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Management of acute kidney injury in gastrointestinal tumor: An overview

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Abstract

Gastrointestinal tumors remain a global health problem. Acute kidney injury (AKI) is a common complication during the treatment of gastrointestinal tumors. AKI can cause a decrease in the remission rate and an increase in mortality. In this review, we analyzed the causes and risk factors for AKI in gastrointestinal tumor patients. The possible mechanisms of AKI were divided into three groups: pretreatment, intrafraction and post-treatment causes. Treatment and prevention measures were proposed according to various factors to provide guidance to clinicians and oncologists that can reduce the incidence of AKI and improve the quality of life and survival rate of gastrointestinal tumor patients.

Key Words: Gastrointestinal tumor; Acute kidney injury; Risk factors; Treatment; Preventive measures; Enhanced recovery pathways

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Core tip: This review analyzed the causes and risk factors for acute kidney injury (AKI) in gastrointestinal tumor patients, and possible mechanism of AKI were divided into three groups: pretreatment, intrafraction and post-treatment causes. In response to these possible causes of AKI, treatment and preventive measures have been proposed based

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on the latest developments. This article intends to provide guidance of AKI during the treatment of gastrointestinal tumor patients to clinicians and oncologists.

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INTRODUCTION

In the past 10 years, with the advancement of medical technology and the vigorous development of tumor-related disciplines, the emergence of related new drugs, and the popularization of cancer screening, cancer patients' survival rate has increased significantly. The rates of related side effects and kidney involvement during tumor treatment have also increased significantly. Acute kidney injury (AKI) is a common complication in cancer patients that can decrease the remission rate, increase mortality, extend hospital stays, and increase costs[1,2]; furthermore, it is associated with poor long-term prognosis[3]. Gastrointestinal cancer is a global health problem with an estimated 3.4 million newly diagnosed cases worldwide in 2018[4]. According to a nationwide study in China, among 1418 cases of malignancy-related AKI (MR-AKI), gastrointestinal cancer was the most common malignancy (50.1%)[5], and the occurrence of MR-AKI increased hospitalization costs and length of stay and significantly increased the medical burden[6].

Therefore, in this review, we analyzed the causes and risk factors for AKI in gastrointestinal tumor patients to determine how to promptly diagnose and prevent AKI and provide guidance for nephrologists and oncologists.

EPIDEMIOLOGY

The incidence of tumor-related AKI is related to the nature, location and severity of the tumor; the presence or absence of complications; the course of the disease; the use of chemotherapy and targeted biological therapy; and the diagnostic criteria for AKI that are adopted[7,8]. The incidence in patients with tumor-related AKI is 7.5%–18.4%[5,9,10]. A population study in Denmark followed 37 267 new cancer patients from 1999 to 2006 and reported 1-year and 5-year risks of AKI of 17.5% and 27%, respectively[11]. In total, 5.1% of patients required renal replacement therapy (RRT) within 1 year of the occurrence of AKI. Jin *et al*[5] found that among patients with tumor-related AKI, gastrointestinal cancer (50.1%) was the most common malignancy. Approximately half of the patients (50.3%) were treated with RRT[5]. Li *et al*[12] concluded that the proportions of esophageal cancer, gastric cancer and bowel cancer patients with AKI were 20.5%, 13.9% and 12.5%, respectively[12]. A smaller study concluded that the incidences of postoperative AKI after gastric cancer and colorectal cancer (CRC) were 14.4%[13] and 11.8%[14], respectively. However, most articles concluded that AKI increased tumor patients' risk of death[11,13].

RISK FACTORS FOR TUMOR-RELATED AKI

The risk factors for AKI in patients with gastrointestinal tumors can be divided into pretreatment, intrafraction and post-treatment causes. Widely recognized risk factors for AKI in cancer patients include the use of nephrotoxic drugs, angiotensin-converting enzyme inhibitors (ACEIs), chemotherapeutics, antibiotics, and nonsteroidal anti-inflammatory drugs (NSAIDs)[15,16]. Risk factors also include age > 65 years[17], pre-existing chronic kidney disease (CKD), and comorbid diseases (such as diabetes and cardiovascular disease)[17,18], sepsis[16], contrast nephropathy[19], low blood volume, preoperative dehydration[20–22], low serum albumin levels[17,20], tumor size[23], anemia[20], heavy tumor burden or extensive metastasis, extensive surgery, surgical methods, intraoperative bleeding, operation time[17,24–28], and

urinary tract obstruction[29] (Table 1).

MECHANISMS OF AKI IN PATIENTS WITH GASTROINTESTINAL TUMORS

Drug-related AKI in gastrointestinal tumor patients

Chemotherapeutic drugs can affect the glomeruli, renal tubules, renal interstitial tissue, or the renal microvascular system. The clinical manifestations can range from the asymptomatic elevation of serum creatinine to acute renal failure (ARF).

Cytotoxic drugs

Chemotherapy for gastrointestinal tumors mainly included neoadjuvant chemotherapy, postoperative adjuvant chemotherapy, and palliative chemotherapy. Traditional treatments are mainly divided into cytotoxic drugs and targeted therapy. In recent years, the use of novel targeted anticancer agents has led to an overall improvement in the prognosis of many patients affected by various malignancies. In recent years, the effectiveness of newly developed drugs has been confirmed for different types of solid tumors, and the survival period has been prolonged. Nevertheless, the incidence of AKI in hospitalized cancer patients seems to be increasing because of aggressive cancer therapies[30]. In Japan, cohort studies have reported drug treatment as the reason for the onset of AKI in 14.4%–25.7% of adult patients[31,32]. Therefore, drug therapy as a cause of AKI in cancer patients cannot be ignored.

