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CLINICAL STUDY

Knowledge mapping and visualized analysis of research progress in onconephrology: a bibliometric analysis

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ABSTRACT

Objectives: Onconephrology is an expanding subspecialty focused on the management of cancer patients with renal injury. This study used a comprehensive bibliometric analysis to emphasize the need for cooperation between oncologists and nephrologists, exploring current trends and future research areas in onconephrology.

Methods: Relevant literature on onconephrology published between 1 January 2000 and 27 April 2024 was retrieved from the Science Citation Index Expanded of the Web of Science Core Collection, followed by manual screening. Bibliometric analyses were performed using CiteSpace, VOSviewer, and Bibliometrix software.

Results: A total of 1,853 publications, including 1,647 articles and 206 reviews, by 11,606 authors from 2,757 institutions in 73 countries, were analyzed. Annual publications generally follow a steadily increasing trend, ranging from 25 to 161 documents. The United States ($n = 464$), The University of Texas MD Anderson Cancer Center ($n = 39$), Meletios A. Dimopoulos ($n = 21$), and *Nephrology Dialysis Transplantation* ($n = 35$) were the most productive country, institution, author, and journal, respectively. Immune checkpoint inhibitors, glomerular filtration rate, and cisplatin were clusters of highly cited references after 2015. Oxaliplatin, calcium, open-label, and thrombotic microangiopathy were trending topics after 2020. Outcome, acute kidney injury, immunotherapy, and chronic kidney disease were keyword bursts that persisted through 2024.

Conclusion: Current research of onconephrology is focusing on chemotherapeutic nephrotoxicity, kidney function assessment, dosing of chemotherapeutic agents in chronic kidney disease, glomerular disease in cancer, immunotherapy, and electrolyte disturbances. Future directions in this field include clinical trials and thrombotic microangiopathy.

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1. Introduction

Onconephrology is a new subspecialty that combines nephrology and oncology, focusing on their complex interactions. In general, onconephrology refers to drug nephrotoxicity and end-organ damage from underlying malignancies beyond cancers of the kidney. Although the term 'onconephrology' was coined in the 2010s [1], the practice of involving nephrologists in the care of cancer patients dates back much further. Common and specific scenarios of onconephrology often include acute kidney injury (AKI), electrolyte disturbances, cancer-related glomerular diseases, paraproteinemia, and hematopoietic stem cell transplant-related kidney disorders. Regardless of the specific form, the mission of onconephrology is to provide multidisciplinary, coordinated, and collaborative care involving both nephrologists and oncologists for cancer patients.

Bibliometric analysis uses mathematical and statistical approaches to analyze research hotspots and provide insight into future directions by evaluating the published literature in a specific field quantitatively or semiquantitatively [2]. In contrast to traditional meta-analysis, which predominantly synthesizes new data, bibliometric analysis instead focuses on analyzing countries, institutions, authors, journals, documents, references, and keywords to accurately capture the hot topics and research prospects in a particular research area. Bibliometric analysis has been successfully applied to identify future research directions in various medical specialties [3].

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Research has dramatically improved our understanding of the underlying pathogenesis and management of onconephrology. Although several narrative reviews on onconephrology have been published recently [4–6], there is still a significant need for comprehensive bibliometric analyses to provide information on development trends and key research topics. This study aimed to analyze the current status of onconephrology and to provide nephrologists and oncologists with an overview of hotspots and clinical insights in the field of onconephrology.

2. Materials and methods

2.1. Data sources and literature inclusion and exclusion criteria

This study, like earlier research [7], used the Science Citation Index Expanded of the Web of Science Core Collection (SCIE-WOS) database for literature searches. The emphasis was on documents focusing on both cancer and kidney diseases, excluding renal cancer and kidney transplant-related issues. Only English-language articles or reviews published between 1 January 2000 and 27 April 2024 (the date of the literature search), were included. Exclusions were: (1) non-article/review documents (e.g., editorials, meeting abstracts); (2) non-English publications; and (3) articles published before 1 January 2020. Two authors (YWW and SLF) screened titles and abstracts to remove irrelevant studies, with disputes resolved by a third author (WW). The search and selection process is outlined in Figure 1.

