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Review Article

Research progress on the intrinsic link between the occurrence and progression of malignant tumors, chronic kidney disease and drug safety

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Abstract: There is a close intrinsic link between the occurrence, development, and medication use of malignant tumors and chronic kidney disease (CKD). Firstly, CKD provides conditions for the occurrence and progression of malignant tumors, such as the accumulation of uremic toxins, inflammatory responses, immune dysfunction, and metabolic disorders. Simultaneously, malignant tumors can damage the kidneys through paraneoplastic effect, direct tumor invasion, or compression. In addition, the treatment of malignant tumors can also trigger or worsen CKD. Therefore, improving early cancer screening for CKD patients and exploring the safety of medications used in CKD patients with concurrent cancer are of significant clinical importance. Based on this, this article reviews recent research on the interaction between CKD and malignant tumors and the safety of medications used, aiming to provide important theoretical basis for improving the diagnosis and treatment of CKD with concurrent cancer and improving the prognosis of these patients.

Keywords: Chronic kidney disease, malignant tumor, intrinsic link, cancer screening, medication safety

Introduction

Epidemiological surveys show that chronic kidney disease (CKD) and malignant tumors are two common disease types worldwide and leading causes of death. The incidence of both is continuously rising [1, 2]. Previously, scholars believed there was no significant potential correlation between the development of CKD and malignant tumors [3]. However, recent studies have shown an intrinsic link between CKD and malignant tumors. For example, the incidence of malignant tumors in CKD patients is much higher than in healthy individuals, and the incidence is even higher in patients undergoing dialysis or kidney transplantation [4, 5]. Simultaneously, studies have confirmed that the incidence of acute and chronic kidney injury and CKD is higher in patients with malignant tumors than in patients with other diseases [6]. The reasons are as follows: on the one hand, both groups share many common factors, such as advanced age, smoking, obesity, and CKD, which increase susceptibility to tumors; on the other hand, the treatment of malignant tumors can directly or indirectly lead to kidney damage [7, 8]. Therefore, exploring the correlation between CKD and malignant tumors is of great significance for improving the prognosis of these two groups of patients [9, 10].

Potential mechanism of CKD as an independent influencing factor of malignant tumors

Literature confirms that CKD, especially in patients with advanced stages or requiring dialysis, leads to severe impairment of the body's metabolic excretion function, resulting in internal environment disturbances (electrolyte imbalances, accumulation of nephrotoxic substances), accompanied by inflammatory responses and decreased immune defense function [11, 12]. Studies have confirmed that changes in the human body environment are a necessary prerequisite for the occurrence and development of malignant tumors [13]. See **Figure 1**.

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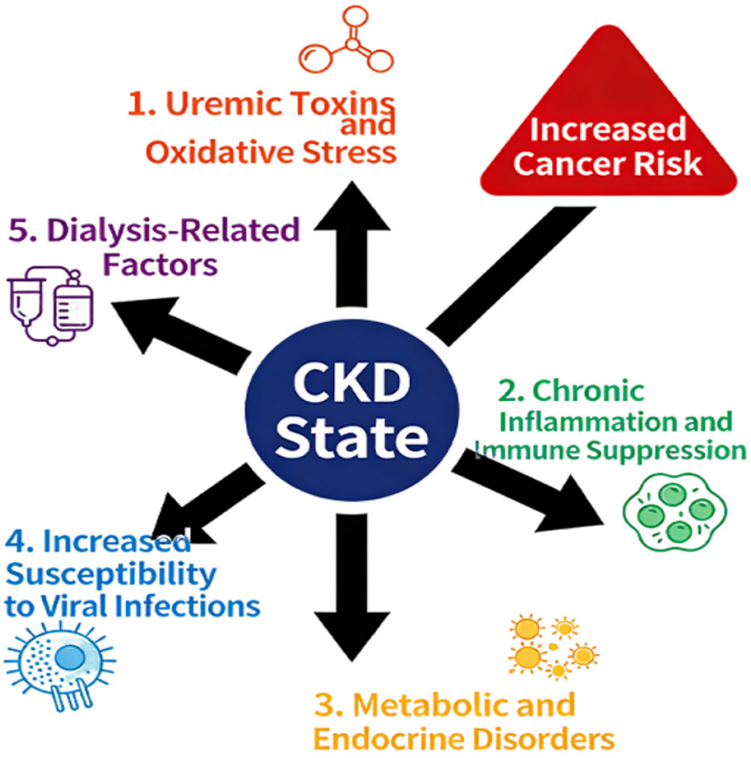

Figure 1. Mechanisms linking chronic kidney disease to increased cancer risk.

