], cardiac [10], gastrointestinal [13] and bariatric surgery [20]. These studies controlled for confounders (diabetes, hypertension, chronic kidney disease, blood product transfusion), leading to the hypothesis that additional obesity-related factors may increase risk for AKI in critically ill patients.

Obesity-related AKI: proposed mechanisms

The exact mechanisms associating obesity and AKI have not been fully understood [27], and pathophysiology of obesity-associated AKI may be multifactorial. A complex interplay of different mechanisms may increase the surgical or medical patient's susceptibility to obesity-associated AKI (Fig. 1). Among them are obesity-induced risk factors such as glomerulopathy, low-grade inflammation, endothelial dysfunction, augmented oxidative stress, activation of the renin–angiotensin–aldosterone system and increased sympathetic nervous system activity. Furthermore obesity-associated risk factors can be identified (metabolic syndrome, hypertension, cardiovascular disease). As in any other patient group, hypotension, sepsis and nephrotoxins may precipitate AKI in obese critically ill patients.

The specific hemodynamic and structural changes in the kidneys of obese patients are collectively called obesity-related glomerulopathy [28]. The early hemodynamic changes in kidney function are characterized by an increase in glomerular filtration rate and effective renal plasma flow, accompanied by increments in filtration fraction and albumin excretion. These hemodynamic changes are associated with glomerulomegaly, mesangial expansion and podocyte injury. The major pathophysiologic mechanisms of the effects of obesity on renal hemodynamics are afferent arteriolar vasoconstriction as well as an added role of efferent arteriolar vasoconstriction due to the stimulation of the renin–angiotensin–aldosterone system (RAAS). The long-term prognosis of obesity-related glomerulopathy is poor. Obesity related glomerulopathy is a distinct form of secundary focal segmental glomerulosclerosis. The hemodynamic effects of overweight on kidney function and albuminuria are magnified in the presence of hypertension or diabetes [28].

Obese patients undergoing surgery present a unique extra-renal comorbidity profile which includes a high prevalence of diabetes, hypertension, hyperlipidemia, and osteoarthritis; thus, those patients are commonly prescribed drugs which include angiotensin-converting-enzyme inhibitors (ACE-I), diuretics and nonsteroidal anti-inflammatory agents.

In a retrospective analysis of 351,000 patients from the American College of Surgeon National Surgical Quality Improvement database, Glance and colleagues [29] showed a two- to threefold increased risk of postoperative AKI in the obese population and shed light on the perioperative comorbid status as a determinant of postsurgery AKI. The

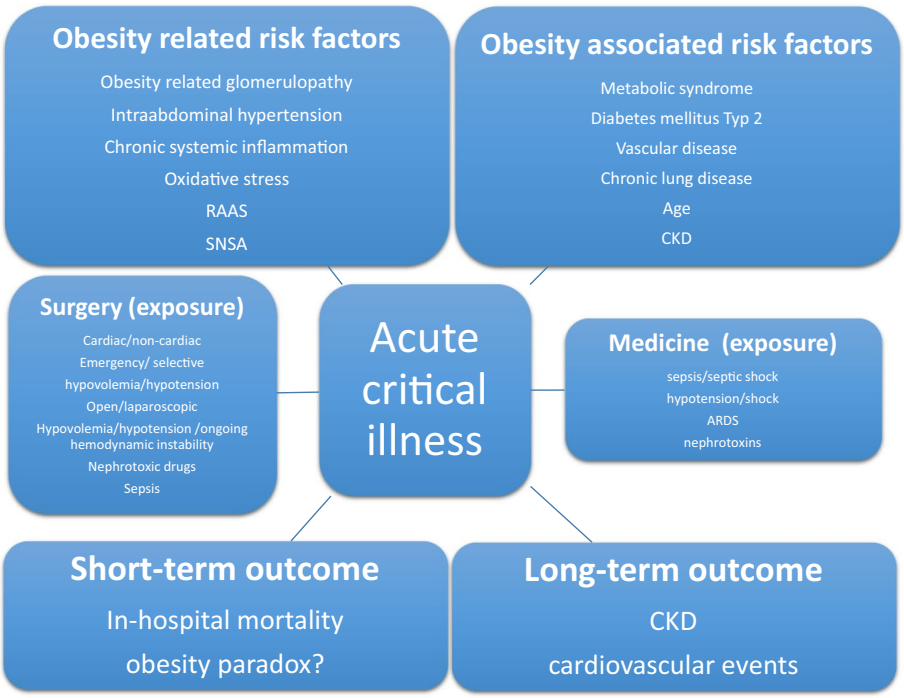

Fig. 1 Pathophysiology of obesity related AKI in critically ill patients

risk was almost seven times higher compared to normal weight individuals if the patient had metabolic syndrome.