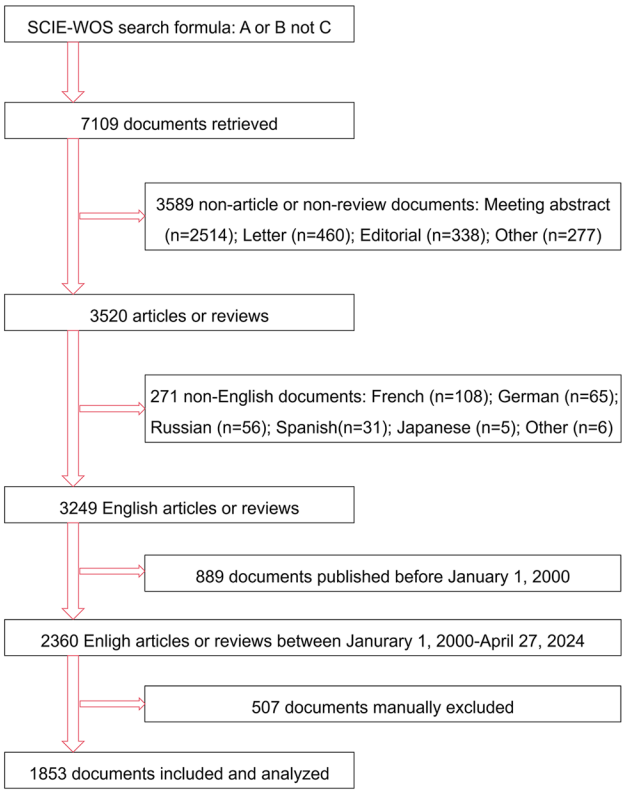


Figure 1. Study flowchart showing the literature retrieval strategy and the process of literature exclusion. The search strategy was the presence of both cancer and kidney diseases, excluding renal cancer itself. So the 'a' in the search formula refers to TS = (onconephrology or onco-nephrology), directly denoting onconephrology. 'B' refers to 'TI = (cancer* or tumo* or oncology or neoplasm* or carcinoma* or sarcoma* or myeloma* or leukemia*) and TI = (renal or kidney or neph* or glome*)', suggesting that the title of the paper must be related to both kidney and cancer. 'C' refers to documents related to kidney cancer itself, the search formula for which is provided in the Supplementary Material.

2.2. Data visualization and analysis

The included documents were first downloaded from SCIE-WOS and stored in plain text format with full records and complete references (Supplementary Material). The analyses of countries, authors, and the generation of co-occurrence and burst graphs were enabled by using CiteSpace (version 6.2.7R, Dr. Chaomei Chen, China) with time slices (2000–2024), years per slice (1), node types (country/author), and g indices and scale factors ($k=25$). The software VOSviewer (Leiden University Center for Science and Technology Studies) was used for the analysis of institutions, journals, cited references, and keywords. During the keyword analysis, terms with similar meanings, such as acute renal failure and acute kidney injury, were grouped together. The journal impact factor (IF) was obtained from the most recent Journal Citation Reports.

3. Results

3.1. Trends of annual publications and cumulative citations

After applying the inclusion and exclusion criteria, a total of 1853 documents, including 1,647 articles and 206 reviews, were included in the final bibliometric analysis. As shown in Figure 2, the number of annual publications generally follows a steadily increasing trend, ranging from a low of 25 documents in 2003 to a high of 161 documents in 2023. Similarly, the number of cumulative citations is also increasing, suggesting that the area of onconephrology is gaining momentum.

3.2. Country and institution analysis

The results of the country and institution analyses are presented in Figure 3. Overall, 2757 institutions from 73 countries contributed to these 1853 documents. The United States ($n=464$), China ($n=287$), Japan ($n=270$), England ($n=116$), and France ($n=112$) were the top 5 countries in terms of total scientific output, accounting for 67.40% of the total onconephrology literature. However, when analyzed with an emphasis on importance as indicated by betweenness centrality, the United States, England, France, Canada, and Germany were hub nodes with betweenness centralities of 0.36, 0.23, 0.19, 0.15, and 0.12, respectively. Institutional analysis revealed that the top 5 institutions with the highest publication volume were the University of Texas MD Anderson Cancer Center ($n=39$), Mayo Clinic ($n=36$), National and Kapodistrian University of Athens ($n=39$), Dana-Farber Cancer Institute ($n=26$), and Yale University ($n=26$).