Accumulation of uremic toxins and tumor development

Research results indicate that impaired renal function promotes the accumulation of uremic toxins throughout the body. Among these compounds, some protein-binding molecules exhibit direct genotoxicity and mutagenicity [14]. Notably, in patients with CKD, the levels of intestinal metabolites such as indoxyl sulfate (IS) and p-cresyl sulfate (PCS) are significantly elevated [15, 16]. Extensive evidence confirms that IS and PCS induce endothelial dysfunction, exacerbate oxidative stress, and promote DNA damage. Crucially, both toxins can act as ligands for aryl hydrocarbon receptors (AhR). Upon activation, AhR translocates to the nucleus, forms a heterodimer with AhR nuclear translocator, and binds to xenobiotic response elements in the promoter regions of target genes. This upregulates a series of pro-proliferative and anti-apoptotic mediators at the transcriptional level, including cyclin D1, c-Myc, and matrix metalloproteinases, while inhibiting pro-apoptotic signals such as Bax. Therefore, activation of the AhR pathway drives uncontrolled cell cycle progression, enhanced invasiveness, and apoptosis resistance - key features in the pathogenesis of various malignant tumors [17, 18].

Meanwhile, excessive accumulation of advanced glycation end products (AGEs) has been proven to be a significant factor promoting tumorigenesis and development. The underlying mechanism involves AGEs binding to their specific receptors, triggering the recruitment of adaptor molecules and activation of classical signaling pathways, most notably the nuclear factor-κB (NF-κB) pathway. NF-κB activation leads to sustained transcriptional upregulation of pro-inflammatory cytokines (e.g., transforming growth factor-β (TNF-α), interleukin-1β, and interleukin-6 (IL-6)), chemokines, and pro-fibrotic factors such as TGF-β. This creates a chronic inflammatory and fibrotic tumor microenvironment, promoting cancer cell proliferation, angiogenesis, epithelial-mesenchymal transition, and metastasis [19, 20]. See **Figure 2**.

Therefore, the pathophysiological accumulation of uremic toxins is closely related to tumorigenesis through specific and well-defined molecular pathways. Therefore, taking proactive and effective measures to enhance the clearance of these toxins is a promising therapeutic strategy and a potential intervention target for reducing the cancer risk in this susceptible population [21].

Potential links between chronic inflammation, immune function, metabolic endocrine disorders and malignant tumors

It is well-established that patients with CKD exhibit a persistent, low-level state of systemic inflammation, characterized by long-term elevated levels of pro-inflammatory mediators such as, tumor necrosis factor-α, and C-reactive protein in circulation [22, 23]. This chronic inflammatory environment becomes a key driv-