The standard drug treatment for gastric cancer is combination chemotherapy with S-1 (tegafur/gimeracil/oteracil), 5-fluorouracil (5-FU), capecitabine plus cisplatin or oxaliplatin as adjuvant chemotherapy for stage II/III disease. S-1 plus cisplatin is administered as the primary treatment for human epidermal growth factor receptor type 2 (HER2)-negative, advanced, recurrent gastric cancer[33]. The standard drug treatments for colon cancer include FOLFOX (oxaliplatin, fluorouracil and leucovorin), CAPOX (capecitabine and oxaliplatin), FOLFIRI (5-FU, folinic acid and irinotecan), *etc.*

Cisplatin, a platinum compound that is mainly eliminated by the kidneys, is a factor that can cause AKI that has been widely studied and verified[34–36]. Cisplatin mainly damages the S3 segment of the proximal tubule, resulting in a decrease in the glomerular filtration rate (GFR). Cisplatin is associated with many mechanisms involved in renal insufficiency. The exposure of renal tubular cells to cisplatin activates complex signaling pathways, leading to renal tubular cell damage and cell death in the proximal tubules. Cisplatin selectively damages proximal tubule cells (indicated by necrosis and apoptosis), and multiple signaling pathways contribute to cisplatin-induced injury and death of renal tubular cells[34,37]. In addition, cisplatin can increase the expression of proinflammatory cytokines [tumor necrosis factor (TNF)- α , interleukin-6, and interferon- γ] and promote the differentiation, maturation, and activation of neutrophils, T cells, and other components in the cellular inflammatory response[38,39]. The severity of AKI in mice with defective inflammatory pathways after exposure to cisplatin was relatively mild, illustrating the potential importance of these mediators[38–41]. Prominent inflammation and damage to the renal vascular system can cause vasoconstriction, decreased blood flow, and ischemic damage. These changes collectively lead to AKI[42]. Furthermore, there is a cisplatin-mediated decrease in the expression and function of sodium-dependent glucose and amino acid transporters[43], which increases the risk of AKI.

Oxaliplatin, however, carries a reduced risk of AKI compared to the previously described platinum agents, including cisplatin and carboplatin. Several cases of oxaliplatin-induced acute tubular necrosis (ATN) have been reported[44–46]; however, only one case has been histopathologically confirmed as acute tubulointerstitial nephritis (ATIN). Here, we present a biopsy-confirmed and dialysis-dependent ATIN case study that shows that oxaliplatin rarely causes acute interstitial nephritis (AIN) [47,48]. Similar to cisplatin, oxaliplatin localizes to the vascular basolateral membrane and is actively transported by human organic cation transporter 2 (OCT2), which mediates the uptake of the drug into the kidneys. Cisplatin is minimally excreted by multidrug and toxin extrusion protein (MATE) 1 on the brush border membrane after its transfer into the cell. However, oxaliplatin is strongly susceptible to cellular transport *via* MATE2-K on the brush border membrane. It is strongly suggested that its nephrotoxicity is weak due to low tissue accumulation in renal tubular epithelial cells [49–51]. However, nephrotoxicity can be considered the result of oxaliplatin in cases of repeated exposure[52]. Carboplatin is known for its low nephrotoxicity, but only case reports have confirmed that carboplatin can cause biopsy-proven AIN[51,53,54].

Table 1 Risk factors for acute kidney injury in gastrointestinal tumor patients

Pretreatment causes	Intrafraction causes	Post-treatment causes
Medications	Gastrointestinal tumor	ICU
Nephrotoxic drugs	Different tumor sizes	Leak of surgical anastomosis
ACEI drugs	Heavy tumor burden	Respiratory failure
Chemotherapeutics	Extensive metastasis	Mechanical ventilation
Antibiotics	Urinary tract obstruction	
NSAIDs		
Age	Surgery	Hypovolemia
Age > 65 yr	Extensive surgery	
	Surgical methods	
	Intraoperative bleeding	
	Operation time	
	IAH	
	Use of diuretics	
Pre-existing CKD	IV contrast	Hemodynamic instability
Comorbid diseases	Chemotherapy	Hypotension
Diabetes	Cytotoxic drugs	
Cardiovascular disease	Targeted therapy	
Hypertension	ICIs	
Sepsis	Tumor lysis syndrome	Sepsis
Volume depletion	Blood transfusion	Retropertitoneal fibrosis
Low blood volume		
Preoperative dehydration		
Gastrointestinal losses		

ACEI: Angiotensin-converting enzyme inhibitor; CKD: Chronic kidney disease; IAH: Intraperitoneal hypertension; ICIs: Immune checkpoint inhibitors; ICU: Intensive care unit; NSAIDs: Nonsteroidal anti-inflammatory drugs.

On the other hand, in addition to drug-related AKI, the side effects of chemotherapy drugs, such as nausea, vomiting, dehydration, and anorexia, can lead to prerenal AKI.