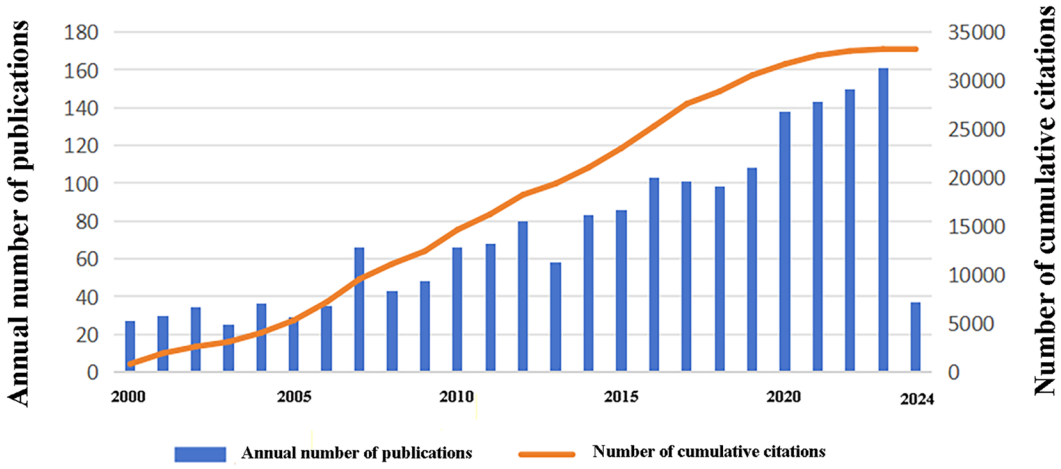


Figure 2. The number of annual publications and cumulative citations on onconephrology from 1 January 2000 to 27 April 2024.

3.3. Analysis of prolific authors and journals

Figure 4 shows that the 1,853 documents published in 601 academic journals were contributed by 11,606 authors. The authors with ≥10 total publications were Meletios A. Dimopoulos ($n = 21$, National and Kapodistrian University of Athens), Kenar D. Jhaveri ($n = 14$, Zucker School of Medicine), Nelson Leung ($n = 13$, Mayo Clinic), Efstathios Kastritis ($n = 11$, National and Kapodistrian University of Athens), Evangelos Terpos ($n = 11$, National and Kapodistrian University of Athens), Jolanta Malyszko ($n = 11$, Medical University of Warsaw), and Paul Cockwell ($n = 10$, Queen Elizabeth Hospital). Among the 16 journals that published ≥20 documents in this field, the top 5 with the most publications were *Nephrology Dialysis Transplantation* ($n = 35$, IF = 6.1), *Internal Medicine* ($n = 32$, IF = 1.2), *Cancer Therapy and Pharmacology* ($n = 31$, IF = 3.0), *Clinical Nephrology* ($n = 28$, IF = 1.1), and PLoS One ($n = 27$, IF = 3.7). Among the top 10 most productive journals, five were nephrology journals, three were multidisciplinary journals, and two were cancer journals.

3.4. Influential documents and highly cited references

The analyses of the most cited documents and references are summarized in Tables 1 and 2, respectively. The timeline view of the cited references, as displayed in Figure 5(A), shows that the references cited between 2000 and 2015 were related to lenalidomide, dose adjustment, acute interstitial nephritis, ibandronate, interleukin-2, multiple myeloma, and iron. In comparison, the clusters identified after 2015 were immune checkpoint inhibitors, glomerular filtration rate, and cisplatin. The reference burst analysis (Figure 5(B)) revealed that the articles 'Clinical Features and Outcomes of Immune Checkpoint Inhibitor-Associated AKI: A Multicenter Study' by Cortazar et al. in 2020 and 'The Incidence, Causes, and Risk Factors of Acute Kidney Injury in Patients Receiving Immune Checkpoint Inhibitors' by Seethapathy et al. in 2019 were recent bursts.

3.5. Keyword analysis

A total of 5,630 keywords were extracted, of which 316 and 54 keywords appeared ≥10 times and ≥50 times, respectively. As shown in Figure 6(A), the top 10 most frequent keywords were acute kidney injury ($n = 537$), cancer ($n = 340$), multiple myeloma ($n = 281$), renal insufficiency ($n = 277$), nephrotoxicity ($n = 269$), risk factors ($n = 249$), chemotherapy ($n = 235$), chronic kidney disease (CKD, $n = 215$), dialysis ($n = 193$), and cisplatin ($n = 189$). Cluster analysis (Figure 6(B)) revealed that these keywords can be grouped into eight clusters, including multiple myeloma (#0), cisplatin (#1), nephrotic syndrome (#2), chronic kidney disease (#3), immunotherapy (#4), oxidative stress (#5), zoledronic acid (#6), and hepatocellular carcinoma (#7). Trend analysis (Figure 7) revealed that immune checkpoint inhibitors, oxaliplatin, calcium, open-label, and thrombotic microangiopathy emerged after 2020. The keyword burst analysis (Figure 8) indicated that outcome, acute kidney injury, immunotherapy, risk factors, and chronic kidney diseases were bursts that still persist through 2024.