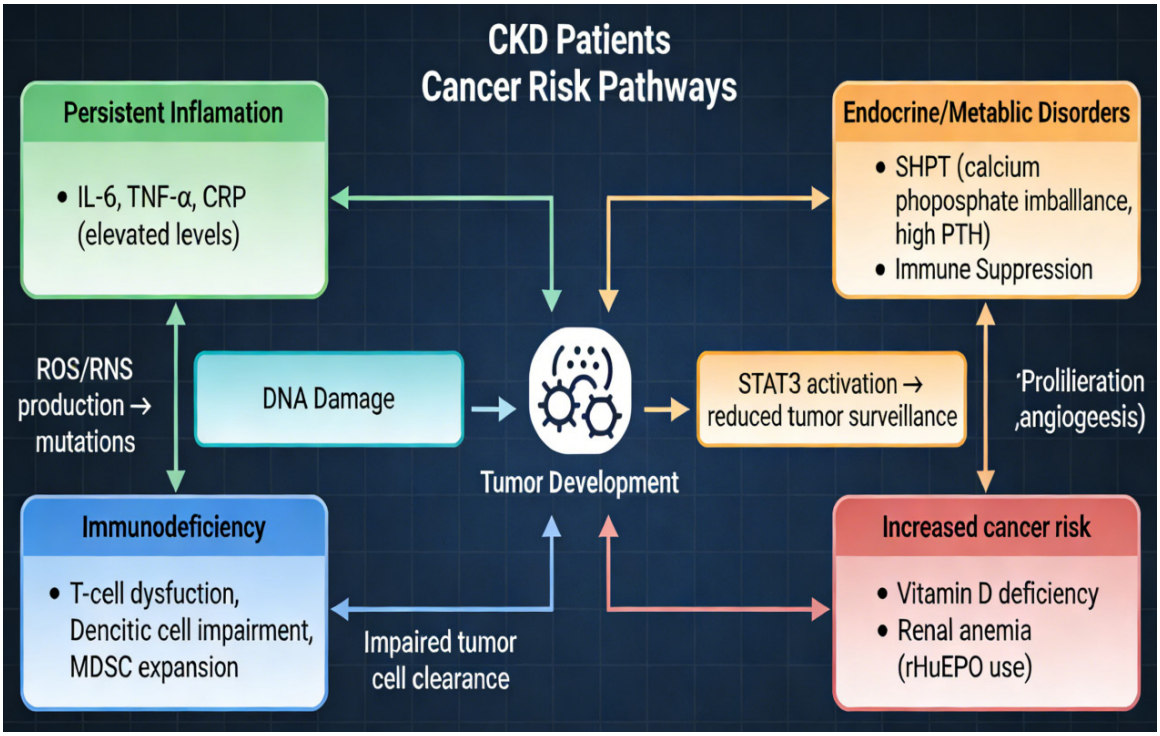

Figure 2. Key pathways and factors promoting tumor development and progression in patients with chronic kidney disease. CKD: Chronic Kidney Disease; IL-6: Interleukin-6; TNF-α: Tumor Necrosis Factor-alpha; CRP: C-Reactive Protein; ROS: Reactive Oxygen Species; RNS: Reactive Nitrogen Species; DNA: Deoxyribonucleic Acid; MDSC: Myeloid-Derived Suppressor Cells; SHPT: Secondary Hyperparathyroidism; PTH: Parathyroid Hormone; STAT3: Signal Transducer and Activator of Transcription 3; rHuEPO: Recombinant Human Erythropoietin.

er of carcinogenesis through multiple interconnected molecular pathways. First, persistent inflammation produces excessive reactive oxygen species and reactive nitrogen species, causing direct oxidative and nitrosogenic damage to genomic DNA. This not only induces mutagenic damage but also impairs DNA repair mechanisms, thereby increasing genomic instability and mutational burden, which is the basis for malignant transformation [24]. Second, inflammatory cytokines such as IL-6 activate key oncogenic signaling pathways. Notably, upon binding to its receptor, IL-6 triggers Janus kinase (JAK)-mediated phosphorylation, leading to dimerization of signal transduction and transcription activator 3 (STAT3). Phosphorylated STAT3 translocates to the nucleus, activating the transcription of genes crucial for cell survival (e.g., Bcl-2, Bcl-xL), proliferation (e.g., Cyclin D1, c-Myc), and angiogenesis (e.g., vascular endothelial growth factor (VEGF)). Simultaneously, this pathway suppresses antitumor immunity by inhibiting dendritic cell maturation and promoting the recruitment of immunosuppressive cells, thereby effectively weakening immune surveillance [25].

The inherent immune dysfunction in CKD exacerbates this pro-tumorigenic environment. Patients exhibit a paradoxical coexistence of nonspecific immune activation and immunosenescence, resulting in a weakened adaptive immune response against newly generated tumor cells [26]. This dysfunction is characterized by impaired T lymphocyte effector function, abnormal dendritic cell development, and pathological proliferation of myeloid-derived suppressor cells (MDSCs). In particular, MDSCs exert a powerful immunosuppressive effect by consuming essential amino acids like arginine, producing reactive oxygen species, and secreting immunosuppressive cytokines such as interleukin-10 and TGF-β, thus creating a favorable environment for tumor escape and progression [27, 28].