Targeted therapy

Targeted agents can improve the survival rate of tumor patients by targeting the molecular mechanisms underlying cancer growth. Recognition of the adverse renal effects of these agents is extremely important for patient care. At present, the targets for gastrointestinal cancer mainly include epidermal growth factor receptor (EGFR), HER-2, vascular endothelial growth factor (VEGF), VEGF receptor (VEGFR), mToc-MET and hepatocyte growth factor. The following is a brief overview of commonly used targeted drugs.

Anti-EGFR monoclonal antibody: Cetuximab is a human mouse chimeric IgG1 monoclonal antibody that specifically binds to the extracellular domain of EGFR and kills tumor cells through antibody-dependent cell cytotoxicity (ADCC). It is often used in combination with chemotherapy for metastatic CRC and can effectively improve the survival rate. Cetuximab can also have nephrotoxic effects, including AKI[55,56]. EGFR is mainly expressed in distal and collecting tubules and is involved in maintaining the integrity of renal tubules. EGFR activation can cause the growth and production of renal tubular epithelial cells during AKI. For patients who are prone to renal injury, anti-EGFR drug therapy may be a second source of AKI. However, the prescribing information from the United States does not provide related dose guidance.

Panitumumab is a humanized IgG2 monoclonal antibody against EGFR that can prevent the activation of autophosphorylation and receptor-associated kinase by binding to EGFR and can effectively improve the prognosis of patients with CRC. AKI was the most common renal adverse event reported in 100 patients treated with pertuzumab[55]. The dose of the drug does not appear to be different for patients with renal insufficiency. For patients with mild to moderate renal injury, there is no need to adjust the dose of pertuzumab, and the clearance of drugs in patients with severe renal injury [creatinine clearance (CrCL) < 30 mL/min] has not been studied.

Nimotuzumab is the first EGFR-targeted drug synthesized in China. It mainly inhibits tumor cell proliferation and angiogenesis through ADCC and complement-dependent cytotoxicity. There is no related AKI report for this drug.

EGFR tyrosine kinase inhibitors: Tyrosine kinase inhibitors (TKIs) can compete for the Mg-ATP binding site on the catalytic region of EGFR-TKIs, block signal transmission, inhibit the activation of mitogen-activated protein kinase, and promote apoptosis. The main drugs for this target are erlotinib and gefitinib. According to the results of relevant clinical studies, the use of gefitinib in patients with advanced esophageal cancer or gastroesophageal junction cancer that progressed after chemotherapy failed to improve overall survival, but the patients' self-reported results suggest that gefitinib may have some palliative benefits[57]. At present, only one study has confirmed that gefitinib causes AKI during treatment of lung cancer[58]. Further study of AKI caused by gefitinib in gastrointestinal cancer is necessary in the future.

Anti-HER-2 monoclonal antibody: Trastuzumab is a recombinant humanized anti-HER-2 IgG1 monoclonal antibody that specifically acts on the extracellular domain of HER-2, inhibiting the activation of HER-2 and the signaling pathway mediated by HER-2, thereby playing a role in gastrointestinal tumors. In recent years, there have been numerous reports of related renal adverse events, and the most frequently reported events include proteinuria and AKI[55]. The US prescribing information for trastuzumab includes dose adjustments for patients with renal impairment (including those undergoing dialysis). For patients with mild to moderate renal injury, there is no need to adjust the dose of pertuzumab, and the drug clearance of patients with severe renal injury (< 30 mL/min) has also not been studied. Trastuzumab emtansine (T-DM1) is a conjugate of trastuzumab and cytotoxic substances. Although there are no reports of T-DM1-related AKI, it is still necessary to pay attention to the possible side effects.

HER-2/TKI: Lapatinib is an oral EGFR/HER-2 dual receptor TKI that mainly inhibits the phosphorylation and activation of tumor cells by inhibiting the ATP-binding sites of HER-2 and EGFR. The FDA Adverse Event Reporting System (FAERS) report found that 48 cases of AKI were reported between 2011 and 2015[55]. The US prescribing information on lapatinib does not propose dose adjustments for patients with renal injury, but it may not be necessary to adjust the dose due to the drug's low renal clearance rate.

Afatinib is a potent and irreversible dual EGFR/HER-2 TKI. There are no reports of afatinib-related AKI.

VEGFR TKIs: Apatinib is a small-molecule VEGFR inhibitor that mainly acts on VEGF2. There is no report regarding AKI.

Regorafenib is a new oral multitarget phosphokinase inhibitor that has a strong inhibitory effect on VEGFR-2, platelet-derived growth factor receptor (PDGFR)- β , fibroblast growth factor receptor-1 and c-kit and thus exerts multiple antitumor effects. There are no reports about the relationship between regorafenib and AKI.

Sunitinib belongs to a class of selective multitarget TKIs that play an antitumor role by interacting with VEGF, PDGFR- β , c-kit, Flt-3 and ret. Sunitinib has been recommended for use in gastrointestinal stromal tumors (GISTs)[59]. It has been reported that sunitinib can cause acute and chronic interstitial nephropathy[60,61].

Imatinib is effective for GISTs and can reduce recurrence and improve overall survival[62]. The FAERS report found that 25 events of imatinib-related renal toxicity were AKI[55]. The US prescribing information on sunitinib concludes that there is no need to adjust the starting dose for patients with mild, moderate and severe kidney injury[63]. There are no reports of imatinib-related AKI.