4. Discussion

This bibliometric analysis revealed that the field of onconephrology is steadily expanding. Moreover, although China and Japan have made significant contributions to this field, their impact on the field is suboptimal. Current research hotspots are diverse and include chemotherapeutic nephrotoxicity, dosing of chemotherapeutic agents in CKD, kidney function assessment, glomerular disease in cancer, immunotherapy, and electrolyte disturbances. Future directions in this field may encompass clinical trials and thrombotic microangiopathy (TMA).

4.1. General information

Although the bidirectional relationship between kidney disease/function and cancer has long been well known, the

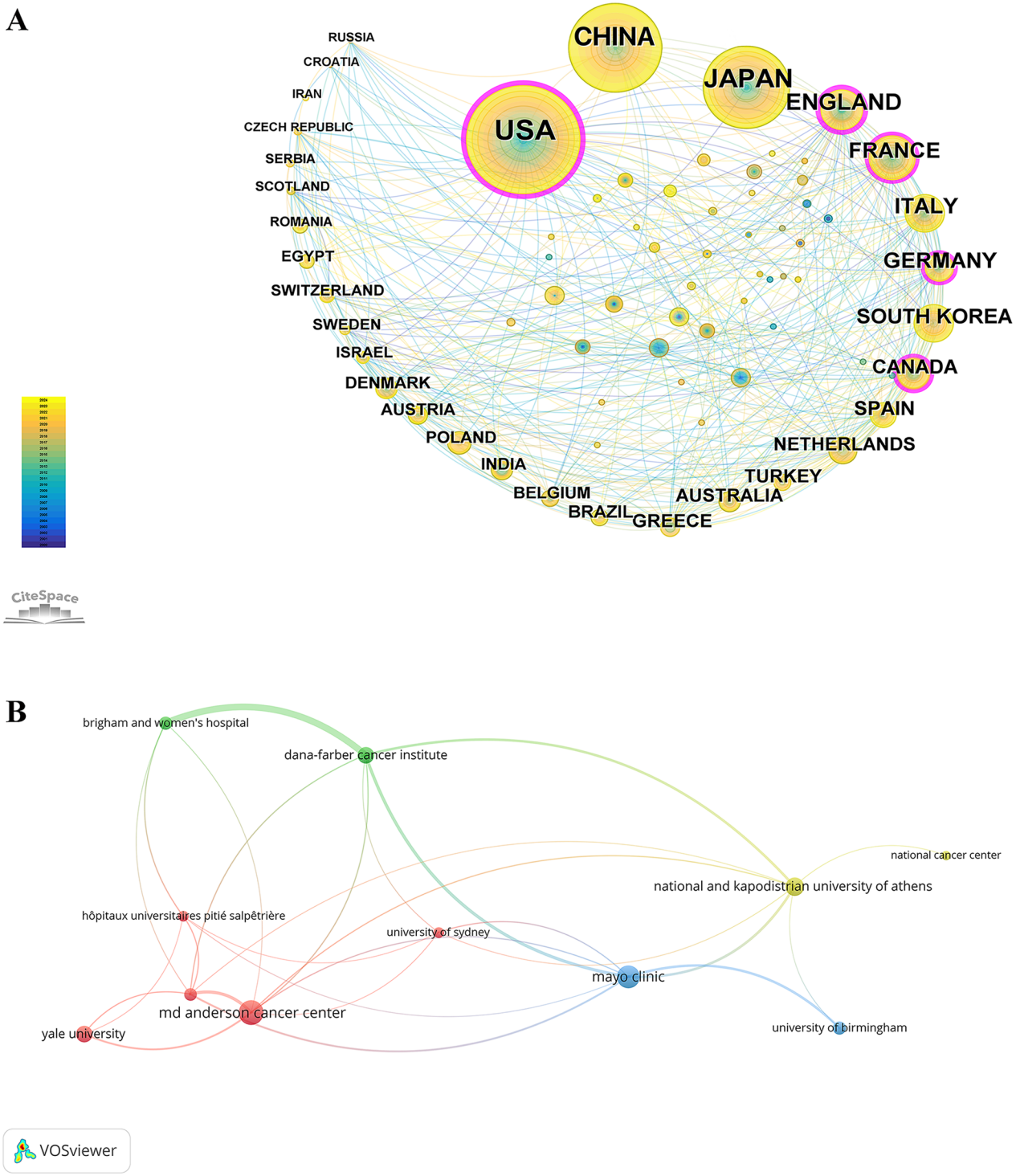


Figure 3. Cooperation network between countries (A) and institutions (B) in the field of onconephrology between 1 January 2000 and 27 April 2024. The size of each node is proportional to the number of papers published by that country. The nodes with pink rings were central nodes with high importance. The lines connecting the nodes illustrate the collaborative relationships between countries, with the thickness of the lines indicating the strength of these collaborations.