Furthermore, the endocrine and metabolic disorders specific to CKD also significantly increase the risk of cancer. Secondary hyper-

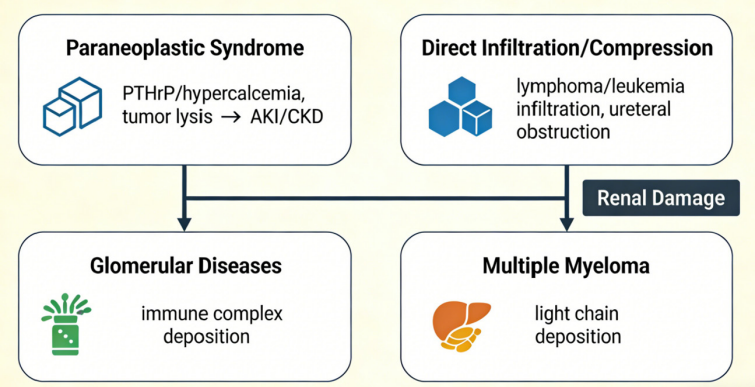

Figure 3. Mechanisms of kidney injury from malignant tumors: paratumor effects, direct invasion, and glomerular diseases. PTHrP: Parathyroid Hormone-related Protein; AKI: Acute Kidney Injury; CKD: Chronic Kidney Disease.

parathyroidism leads to hyperphosphatemia, hypocalcemia, and elevated parathyroid hormone levels, which, combined with vitamin D deficiency, constitute a mitotic environment. Parathyroid hormone and abnormal calcium signaling can abnormally activate proliferation pathways, while the lack of active vitamin D (1,25-dihydroxyvitamin D3) results in the loss of key anti-proliferative and pro-differentiation signals mediated by the vitamin D receptor (VDR), thereby reducing intrinsic tumor suppression [29, 30]. Additionally, while recombinant human erythropoietin is used to correct renal anemia, long-term high-dose use has been shown to be associated with an increased risk of certain malignancies, such as breast cancer. It is speculated that this risk stems from the expression of functional erythropoietin receptors (EPOR) on the surface of certain tumor cells. After erythropoietin binds to these receptors, it can directly stimulate tumor cell proliferation and survival through the JAK2/STAT5 signaling pathway and enhance tumor angiogenesis [31].

In conclusion, the combined effects of chronic inflammation, severe immune dysregulation, and specific endocrine and metabolic disorders in CKD patients create a multifactorial environment highly conducive to tumor development and progression. Therefore, actively correcting these potential disorders, such as managing secondary hyperparathyroidism, supplementing with vitamin D, and carefully assessing the risk-benefit ratio of recombinant human erythropoietin therapy, are key strategies for preventing and controlling cancer in this susceptible population [32].

Renal dialysis and the development of malignant tumors

Epidemiological studies have strongly confirmed that patients who receive long-term dialysis exhibit a markedly elevated risk of acquired cystic kidney disease-associated renal cell carcinoma (ACKD-RCC), up to several tens of times higher [33]. The mechanism of this increased carcinogenicity stems from the cystic environment induced by long-term dialysis. In these acquired renal cysts, the lining epithelial cells are subjected to chronic oxidative stress, persistent inflammatory signaling, and abnormal stimulation by growth factors. Oxidative stress mediated by excessive reactive oxygen species causes DNA damage and exacerbates genomic instability. At the same time, local inflammation characterized by elevated cytokine levels activates pro-survival pathways such as NF-κB, which inhibits apoptosis. In addition, abnormal autocrine/paracrine regulation of growth factors like VEGFand TGF-α provides persistent mitotic and angiogenesis-promoting signals through their respective receptor tyrosine kinases. The synergistic effect of these stimuli drives the malignant transformation of cyst-lining cells, ultimately leading to ACKD-RCC [34]. See **Figure 3**.