Anti-VEGF and anti-VEGFR monoclonal antibodies: Anti-VEGF and anti-VEGFR monoclonal antibodies mainly include bevacizumab and ramucirumab, which prolong the survival time of patients with gastrointestinal tumors and delay recurrence[64,65]. The most common symptom of both drugs is asymptomatic proteinuria, and there are

no reports of drug-related AKI.

B-Raf inhibitors: Vemurafenib and dabrafenib are orally available small-molecule kinase inhibitors targeting mutations that activate B-Raf. B-Raf is a member of the Raf family of growth-signal transduction protein kinases. Mutations in B-Raf result in the constitutive activation of this signaling pathway, leading to uncontrolled cell growth [66]. B-Raf mutation is also present in CRC and is used in the treatment of metastatic CRC, although the therapeutic effect is not as good as in malignant melanoma [67]. In the phase II study of the vemurafenib trial, one of the 132 participants died from vemurafenib-induced AKI [68]. Hence, regular monitoring of kidney function is recommended during treatment.

Immune checkpoint inhibitors

Immune checkpoint inhibitors (ICIs) are a novel and promising anticancer therapy. This novel class of drugs includes humanized antibodies that inhibit downstream immunity pathways [including cytotoxic T-lymphocyte antigen 4 (CTLA-4) and programmed cell death 1 (PD-1) or its ligand (PD-L1)] with the objective of enhancing the antitumor immune response. PD-1 inhibitors such as nivolumab and pembrolizumab and PD-L1 inhibitors such as atezolizumab, avelumab and vedolizumab (durvalumab) have been approved for multiple indications. However, by increasing the immune system's activity, ICIs can cause inflammatory side effects; these are called immune-related adverse events (irAEs). Renal toxicities have been increasingly recognized as complications of ICIs.

In a pooled analysis of > 3000 patients treated with ICIs, the overall incidence of AKI was 2.2%. In contrast, the incidence of severe AKI was 0.6%, and the incidence of renal irAEs was 1.9% with nivolumab and 2.0% with ipilimumab monotherapy. AKI occurred more frequently in patients who received combination therapy with ipilimumab and nivolumab (4.9%) than in patients who received monotherapy with ipilimumab (2.0%), nivolumab (1.9%) or pembrolizumab (1.4%) [69]. A review study of a PD-1 inhibitor with > 10 000 patients found a total incidence of AKI of 2.2%. The pooled incidence of AKI with nivolumab treatment was 2.3%, and that with pembrolizumab was 2.0% [70]. However, there are few reported cases of kidney damage caused by PD1 in gastrointestinal tumors. One case of nivolumab-induced acute granulomatous tubulointerstitial nephritis (TIN) in a patient with gastric cancer has been reported [71].

ICIs can fight cancer tolerance through the physiological downregulation of immune responses [72,73]. The principal mechanism of ICIs is the blockade of immune checkpoints, including CTLA-4 on the surface of T cells and PD-1 and its receptor PD-L1, which reactivates quiescent T cells in the tumor microenvironment and enables them to resume their antitumor activity and ability to mediate tumor cell death. Treatment with ICIs produces many cytokines, and inflammatory factors can cause kidney tissue damage [74,75].

Studies have also shown that DNA mismatch repair-deficient (dMMR)/microsatellite instability-high (MSI-H) (dMMR/MSI-H) CRCs are associated with a higher mutational burden and that these patients benefit less from conventional chemotherapy and have shorter overall survival and dense immune cell infiltration [76]. PD1-blocking antibodies, pembrolizumab and nivolumab have shown efficacy in patients with dMMR-MSI-H metastatic CRC, which highlights the enormous therapeutic prospects [77].

Tumor lysis syndrome

Tumor lysis syndrome (TLS) is a tumor emergency caused when many tumor cells lyse, releasing large amounts of potassium, phosphorus and nucleic acid into the systemic circulation. TLS occurs mainly during chemotherapy in hematological tumors. TLS can also occur in gastrointestinal tumors [78-81]. The primary mechanism is nucleic acid catabolism to uric acid, which leads to hyperuricemia and significant uric acid excretion. The increase can lead to uric acid deposition in the renal tubules, which can also cause renal vasoconstriction, renal autoregulation damage, renal blood flow reduction, and inflammation, resulting in AKI [82]. Hyperphosphatemia and calcium phosphate deposition in the renal tubules can also cause AKI. This releases many intracellular substances (potassium, phosphorus and nucleic acid, which can be metabolized to produce uric acid) into the systemic circulation. The metabolic consequences include hyperkalemia, hyperphosphatemia, secondary hypocalcemia, hyperuricemia, and AKI [83].