term 'onconephrology' was coined in the early 2010s. In fact, it was not until 2019 that an international conference on onconephrology was held to address the key issues at the challenging clinical interface of onconephrology [8]. Interestingly, our bibliometric analysis indicated that practice and multidisciplinary cooperation have already been present, as indicated by the number of publications in this field.

Country, institution, and author analysis suggested that Western countries and researchers are the driving force in this challenging endeavor. Although China and Japan ranked 2nd and 3rd in terms of scientific output, none of the top 10 institutions or researchers were based in China or Japan, highlighting that these nations should cultivate more influential research with greater scientific impact. The journal

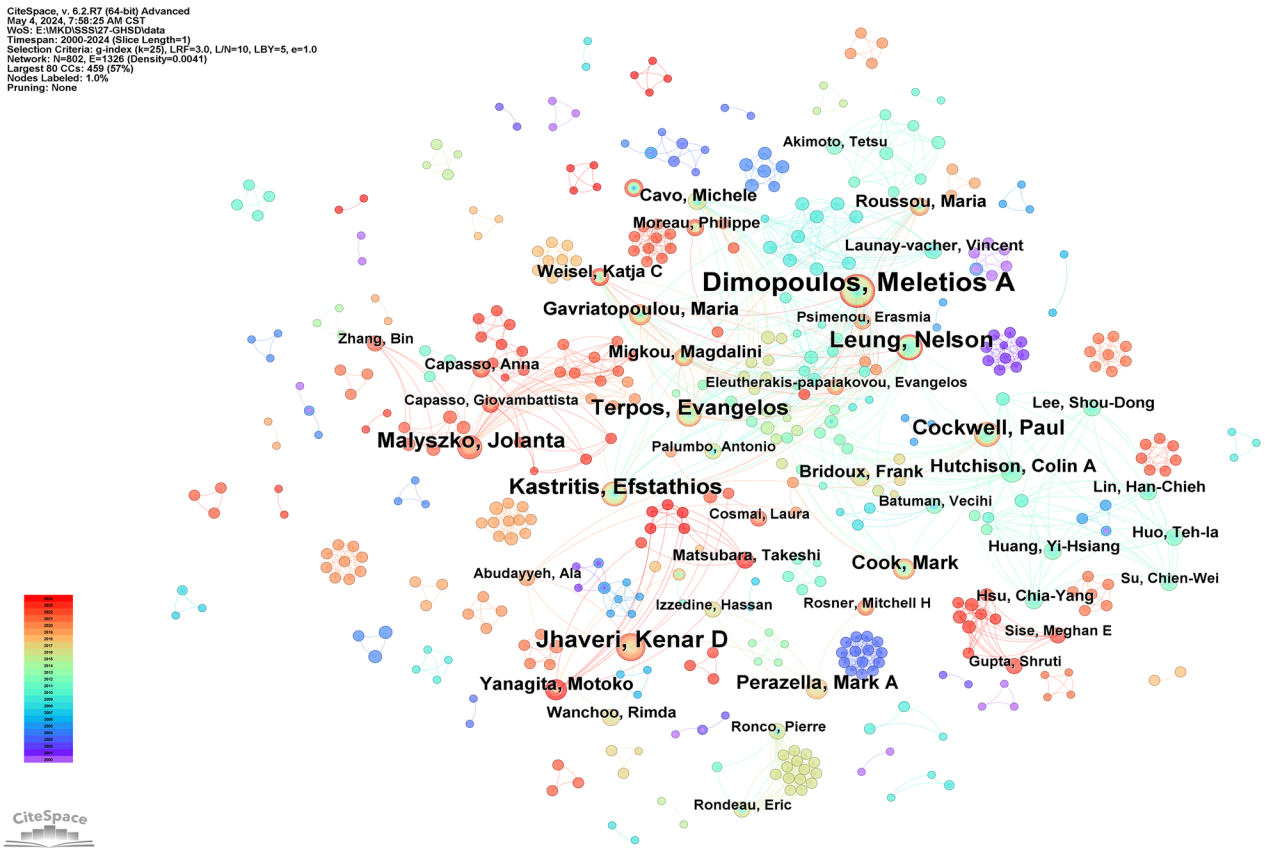


Figure 4. Collaboration network between scholars on onconephrology. The size of the node is proportional to the research output of the author, and those marked with rings are hub authors. Different colors represent various author collaboration clusters.