Besides the native kidneys, renal transplant recipients face a 2 to 5-fold higher overall cancer risk than the general population, largely attributed to long-term, potent immunosuppressive therapy [35]. This increased risk operates through a dual mechanism. First, immunosuppressants such as calcineurin inhibitors (e.g., cyclosporine, tacrolimus), azathioprine, and mycophenolate mofetil systematically impair T-cell-mediated immune surveillance, weakening the body's ability to recognize and eliminate malignant or virus-transformed cells. Second, some of these drugs possess direct pro-oncogenic properties. For instance, cyclosporine A not only has immunosuppressive effects but also actively promotes

Table 1. Increased cancer risk and main potential mechanisms in patients at different stages of CKD

CKD Status	Significantly Increased Cancer Risk	Main Potential Mechanisms
Non-dialysis CKD patients	Slightly increased risk of kidney, bladder, and colorectal cancer.	Accumulation of uremic toxins (e.g., IS, PCS), chronic systemic inflammation, metabolic disorders (e.g., secondary hyperparathyroidism, vitamin D deficiency), oxidative stress, gut microbiota dysbiosis.
Maintenance hemodialysis patients	Significantly increased risk of kidney cancer (especially ACKD-RCC), thyroid cancer, and multiple myeloma; increased risk of upper urinary tract transitional cell carcinoma.	Inadequate dialysis, recurrent chronic inflammation, acquired cystic kidney disease, bio-incompatibility of dialysate, exposure to endotoxins in dialysis water, immune dysfunction, cumulative age-related effects.
Peritoneal dialysis patients	Possible increased risk of peritoneal mesothelioma and gastrointestinal cancers (especially colorectal cancer).	Chronic peritoneal inflammation/fibrosis, stimulation by high-glucose dialysate (formation of AGEs), recurrent peritonitis, altered local immune environment in the peritoneal cavity, metabolic abnormalities due to long-term glucose absorption.
Kidney transplant recipients	Significantly increased risk of non-melanoma skin cancer (NMSC), post-transplant lymphoproliferative disorder (PTLD), Kaposi's sarcoma, renal cell carcinoma, and cervical cancer.	Long-term potent immunosuppressive therapy (impaired immune surveillance), viral reactivation or infection (EBV, HPV, HHV-8, HBV/HCV), direct carcinogenic effects of immunosuppressants (e.g., promotion of the TGF-β pathway), chronic antigenic stimulation, persistent inflammatory state associated with graft dysfunction.

CKD: Chronic Kidney Disease; IS: Indoxyl Sulfate; PCS: p-Cresyl Sulfate; ACKD-RCC: Acquired Cystic Kidney Disease-associated Renal Cell Carcinoma; RCC: Renal Cell Carcinoma; AGEs: Advanced Glycation End Products; NMSC: Non-Melanoma Skin Cancer; PTLD: Post-Transplant Lymphoproliferative Disorder; EBV: Epstein-Barr Virus; HPV: Human Papillomavirus; HHV-8: Human Herpesvirus 8 (Kaposi's sarcoma-associated herpesvirus); HBV: Hepatitis B Virus; HCV: Hepatitis C Virus; TGF-β: Transforming Growth Factor-beta.

tumor progression by enhancing the transcription and secretion of TGF-β. Subsequently, elevated TGF-β levels activate Smad-dependent signaling pathways in tumor cells, promoting epithelial-mesenchymal transition, enhancing invasiveness, and accelerating metastasis [36]. Finally, chronic renal failure and long-term dialysis itself cause persistent, cumulative damage to the immune system, leading to acquired immunodeficiency. This compromised immunity significantly increases susceptibility to oncogenic viral infections. Some viruses have been shown to be direct carcinogens: chronic hepatitis B virus and hepatitis C virus infection are etiologically associated with hepatocellular carcinoma and certain subtypes of non-Hodgkin's lymphoma, primarily through viral proteins mediating disruption of host cell cycle checkpoints and inducing chronic inflammation [37, 38]. Similarly, persistent infection with high-risk human papillomavirus (HPV) strains increases the risk of cervical, anogenital, and oropharyngeal cancers, while uremia-associated immune dysfunction hinders the clearance of these viruses. The oncoproteins E6 and E7 of HPV drive carcinogenesis by inactivating key tumor suppressor p53 and retinoblastoma protein, respectively, leading to uncontrolled cell proliferation [39]. See **Table 1**.