SURGERY-RELATED AKI

Surgery is the most essential treatment for gastrointestinal tumors. Gastrectomy with lymph node dissection constitutes an essential component of multimodal treatment for resectable gastric cancer[84]. Surgical resection is the only treatment that can cure localized colon cancer. The goal of surgical resection of primary colon cancer is to remove the tumor, large vessel pedicles, and lymphatic drainage area of the affected colon[85]. Nevertheless, surgery is also obviously related to AKI. Studies report a prevalence of 3%–35% for postoperative AKI and 0.5%–25% for all-cause postoperative mortality within the first year in patients who have undergone major abdominal surgery[86]. Postoperative AKI occurred in 17.4%–20.3% of patients who underwent laparoscopic CRC resection[27,87]. Of 4718 patients who underwent gastric cancer surgery, 14.4% developed postoperative AKI[13]. Compared with patients without AKI, patients with AKI were associated with increased 8–30-d mortality and 31–90-d mortality[87]. In a Danish population-based study of mortality after emergency surgery for colon cancer, mortality during the first 30 d after surgery was increased in patients with decreased kidney function who were receiving RRT[88]. A small cohort study that included 288 medical records from elective rectal cancer surgeries found an in-hospital mortality rate of 18.2% in patients with AKI, whereas the in-hospital mortality rate of patients without AKI was 0.7%[89].

AKI is a multifactorial condition. For example, anesthetics have an impact on the occurrence of postoperative AKI. First, stable intraoperative hemodynamics (especially a mean arterial pressure > 55 mmHg)[90,91] and average blood volume can help maintain renal perfusion and reduce postoperative AKI. Second, inhaled anesthetics can temporarily and reversibly inhibit renal function, decreasing renal blood flow, GFR, urinary sodium excretion, and urine output. Possible mechanisms include the loss of renal self-regulation, decreased renal blood flow, neuroendocrine response and neurohumoral factors[92]. A preoperative nil-by-mouth regimen, perioperative blood and intravascular fluid loss, extravasation of fluid from the vascular compartment (third-space effect), insensible fluid losses may also contribute to the occurrence of AKI. AKI can also develop from complications such as sepsis or electrolyte derangement associated with ileus[87]. Major surgery introduces the risk of fluid depletion at several stages. Furthermore, hypotension can lead to dysfunctional intrarenal microcirculation due to patchy hypoperfusion areas in the kidney and potentially add to the risk of developing AKI[93–95].

The perioperative fluid management is also notably related to AKI. The association of perioperative fluid overload with worsening postoperative morbidity is well established[96,97]. In recent studies, oliguria was significantly associated with AKI. Several recent studies demonstrated that intraoperative oliguria was associated with postoperative AKI in patients undergoing major abdominal surgery. Studies have also reported that intraoperative oliguria is significantly associated with increased postoperative AKI[98–100]. The possible mechanism of oliguria is as follows: Many factors, including the overall hemodynamic status, sympathetic activity, and the effects of hormones such as aldosterone and antidiuretic hormone influence urine output. Renal hypoperfusion in perioperative settings can be caused by hypovolemia, systemic vasodilatation (due to anesthesia or inflammation), positive pressure ventilation, or low cardiac output[101]. However, some studies showed that additional intravenous fluids or diuretics did not protect against AKI in oliguric patients[102, 103]. Additionally, there have been several studies regarding intraoperative oliguria with AKI in patients undergoing abdominal surgery. This unresolved problem needs further research.

The choice of surgical method can also affect the occurrence of AKI. The use of the laparoscopic approach in treating CRC has been shown to promote recovery and reduce postoperative pain, the length of hospital stay, blood loss volumes, and complication rates[104,105]. Pneumoperitoneum, considered essential for adequate exposure in laparoscopic surgery, is associated with increased intraabdominal pressure (IAP) and its associated hormonal modifications[106]. Demyttenaere *et al*[107] reported decreased renal function and renal blood flow during pneumoperitoneum, which could be linked to AKI.

High IAP can be observed after abdominal surgery due to reduced abdominal compliance, fluid overload or capillary leakage[108]. When IAP remains elevated (> 12 mmHg) for a prolonged time, it can progress to intra-abdominal hypertension (IAH)[74], which is characterized by decreased renal arterial inflow and venous outflow and leads to AKI[108,109].

Systemic inflammation can be triggered by many factors, both intraoperatively and postoperatively. Sepsis, ischemic injury, trauma, and the surgery itself can all lead to inflammation. These triggers cause the release of proinflammatory cytokines and damage-associated molecular patterns that exert pleiotropic effects, leading to alterations in the renin-angiotensin-aldosterone system, microcirculation, and endothelial cell integrity. They also cause oxidative stress, initiate the apoptosis cascade, and alter coagulation pathways with the formation of microvascular thrombi. All of these effects lead to organ stress and, ultimately, organ injury[110,111]. All of these factors may be present in gastrointestinal tumor surgery.

Increasing evidence has demonstrated that intraoperative blood transfusions may contribute to organ injury in susceptible patients by promoting a proinflammatory state, exacerbating oxidative tissue stress, and activating leukocytes and the coagulation cascade, thus paradoxically impairing oxygen delivery[112,113]. The use of hydroxyethyl starch (HES) has been associated with AKI[114]. However, this association has not been demonstrated in the surgical setting, particularly after gastroenterological surgery[115].

CONTRAST-INDUCED AKI

There are few studies of postoperative AKI caused by contrast agents in gastrointestinal tumors, and only one study of gastric surgery patients revealed that the use of contrast agents was an independent predictor of postoperative AKI[13]. Nevertheless, a prospective study concluded that there was no association between preoperative intravenous contrast administered for computed tomography (CT) up to 7 d before surgery and postoperative AKI. The authors claimed that the risk of contrast-induced nephropathy should not be a reason for avoiding contrast-enhanced CT[19].