Excess abdominal fat increases abdominal pressure, and obese ICU patients are at an increased risk for intra-abdominal hypertension, which may cause renal dysfunction from both renal venous congestion and poor arterial perfusion. However, it is not clear whether intra-abdominal hypertension in the setting of critical illness will have the same renal damaging effects in obese as non-obese patients [4].

Finally, the adipose tissue is not inert. Obesity is associated with increased levels of pro-inflammatory cytokines [30], endothelial dysfunction [31] and oxidative stress [10], as well as with increased activity of the sympathetic nervous system, and stimulation of the renin–angiotensin–aldosterone system [32]. The exact relationship between these obesity-induced factors and postoperative AKI has not been completely understood [27]. A recent study by Billings et al. [10] found that markers of oxidative stress are stronger predictors of postoperative AKI than inflammatory markers in obese patients undergoing cardiac surgery.

Acute kidney injury in critically ill, obese patients: the diagnostic challenge

Current definitions and staging systems of AKI (RIFLE, AKIN or KDIGO) are based on changes in serum creatinine and reductions in urine output as surrogates of the decline of glomerular filtration rate (GFR). The AKIN and KDIGO criteria avoid using GFR as a marker in AKI as there is no dependable way to measure GFR. Furthermore, estimated GFR rates are unreliable when used in the non-steady state conditions found in AKI. The limitations of current consensus criteria for AKI should be kept in mind when interpreting renal function in obese patients [33].

The use of a weight-adjusted urine output as threshold for diagnosing AKI makes sense, but can lead to over diagnosis (false-positive diagnosis) of AKI in obese patients. Therefore, the European Renal Best Practice (ERBP) statement suggests “ideal” body weight, i.e., age, length and gender-normalized weight [34].

The use of serum creatinine defined AKI may be more valid in the obese population. Studies have demonstrated an association of raised serum creatinine levels with increased mortality among obese patients with ARDS [11]. However, single-point measurements of serum creatinine have important limitations in critically ill, morbidly obese patients for several reasons: (1) All serum creatinine-based definitions require the measurement of “baseline” pre-insult renal function. Ideally, this value reflects the patient’s steady state kidney function just before the renal insult. However, information on baseline kidney function is not always available. At present, there is no recognized standard to determine baseline renal function. (2) Obese patients have higher serum creatinine concentrations due to higher creatinine generation rates compared to non-obese controls with comparable GFR. (3) Renal excretion rates of creatinine in obese patients are higher due to hyperfiltration and enhanced tubular creatinine secretion. (4) Creatinine-based criteria for AKI do not take into account renal functional reserve, as serum creatinine increases only after 50% of the nephrons have been lost

[35]. (5) The diagnosis of AKI may be delayed or missed in patients with significant fluid volume overload. (6) The serum creatinine concentration may take 24–36 h to rise after a definite renal insult. (7) Serum creatinine concentrations do not provide any information about the specific etiology of AKI. Taken together, serum creatinine-based criteria may delay or miss the diagnosis of AKI in critically ill obese patients with glomerular hyperfiltration.

Estimation of pre-insult or post-AKI GFR with equations based on serum creatinine concentrations and demographic and/or anthropometric data is subject to systemic bias when adjustments for body composition are not made for the obese population. These methods of measured or estimated GFR were established in lean individuals. The true creatinine clearance and Cockcroft–Gault formula may grossly overestimate GFR when measured body weight is used as parameter. They should not be used in obese individuals. The MDRD and CKD-EPI equations do not include total weight and appear to be more dependable in obesity [32, 33, 36].

It is unknown whether novel biomarkers for AKI combined with traditional markers of renal function (creatinine) allow the detection of subtle changes in tubular damage before rises of serum creatinine. There are limited data on the performance of the novel biomarkers in obese patients with AKI. Urinary neutrophil gelatinase-associated lipocalin (NGAL) was evaluated as a predictive marker of AKI in clinically severe obese patients who underwent bariatric surgery. Two out of twenty-three patients developed AKI during the immediate postoperative period. In both patients, urinary NGAL excretion increased before the values of serum creatinine increased and led to an earlier diagnosis of AKI [37].