Gut microbiota imbalance: an emerging metabolic and immune regulatory hub for tumor development

In recent years, the role of gut microbiota imbalance in the development of CKD-related tumors has received increasing attention, becoming a key link between uremic toxins, chronic inflammation, and immune disorders. Gut microbiota imbalance is common in CKD patients, characterized by a decrease in beneficial bacteria that produce short-chain fatty acids (SCFAs), such as Bifidobacteria and Lactobacilli, while excessive proliferation of protein-fermenting bacteria that produce uremic toxin precursors, such as indole and phenolic bacteria [40]. This imbalance profoundly affects the host's tumor susceptibility through a dual mechanism of the "gut-kidney axis" and the "gut-tumor axis".

First, dysbiosis directly exacerbates the accumulation of uremic toxins. As mentioned earlier, indole and p-cresol produced by bacterial metabolism are converted into IS and PCS after being metabolized by the host. Increased intestinal permeability during CKD (i.e., "leaky gut") makes these toxin precursors and themselves more readily translocated into the bloodstream [41]. Therefore, dysbiosis is one of the root causes of elevated levels of protein-bound toxins such as IS and PCS, which in turn exert their direct genotoxic and pro-cancer effects by activating pathways such as AhR.

Secondly, dysbiosis remodels the tumor microenvironment and host immunity through metabolites. The reduction of beneficial bacteria leads to a decrease in the production of short-chain fatty acids (such as butyrate), which have anti-inflammatory effects and maintain the integrity of the intestinal barrier [42]. Butyrate is not only a major energy source for colonic epithelial cells, but it can also act as an inhibitor of histone deacetylase, inhibiting tumor cell proliferation and inducing apoptosis through epigenetic regulation. Its deficiency weakens the local anti-tumor barrier in the gut. At the same time, dysbiosis continuously activates systemic low-grade inflammation through pattern recognition receptors (such as TLR4), releasing cytokines such as IL-6 and TNF-α, providing fertile "soil" for tumor growth [43]. More importantly, gut microbiota and its metabolites can directly modulate systemic antitumor immunity. For example, specific microbiota can affect the proliferation and function of MDSCs, regulate the balance between cytotoxic T cells and regulatory T cells, and thus affect the effectiveness of immune surveillance [44].

Furthermore, there are complex interactions between gut microbiota and cancer treatment (including immunosuppression after renal transplantation). Some immunosuppressants (such as mycophenolate mofetil) may further disrupt the composition of the gut microbiota, and the metabolism of the microbiota can affect the bioavailability and efficacy of these drugs, forming a two-way effect that may indirectly affect cancer risk [45].

In summary, gut microbiota dysbiosis is a core amplifier and integrator of increased cancer risk in CKD patients. It is not only the "source" of uremic toxins, but also systematically shapes the host internal environment that promotes tumorigenesis through three core pathways: metabolism, inflammation, and immunity. Therefore, intervention strategies targeting the gut microbiota (such as probiotics, prebiotics, dietary adjustments, or fecal microbiota transplantation) are not only a new direction for managing CKD complications, but also a potential innovative target for reducing cancer risk in this population [46]. Future research needs to further clarify the causal roles of specific strains and their metabolites in order to promote precision intervention.

Treatment of malignant tumors can trigger or worsen CKD

Malignant tumors can affect kidney function in a variety of ways, including direct or indirect effects of the tumor and nephrotoxicity associated with anticancer drugs. Antitumor therapy is the most common cause of CKD [40].