CANCER-RELATED REASONS

Gastrointestinal tumors, especially CRC, can also directly invade the kidneys or ureter, resulting in ureteral obstruction, and affected patients experience hydronephrosis, especially when the tumor is in the ascending colon or descending colon (that is, near the kidneys). When the tumor enlarges, it may directly invade the kidney. The tumor may cause lymph node metastasis, lymph node enlargement, fusion into a mass, ureteral invasion or kidney invasion.

PREVENTIVE MEASURES AND TREATMENTS FOR AKI IN GASTROINTESTINAL TUMOR PATIENTS

As mentioned earlier, there are many factors in the diagnosis and treatment of gastrointestinal tumors that can affect the occurrence and development of AKI alone or in combination and can even increase the risk of death. When treating AKI in gastrointestinal tumor patients, it is necessary to comprehensively evaluate the patient's general condition and identify all factors that may affect renal function, including cancer- and non-cancer-related factors. Compared with the general population, oncology patients require increase attention during AKI treatment. In a study of solid tumor patients admitted to intensive care units, AKI was chiefly related to sepsis (80%), hypovolemia (40%) and outflow tract obstruction (17%)[116]. When AKI is caused by postrenal factors, the obstruction should be relieved first, and a J-tube should be placed in a timely manner to ensure smooth drainage. Timely imaging examinations and disease management in patients with gastrointestinal tumors are important because these patients constitute a high-risk group for postrenal AKI[117]. If AKI is not due to a postrenal factor, maintaining adequate hydration is the most important intervention for prerenal AKI and ATN and is easily administered. As previously discussed, AKI can be caused by a variety of factors, such as insufficient volume (due to chemotherapy-related nausea, vomiting, and diarrhea) and/or drugs (such as diuretics). Hypercalcemia or the use of drugs that affect autoregulation of the kidneys (such as ACEIs/ARBs or NSAIDs) can further increase the risk and severity of prerenal AKI. Insufficient fluid management during the perioperative period, blood loss, and water loss caused by the surgical process can lead to insufficient renal

perfusion and induce the occurrence of AKI. Therefore, the key is to ensure hemodynamic stability.

Liquid management

Hypovolemia is a common cause of AKI during the perioperative period[20-22]; the body fluids are redistributed, the amount of extracellular fluid decreases, and the amount of fluid in the third space increases. Limiting the amount of fluid replacement and ensuring an appropriate amount of hydration can prevent the increase in cavity pressure and organ edema caused by fluid entering the interstitial space, thereby improving the patient's prognosis. Goal-directed fluid therapy (GDFT) has been implemented in the clinic. GDFT is defined as the use of timely monitoring during the perioperative period, the development of an individualized rehydration plan, and the management of patient hemodynamic parameters through volume adjustment and vasoactive drugs. To approach a normal physiological state, it is important to ensure sufficient cardiac output, meet the oxygen demands of the kidneys and other organs, and prevent organ failure. Brienza *et al*[118] conducted a meta-analysis that included 4220 perioperative patients, and the results showed that maintaining optimal hemodynamics during the perioperative period could reduce the risk of renal damage. Compared with traditional fluid rehydration methods, GDFT reduces the incidence of AKI during the perioperative period. Water overload, myocardial ischemia and excessive use of catecholamines can increase the risk of perioperative AKI, and avoiding excessive infusion and reducing catecholamine dosages can reduce this risk [118]. In addition, it should be noted that urine output is not an ideal target parameter for GDFT. A study of noncardiac surgery patients with normal essential renal function revealed no significant correlation between oliguria and AKI, but vasopressin and diuretics were related to ARF.

When a patient in a hypovolemic state develops oliguria without fluid therapy, long-lasting renal hypoperfusion may eventually develop into ARF[103]. Conversely, inappropriate diuretics may cause prerenal AKI; therefore, unless there is clear evidence of fluid overload, diuretics are not recommended during the perioperative period[119,120].

Compared with traditional fluid therapy, the intraoperative application of GDFT can reduce fluid usage and postoperative complications, including wound infection, intestinal obstruction, AKI, pulmonary edema and heart failure[121-123]; shorten the hospital stay[121,124]; maintain perioperative hemodynamic stability[125,126]; and reduce the level of circulating lactic acid[127]. Meta-analyses by Brienza *et al*[118] and Egal *et al*[128] also showed that compared with the traditional fluid group, the GDFT group had a decreased risk of postoperative AKI. There are no reliable kidney-targeted drugs that can prevent and reduce the occurrence of AKI[129]. Hence, maintaining renal perfusion is still the most important preventive measure for protecting renal function[100].

Jhanji *et al*[130] concluded that GDFT can optimize stroke volume, maintain the microcirculation blood flow rate and improve tissue oxygenation, which may be one of the mechanisms of improved prognosis[130]. GDFT can maintain the expected cardiac output so that the kidneys have sufficient blood supply and can reduce renal vasoconstriction[118]. It is generally believed that GDFT is a fluid management strategy that can be used to improve systemic blood perfusion, maintain normal renal perfusion and improve tissue oxygenation. Sufficient cardiac output also reduces the contraction of renal blood vessels, thereby reducing AKI. Liquid selection in goal-oriented fluid therapy that restricts the intake of chlorine-containing fluids can reduce the risk of AKI and improve mortality[131]. Therefore, a large amount of normal saline is not recommended during the perioperative period. As mentioned above, HES is related to AKI, and 6% HES should be used reasonably according to the indications. However, for patients at a high risk of AKI and those with preexisting renal insufficiency, the use of HES should be avoided[132]. In terms of the intraoperative GDFT strategy, crystalloid or colloidal fluid should be selected as the background fluid. A randomized controlled study of patients undergoing colon surgery showed that the use of crystalloid versus colloidal fluid in GDFT had little effect on postoperative complications[133].