Outcomes of critically ill patients and obesity-associated perioperative AKI

Postoperative AKI is common and potentially lethal in patients with a high burden of comorbidities. The sequelae of postoperative AKI are characterized by an increase in in-hospital morbidity and mortality, prolonged hospital stay, incident chronic kidney disease or accelerated progression of preexistent CKD to end-stage renal disease [38]. Survivors, particularly with CKD and its associated cardiovascular disorders often have a reduced quality of life and consume substantially greater health care resources than the general population. This is mainly due to rehospitalizations and unplanned intensive care unit admissions [38].

Current literature investigating the effects of obesity-related AKI on ICU mortality or long-term survival is conflicting. The secondary analysis of 5232 patients with AKI requiring RRT from 53 Austrian ICUs indicated an apparently contradictory pattern of perioperative outcomes in obese ICU patients, now known as the obesity paradox. Risk-adjusted hospital mortality followed a U-shaped pattern, with the highest mortality in underweight and morbidly obese patients and with the lowest mortality in obese patients (BMI $\geq$ 30). The authors concluded that obese patients seem to have a substantial survival benefit compared to underweight or normal weight patients [12]. Moreover, the retrospective study of patients with ARDS showed that the proportion of ARDS patients with AKI who died decreased as BMI increased [11].

However, there has been no satisfactory or clear explanation for this observation in obese ICU patients with AKI. Generally, the obesity paradox has been criticized on the grounds of being an artifact arising from biases in observational studies (BMI, traditional markers of AKI definition and grading, or other confounders) [39]. Another concern is the reverse causation due to illness-induced weight loss. That is, it may not be low BMI that is increasing the risk of mortality (and thereby indicating that obesity is associated with a lower risk of death), but rather unintentional weight loss before death in sicker patients being a risk factor for mortality. Classifying those individuals as lean greatly inflates the mortality rate in the normal and underweight categories of BMI, while lowering the risk in the higher BMI categories.

Recently an analysis by Danziger et al. [9] found that in-hospital and 1-year survival rates associated with an episode of AKI in medical and surgical ICU patients were similar across all body mass index categories. The retrospective cohort study by Tolpin et al. [40] on 10,863 patients undergoing primary coronary artery grafting surgery with cardiopulmonary bypass reported that obese patients with preoperative renal insufficiency had higher rates of postoperative myocardial infarction or low cardiac output syndrome as well as increased hospital stays than non-obese patients with preoperative renal insufficiency. In contrast, obese patients with normal preoperative renal function were associated only with an increased risk of postoperative wound infections. The observational Australian multicenter study reported that AKI in bariatric surgery patients requiring critical care support led to a prolonged hospitalization and was associated with higher mortality [41].

Currently, the balance of limited evidence does not point to a higher or lower mortality in patients with obesity-associated AKI. The discrepancy in reported data may be explained by differences in the study design or patient characteristics. In addition, there is no “standard” obese patient, as obese patients differ in BMI and burden of co-morbidities that may influence outcomes. This was evidenced by Glance et al. [29] who identified a subpopulation of obese patients with metabolic syndrome that had a dramatically higher risk of complications after non-cardiac surgery. In particular, obese patients with metabolic syndrome experienced a two- to threefold higher risk of cardiovascular

events, a 1.5- to 2. 5-fold higher risk for pulmonary complications, a twofold higher risk for neurologic complications, a three- to sevenfold higher risk of AKI and twofold higher risk of death. Therefore, in balance, patients' BMI may be one of the many factors that could affect outcomes in critically ill patients with AKI.

Prevention of AKI in obese surgical or medical patients

Recognition of obesity as a novel risk factor for AKI and heightened awareness of the increased susceptibility of patients with AKI should result in early and concrete changes in patient care. The early recognition of renal failure by a combination of novel biomarkers and conventional serum markers may help to prevent a more severe course of AKI. However, this remains speculative as there are only limited data on the performance of the novel biomarkers in obese patients with AKI. Attention to mitigating perioperative risks by minimizing exposure to nephrotoxins, optimization of the hemodynamic correction of fluid depletion without causing fluid overload and frequent re-assessment of the response to these interventions may help in reducing the risks of AKI in obese patients.

Resuscitation of obese patients with hemodynamic instability poses additional challenges in the absence of measurements of actual body weight. Prescription of essential drugs on formula-based weight adjustment or non-weight-based dosing can lead both to under- or overdosing.