Direct or indirect effects of tumors

Studies have confirmed that tumors can affect renal function directly or indirectly, mainly in the following ways. First, paraneoplastic syndromes, lung cancer, and gastrointestinal malignancies can secrete hormone-like substances (such as parathyroid hormone-related peptide), causing hypercalcemia and potentially leading to tumor lysis syndrome, which in turn can cause acute kidney injury (AKI), and AKI may develop into CKD [41]. At the same time, malignant tumors themselves can infiltrate or compress the renal parenchyma. For example, malignant lymphoma and leukemia can directly infiltrate the kidneys; retroperitoneal tumors (such as neurogenic tumors, liposarcomas, or leiomyosarcomas) or pelvic gynecological tumors can compress the ureter, leading to dynamic renal obstruction, which may cause long-term renal insufficiency or renal failure [42]. Furthermore, statistical data shows that solid malignant tumors can cause glomerular diseases, including membranous nephropathy, minimal change disease, and other forms of glomerulonephritis, which may be related to the deposition of immune complexes induced by malignant tumor antigens or cross-immune reactions against common antigens [43]. In addition, certain specific types of tumors can also cause kidney damage. Monoclonal immunoglobulin light chains produced by plasma cell

diseases such as multiple myeloma are directly deposited in the kidneys, causing light chain amyloidosis or light chain deposition disease, thereby leading to kidney damage [44].

Nephrotoxicity of tumor-related therapy

Cytotoxic drugs and targeted therapies: Currently, the mainstream view is that tumor treatment is the main cause of renal function loss, and this is the result of multiple factors working together [45]. First, traditional cytotoxic chemotherapy drugs include platinum-based drugs (cisplatin, carboplatin) and methotrexate (MTX). Among them, cisplatin is a classic example of a drug with strong nephrotoxicity. It can accumulate in renal tubular epithelial cells, induce oxidative stress and apoptosis, and thus cause AKI. Repeated use can also lead to irreversible chronic kidney injury. Secondly, targeted therapies, such as anti-VEGF pathway drugs (bevacizumab, sunitinib, sorafenib, etc.) and mammalian target of rapamycin (mTOR) inhibitors (everolimus, tesimolimus), while inhibiting VEGF signaling and reducing cell proliferation, can damage the integrity and repair capacity of glomerular capillary endothelial cells, leading to proteinuria, hypertension, and even thrombotic microangiopathy; the latter may cause proteinuria and acute interstitial nephritis, resulting in kidney damage.

Treatment of other tumor-related kidney injuries: Currently, immune checkpoint inhibitors, drugs for treating bone metastases, radiotherapy, and hematopoietic stem cell transplantation, which are widely used in the treatment of various tumors, can all lead to kidney injury [46]. For example, programmed death protein-1/programmed death protein ligand-1 inhibitors and cytotoxic T-lymphocyte-associated antigen 4 inhibitors may induce acute interstitial nephritis or glomerulonephritis, possibly related to the activation of T lymphocytes and their attack on normal kidney tissue expressing the corresponding antigens, leading to immune-related nephritis [47]. Bisphosphonates used to treat bone metastases are directly associated with collapsed focal segmental glomerulosclerosis and acute tubular injury [48]. As for kidney injury associated with radiotherapy and hematopoietic stem cell transplantation, radiotherapy itself can cause radiation nephropathy, manifested as hypertension, proteinuria, and progressive renal failure. Meanwhile, highdose chemoradiotherapy before transplantation, graft-versus-host disease (GVHD) after transplantation, and calcineurin inhibitors used to prevent GVHD can all induce AKI and the transformation of AKI into CKD.

Tumor development and clinical management of CKD

At present, most scholars tend to conduct early screening for CKD patients, while cancer patients can protect their kidney function by adjusting the dosage, frequency, and method of drug administration.

Tumor screening for CKD patients

Studies have confirmed that cancer screening for CKD patients can effectively and proactively detect tumors early and improve the overall prognosis of patients [49]. Similarly, different effective prevention methods should be adopted for different states of CKD. For CKD patients who generally do not require dialysis or transplantation, the main approach is to follow the cancer screening guidelines for the general population (breast cancer, cervical cancer, digestive system tumors, lung tumors, etc.), while focusing on screening for cancers related to CKD (such as renal cancer and urothelial carcinoma) [50]. For patients undergoing long-term (> 3 years) dialysis or kidney transplantation, lifelong renal ultrasound screening is recommended to monitor for ACKD-RCC and to implement other tumor surveillance programs [51-53].