Although there was no association between preoperative intravenous contrast administered for CT before surgery and postoperative AKI[19], because patients with hypovolemia are more likely to develop contrast-induced AKI (CI-AKI) than patients with euolemia, guidelines recommend adequate hydration, especially for patients with risk factors[134]. The author also believes that for patients with CKD, it is necessary to pay close attention to renal function during follow-up to prevent AKI.

Infections increase morbidity in AKI patients; therefore, it is also vital to identify related symptoms early so that therapy can be started appropriately. It is necessary to consider that treatment can lead to further kidney complications, including AKI, under certain circumstances. This change in kidney function can necessitate adjusting a patient's cancer care, including chemotherapy options, diagnostic evaluation options, and other types of supportive care[117].

In cases of sepsis-associated AKI, it is essential to optimize fluid therapy and withdraw nephrotoxic drugs. Additionally, the early initiation of RRT before the development of fluid overload may improve treatment outcomes[71].

Treatment and prevention with ICIs

For irAEs caused by PD-1/PD-L1, the use of glucocorticoids, TNF- α antagonists, mycophenolate mofetil, or other drugs for temporary immunosuppression can effectively address most of these issues. The most common irAE is ATN. Although the incidence of irAEs is very low, it is still worthy of attention. A study described 13 patients with ICI-induced AKI who underwent kidney biopsy. Among them, 12 patients had ATIN, 11 received glucocorticoid therapy and nine of them had improvement in renal function[69]. Although patients with thrombotic microangiopathies received glucocorticoid therapy, their condition did not improve. The American Society of Clinical Oncology guidelines summarize the management of nephrotoxicity[135]. Different treatments are given according to the increase in the blood creatinine level. Generally, the blood creatinine level is 2–3 times higher than the baseline value, and hormone therapy should be administered. Patients can be treated with renal replacement when necessary. The expression of PD-L1 on cancer cells is the premise of establishing therapy; if PD-L1 is not highly expressed on malignant cells, the use of PD-L1 should be avoided to prevent the occurrence of AKI[136].

Treatment and prevention with chemotherapy

Regardless of whether neoadjuvant chemotherapy, postoperative adjuvant chemotherapy, or late palliative chemotherapy is used, we should fully assess the risk factors for AKI before chemotherapy is initiated. The establishment of a diagnosis of drug nephrotoxicity may be challenging in oncology patients treated with numerous agents. In addition to their immediate toxic effects on the renal parenchyma, these agents can decrease renal functional reserve[137]. The medications most often associated with AIN are calcineurin inhibitors, antibiotics, proton pump inhibitors and herbal medications[138]. Steroid therapy is effective for the treatment of AIN caused by different types of medications[139].

For example, the simultaneous use of NSAIDs and nephrotoxic antibiotics should be avoided, and contrast-induced nephropathy should be considered and prevented during the assessment of tumor-related disease. At the same time, renal function should be fully evaluated before chemotherapy. If patients are diagnosed with CKD, an evaluation should be made to determine when the dose of chemotherapy drugs should be reduced or a different type of drug should be used. Additionally, the blood volume of tumor patients should be evaluated in a timely manner before medication to prevent the excessive use of diuretics, avoid insufficient blood volume, prevent infection and avoid sepsis. Once AKI occurs, suspicious drugs should be stopped, the etiology and mechanism of AKI should be analyzed and determined, and interventions for prerenal factors, such as supplementing capacity, correcting hypercalcemia, resolving hypercoagulability, and discontinuing ACEIs/ARBs, should be taken.

Treatment and prevention of TLS-related AKI: TLS-related AKI usually occurs approximately 24 h after chemotherapy. Prevention is critical, especially for tumor patients with a high tumor burden, a low introductory GFR, and sensitivity to chemotherapy drugs. Correcting electrolyte disturbances, reducing blood uric acid, and ensuring adequate hydration can reduce the risk of AKI, and high-risk patients can also be given intravenous fluids before chemotherapy[140]. Hydration can reduce blood uric acid, blood phosphorus, and blood potassium concentrations, increase renal blood flow, and keep electrolyte balance.

Treatment and prevention with chemotherapeutic drugs: Cisplatin is one of the most commonly used antitumor and nephrotoxic drugs. In patients with pre-existing renal impairment, the best method for cisplatin therapy is unknown. The US FDA has not approved any kidney-related dose adjustment guidelines. Studies suggest that patients with renal impairment should receive a reduced dose of cisplatin, but there is still a lack of data[141]. Clinical trial protocols usually require serum creatinine < 177

$\mu\text{mol/L}$ or $\text{CrCl} \geq 60 \text{ mL/min}$ for patients to receive a full dose of cisplatin. The manufacturer recommends that unless or until serum creatinine is $< 1.5 \text{ mg/100 mL}$ and/or blood urea nitrogen is $< 25 \text{ mg/100 mL}$, multiple courses of cisplatin should be given.