Conclusions

Obesity is associated with an increased prevalence of AKI in a variety of medical and surgical patient groups, and obesity is an independent risk factor for AKI in patients admitted to intensive care, critically ill patients after surgery and those with ARDS. The complex pathophysiology of postoperative AKI and obesity has not been fully understood, but may involve obesity-related risk factors, patient-associated extra-renal and renal comorbidity and intraoperative factors. Whether or not obese patients with AKI have a lower risk of dying during critical illness compared with normal or underweight individuals remains unclear and preventing strategies to mitigate the higher risk are speculative and yet to be tested.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants performed by any of the authors.

References

1. Apovian CM (2013) The clinical and economic consequences of obesity. Am J Manag Care 19:s219–s228
2. WHO (2000) Obesity: preventing and aaging the global epidemic. Report of a WHO consultation. WHO Technical Report Series: 498
3. Suneja M, Kumar AB (2014) Obesity and perioperative acute kidney injury: a focused review. J Crit Care 29:694.e1–694.e6
4. Shashaty MG, Stapleton RD (2014) Physiological and management implications of obesity in critical illness. Ann Am Thorac Soc 11:1286–1297
5. Goh R, Darvall J, Wynne R, Tatoulis J (2016) Obesity prevalence and associated outcomes in cardiothoracic patients: a single-centre experience. Anaesth Intensive Care 44:77–84
6. Akinnusi ME, Pineda LA, El Solh AA (2008) Effect of obesity on intensive care morbidity and mortality: a meta-analysis. Crit Care Med 36:151–158
7. De Jong A, Molinari N, Pouzeratte Y, Verzilli D, Chanques G, Jung B et al (2015) Difficult intubation in obese patients: incidence, risk factors, and complications in the operating theatre and in intensive care units. Br J Anaesth 114:297–306
8. Robinson MK, Mogensen KM, Casey JD, McKane CK, Moromizato T, Rawn JD et al (2015) The relationship among obesity, nutritional status, and mortality in the critically ill. Crit Care Med 43:87–100
9. Danziger J, Chen KP, Lee J, Feng M, Mark RG, Celi LA et al (2016) Obesity, acute kidney injury, and mortality in critical illness. Crit Care Med 44:328–334
10. Billings FTT, Pretorius M, Schildcrout JS, Mercaldo ND, Byrne JG, Ikizler TA et al (2012) Obesity and oxidative stress predict AKI after cardiac surgery. J Am Soc Nephrol 23:1221–1228
11. Soto GJ, Frank AJ, Christiani DC, Gong MN (2012) Body mass index and acute kidney injury in the acute respiratory distress syndrome. Crit Care Med 40:2601–2608
12. Druml W, Metnitz B, Schaden E, Bauer P, Metnitz PG (2010) Impact of body mass on incidence and prognosis of acute kidney injury requiring renal replacement therapy. Intensive Care Med 36:1221–1228
13. Kelz RR, Reinke CE, Zubizarreta JR, Wang M, Saynisch P, Even-Shoshan O et al (2013) Acute kidney injury, renal function, and the elderly obese surgical patient: a matched case-control study. Ann Surg 258:359–363
14. Ward DT, Metz LN, Horst PK, Kim HT, Kuo AC (2015) Complications of morbid obesity in total joint arthroplasty: risk stratification based on bmi. J Arthroplast 30:42–46
15. Yap CH, Mohajeri M, Yii M (2007) Obesity and early complications after cardiac surgery. Med J Aust 186:350–354
16. Kumar AB, Bridget Zimmerman M, Suneja M (2014) Obesity and post-cardiopulmonary bypass-associated acute kidney injury: a single-center retrospective analysis. J Cardiothorac Vasc Anesth 28:551–556
17. O'Sullivan KE, Byrne JS, Hudson A, Murphy AM, Sadlier DM, Hurley JP (2015) The effect of obesity on acute kidney injury after cardiac surgery. J Thorac Cardiovasc Surg 150:1622–1628
18. Tekeli Kunt A, Parlar H, Findik O, Duzyol C, Baris O, Balci C (2016) The influence of metabolic syndrome on acute kidney injury occurrence after coronary artery bypass grafting. Heart Surg Forum 19:E099–E103