Dose adjustment strategy of drug therapy for patients with CKD and cancer

Relevant guidelines recommend that the principles of medication use in patients with CKD and cancer should comprehensively consider changes in pharmacokinetics and efficacy, pharmacokinetic alterations, guidance on the risk of cumulative nephrotoxicity, and accurate assessment of renal function [54]. For example, for drugs primarily excreted by the kidneys (such as methotrexate, cisplatin, bleomycin, pemetrexed, etc.), dosage adjustment must be made based on estimated glomerular filtration rate or their use should be avoided [55-57]. For drugs metabolized by the liver or with high pro-

Table 2. Dose adjustment reference of commonly used anticancer drugs in patients with CKD and cancer

Drug category/ name	Main excretion pathways	Risk of nephrotoxicity	EGFR ≥ 60 mL/min	EGFR 30-59 mL/min	EGFR 15-29 mL/min	EGFR < 15 mL/min or dialysis	Clinical strategies
Platinum: cisplatin	Kidney	Polar altitude	Full dose	Use with caution, greatly reduce or avoid	Avoid using	Forbidding	Strong emetic, must be fully hydrated, monitoring electrolyte.
Platinum: Carboplatin	Kidney	Medium-low	Calculated by AUC	Calculated by AUC, but eGFR was calculated by Calvert formula	caution	Medication after dialysis	Calvert formula: dose = AUC × (eGFR + 25).
Antimetabolic drug: Methotrexate	Kidney	High (high dose)	Full dose	Decrement	Avoid large doses; blood concentration should be closely monitored in low dose	Disable large doses; be cautious in small doses	When large doses are used, hydration, alkalization of urine, calcium folinate rescue, and monitoring of blood concentration to < 0.
Antimetabolic drug: pemetrexed	Kidney	Cataphyll	Full dose	Reduced to 75%	Reduced to 50%	It is recommended to avoid or extremely low doses	Supplement folic acid and vitamin B12.
Targeted drug: sunitinib	Liver (bile)	Middle (proteinuria, hypertension)	Full dose	Full dose	Guard	Guard	Monitoring of blood pressure, urine protein; liver function should be adjusted when incomplete.
Immunotherapy: Paporizumab	Non-kidney (protein degradation)	Low (but may induce immune nephritis)	Full dose	Full dose	Full dose	Full dose	Monitoring of immune-related adverse events.

EGFR: Estimated Glomerular Filtration Rate; AUC: Area Under the Curve (referring to the area under the drug concentration-time curve); eGFR: estimated Glomerular Filtration Rate; mL/min: milliliters per minute; Calvert formula: The Calvert Formula (a dosing calculation formula for carboplatin); CKD: chronic kidney disease.

tein binding (such as most targeted therapies, paclitaxel, etc.), which are primarily metabolized by the liver, dosage adjustment guidelines may not be clear, thus requiring closer monitoring of their toxicity [58-60]. **Table 2** lists dosage adjustment recommendations for some common anticancer drugs.

Conclusions and future prospects

CKD and cancer have a pathophysiologically mutually reinforcing relationship. CKD promotes inflammation, oxidation, and decreased immune function, and the systemic environment increases the risk of cancer. Conversely, cancer and its various treatments can also lead to CKD. Therefore, the principle of "treating kidney disease and cancer simultaneously" should be followed in clinical practice. Future research directions include: firstly, identifying specific molecular pathways associated with CKD-related carcinogenesis as potential targets for future interventions; secondly, strengthening research on CKD-related risks and drug metabolism in cancer patients to obtain accurate pharmacokinetic and efficacy data and improve medication safety; and finally, utilizing big data and artificial intelligence to develop precise cancer screening and risk assessment tools for CKD patients and exploring new renal replacement therapy methods (especially research on the impact of different dialysis modalities on reducing the metabolism of anticancer drugs).

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Disclosure of conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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