Carboplatin and oxaliplatin are safe for the kidneys. Studies included renal biopsies have shown that both carboplatin and oxaliplatin can cause not only ATN but also AIN[53,142]. Steroid treatment for AIN is controversial. In cases in which renal failure progresses even after drug discontinuation, steroid administration is recommended if the interstitial fibrosis area is 75% or less[143]. In cases in which it is unclear whether AIN is drug-induced, it is essential to discontinue the suspected drugs as soon as possible.

5-FU, capecitabine and irinotecan delivered *via* a nonrenal pathway rarely cause AKI. Therefore, patients with renal dysfunction do not need to receive an adjusted dose, but oral fluoropyrimidine capacity is contraindicated in patients with severe renal impairment ($\text{CrCl} < 30 \text{ mL/min}$). Once AKI is diagnosed, the dose should be reduced by 25%.

Gemcitabine-associated thrombotic microangiopathy (TMA) is believed to be rare, with an estimated incidence rate of 0.015%. TMA treatment includes the withdrawal of gemcitabine, antihypertensive therapy, plasma exchange and dialysis[144]. Related studies were unable to determine the role of hormones in TMA, and there is still no suitable preventive method[145].

VEGF pathway inhibitors (bevacizumab and aflibercept) are a class of antiangiogenic small-molecule TKIs (sunitinib, sorafenib, pazopanib, *etc.*), and although proteinuria is a common effect of all VEGF pathway-targeted drugs, the factors related to the occurrence and severity of proteinuria remain unclear. At present, there is no research finding indicating that VEGF pathway inhibitors induce AKI[48]. In summary, renal biopsy is always the gold standard for the diagnosis of drug-related AKI[146].

Prevention-related factors in perioperative AKI

IAH can cause a decrease in renal blood flow. However, for gastrointestinal tumor patients undergoing laparoscopic surgery, the duration of pneumoperitoneum during surgery should be kept as short as possible. Blood transfusions also play an essential role in AKI because they also cause hyperkalemia; therefore, unnecessary blood transfusions should be avoided in patients undergoing gastric surgery. In addition to regularly checking kidney function during the perioperative period, checking urine output every six hours is an excellent way to evaluate the occurrence of AKI.

Enhanced recovery pathways

The hallmark of enhanced recovery pathways (ERPs) is the bundled application of evidence-based perioperative interventions. While no two programs are identical, core components include establishing a patient safety climate, creating multidisciplinary teams, and providing comprehensive patient education, multimodal analgesia, minimally invasive surgical techniques, goal-directed fluid administration and optimized nutrition[147,148]. ERPs have gained widespread popularity across gastrointestinal surgical subspecialties as a means to hasten postoperative recovery [149,150]. ERPs resemble an overall plan; standardized ERP measures for colorectal surgery hospital patients include: (1) Patient education; (2) Avoidance of routine bowel preparation; (3) Precise fluid intake up to 2 h before surgery; (4) Administration of a preoperative oral carbohydrate solution (*i.e.*, PreOP, Nutricia; Numico, Zoetermeer); (5) Avoidance of preoperative sedatives; (6) Epidural analgesia for open surgery; (7) Protocolized fluid administration with intraoperative GDFT *via* noninvasive hemodynamic monitoring using Vigileo/FloTrac or esophageal Doppler (EDM Dexter Medical, Inc., Irvine, TX); (8) Avoidance of hypothermia; and (9) Avoidance of routine surgical drains[151]. During the treatment process, a multidisciplinary team proposed optimizing fluid therapy, avoiding fasting, using minimally invasive surgical methods and early feeding, and using specifically proposed interventions to solve preoperative hypoproteinemia, the most prominent of which is preoperative nutritional optimization. Nutritional optimization is the core component of an ERP with potential intervention points. Successful nutritional intervention before and after surgery is related to improved bowel recovery. Further studies are necessary to evaluate the feasibility and effectiveness of risk factor mitigation or the individualization of bundled therapies to reduce AKI in ERPs for colorectal surgery[151].

Continuous RRT

Despite these temporary measures, patients with gastrointestinal tumors may still develop AKI during treatment and require RRT. They may also develop other initial indications for continuous RRT (CRRT), such as metabolic acidosis, persistent oliguria or anuria, and hyperkalemia, which are difficult to correct.

The decision to discontinue RRT in patients with AKI is based on the following clinical scenarios: Intrinsic kidney function has adequately improved to meet demands, the disorder that prompted the need for renal support has improved, or CRRT is no longer consistent with the goals of care. There is no definitive prospective evidence to guide clinicians, but urine output appears to be predictive of successful RRT discontinuation. In one study of patients on CRRT, 24-h urine output > 400 mL/d in those not taking diuretics or > 2300 mL/d in those taking diuretics was associated with a > 80% chance of successful RRT discontinuation. Other studies have suggested that the quantitation of timed urinary creatinine and urea excretion may be helpful [152,153].

CONCLUSION

Tumor treatment has developed rapidly in the past two decades, and the survival durations of patients with gastrointestinal tumors have been prolonged. However, the prevalence of factors that cause AKI has also increased significantly. Therefore, nephrologists and oncologists need to pay attention to patients' renal function during each treatment. However, the author is pleased to see that research on goal-oriented therapy and ERPs has been published in recent years that may reduce the occurrence of AKI. Future research should focus on more on ERPs and on GDFT.

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