19. Weingarten TN, Gurrieri C, McCaffrey JM, Ricter SJ, Hilgeman ML, Schroeder DR et al (2013) Acute kidney injury following bariatric surgery. Obes Surg 23:64–70
20. Abdullah HR, Tan TP, Vaez M, Deb C, Farag N, Jackson TD et al (2016) Predictors of perioperative acute kidney injury in obese patients undergoing laparoscopic bariatric surgery: a single-centre retrospective cohort study. Obes Surg 26:1493–1499
21. Sharma SK, McCauley J, Cottam D, Mattar SG, Holover S, Dallal R et al (2006) Acute changes in renal function after laparoscopic gastric surgery for morbid obesity. Surg Obes Relat Dis 2:389–392
22. McCullough PA, Gallagher MJ, Dejong AT, Sandberg KR, Trivax JE, Alexander D et al (2006) Cardiorespiratory fitness and short-term complications after bariatric surgery. Chest 130:517–525
23. Morgan DJ, Ho KM, Armstrong J, Baker S (2015) Incidence and risk factors for intensive care unit admission after bariatric surgery: a multicentre population-based cohort study. Br J Anaesth 115:873–882
24. Thakar CV, Kharat V, Blanck S, Leonard AC (2007) Acute kidney injury after gastric bypass surgery. Clin J Am Soc Nephrol 2:426–430
25. Shashaty MG, Meyer NJ, Localio AR, Gallop R, Bellamy SL, Holena DN et al (2012) African American race, obesity, and blood product transfusion are risk factors for acute kidney injury in critically ill trauma patients. J Crit Care 27:496–504
26. Perregaard H, Damholt MB, Solgaard S, Petersen MB (2016) Renal function after elective total hip replacement. Acta Orthop 87:235–238
27. Varrier M, Ostermann M (2014) Novel risk factors for acute kidney injury. Curr Opin Nephrol Hypertens 23:560–569
28. Eknoyan G (2011) Obesity and chronic kidney disease. Nefrologia 31:397–403
29. Glance LG, Wissler R, Mukamel DB, Li Y, Diachun CA, Salloum R et al (2010) Perioperative outcomes among patients with the modified metabolic syndrome who are undergoing noncardiac surgery. Anesthesiology 113:859–872
30. Alexopoulos N, Katritsis D, Raggi P (2014) Visceral adipose tissue as a source of inflammation and promoter of atherosclerosis. Atherosclerosis 233:104–112
31. Romero-Corral A, Sert-Kuniyoshi FH, Sierra-Johnson J, Orban M, Gami A, Davison D et al (2010) Modest visceral fat gain causes endothelial dysfunction in healthy humans. J Am Coll Cardiol 56:662–666
32. Kwakernaak AJ, Toering TJ, Navis G (2013) Body mass index and body fat distribution as renal risk factors: a focus on the role of renal haemodynamics. Nephrol Dial Transplant 28(Suppl 4):iv42–iv49
33. Bucaloiu ID, Perkins RM, DiFilippo W, Yahya T, Norfolk E (2010) Acute kidney injury in the critically ill, morbidly obese patient: diagnostic and therapeutic challenges in a unique patient population. Crit Care Clin 26:607–624
34. Ad hoc working group of E, Fliser D, Laville M, Covic A, Fouque D, Vanholder R et al (2012) A european renal best practice (ERBP) position statement on the kidney disease improving global outcomes (KDIGO) clinical practice guidelines on acute kidney injury: part 1: definitions, conservative management and contrast-induced nephropathy. Nephrol Dial Transplant 27:4263–4272
35. Sharma A, Mucino MJ, Ronco C (2014) Renal functional reserve and renal recovery after acute kidney injury. Nephron Clin Pract 127:94–100
36. Lemoine S, Guebre-Egziabher F, Sens F, Nguyen-Tu MS, Juillard L, Dubourg L et al (2014) Accuracy of GFR estimation in obese patients. Clin J Am Soc Nephrol 9:720–727
37. Koukoulaki M, Spyropoulos C, Hondrogiannis P, Papachristou E, Mitsi E, Kalfarentzos F et al (2013) Neutrophil gelatinase-associated lipocalin as a biomarker of acute kidney injury in patients with morbid obesity who underwent bariatric surgery. Nephron Extra 3:101–105
38. Schiffl H, Lang SM, Fischer R (2012) Long-term outcome of survivors of ICU acute kidney injury requiring renal replacement therapy: a ten year prospective cohort study. Clin Kidney J 5:297–302
39. Robert R, Frat JP, Hauet T (2011) Obesity and acute kidney injury: fact or artifact? Intensive Care Med 37:164 **(author reply 165)**
40. Tolpin DA, Collard CD, Lee VV, Elayda MA, Pan W (2009) Obesity is associated with increased morbidity after coronary artery bypass graft surgery in patients with renal insufficiency. J Thorac Cardiovasc Surg 138:873–879
41. Morgan DJ, Ho KM (2015) Acute kidney injury in bariatric surgery patients requiring intensive care admission: a state-wide, multicenter, cohort study. Surg Obes Relat Dis 11:1300–1306