analysis also indicated that the research activities in the field of onconephrology encompass related specialties, including nephrology, cancer, and multiple disciplines. Therefore, interested researchers can obtain the latest information from these journals and should contribute their studies to these journals.

4.2. Current research hotspots

The timeline view analysis of the most highly cited references and the keyword analysis revealed that current hotspots in the field of onconephrology are broad and involve both solid and hematologic malignancies. In addition, chemotherapeutic nephrotoxicity, the dosing of chemotherapeutic agents in CKD patients, glomerular diseases in cancer patients, kidney function measurement, electrolyte disturbances, immunotherapy, and basic research have emerged as current hotspots.

4.2.1. Types of malignancies and treatment modalities involved

Among the top 10 most cited documents and references, 6/10 and 6/10 were related to multiple myeloma, indicating that multiple myeloma-related renal insufficiency is still the most important and hottest topic in onconephrology. Approximately half of multiple myeloma patients are estimated to develop renal dysfunction during the course of treatment [9]. More importantly, numerous studies have associated renal impairment with decreased overall survival and premature mortality in multiple myeloma patients [10]. The main mechanism of renal injury in multiple myeloma is the overproduction of free light chains, which are nephrotoxic because they promote proximal tubule apoptosis and induce inflammation with subsequent interstitial fibrosis [11]. In addition, other conditions associated with multiple myeloma, such as amyloidosis, monoclonal immunoglobulin deposition disease, light-chain proximal tubulopathy, and cryoglobulinaemia, may also cause renal impairment. Notably, other multiple myeloma factors, such as dehydration, hypercalcemia, and tumor lysis syndrome, and treatment-related factors, such as hematopoietic stem cell transplantation, are also essential etiologies. Our bibliometric results also suggest that solid tumors, represented by the cluster 'hepatocellular carcinoma', are also involved. In fact, many treatments for solid tumors, such as targeted therapy and immunotherapy, have also been associated with a variety of renal complications [12]. For example, immune checkpoint inhibitors, such as programmed cell death (PD)-1/PD-L1, have been found to cause hypophosphatemia, proteinuria, acute interstitial nephritis, and acute tubular necrosis [13].

Our study revealed that in addition to conventional cytotoxic chemotherapies, emerging novel cancer therapies, such as immunotherapies (immune checkpoint inhibitors and chimeric antigen receptor-T [CAR-T] cell therapy) and targeted

Table 1. Top 10 most highly cited documents in the field of onconephrology between 1 January 2000 and 27 April 2024.

Rank	First author	Publication year	Document title	Journal	Impact factor	Citation number
1	Solano et al.	2006	A comparison of symptom prevalence in far advanced cancer, AIDS, heart disease, chronic obstructive pulmonary disease and renal disease. J Pain Symptom Manage	J Pain Symptom Manage	4.7	750
2	Launay-Vacher et al.	2007	Prevalence of Renal Insufficiency in cancer patients and implications for anticancer drug management: the renal insufficiency and anticancer medications (IRMA) study	Cancer	6.2	338
3	Knudsen et al.	2000	Renal failure in multiple myeloma: reversibility and impact on the prognosis. Nordic Myeloma Study Group	Eur J Haematol	3.1	312
4	Dimopoulos et al.	2010	Renal impairment in patients with multiple myeloma: a consensus statement on behalf of the International Myeloma Working Group	J Clin Oncol	45.3	304
5	Dimopoulos et al.	2016	International Myeloma Working Group Recommendations for the Diagnosis and Management of Myeloma-Related Renal Impairment	J Clin Oncol	45.3	252
6	Volarevic et al.	2019	Molecular mechanisms of cisplatin-induced nephrotoxicity: a balance on the knife edge between renoprotection and tumor toxicity	J Biomed Sci	11	249
7	Clark et al.	2005	Plasma exchange when myeloma presents as acute renal failure: a randomized, controlled trial	Ann Intern Med	39.2	244
8	Wanchoo et al.	2017	Adverse Renal Effects of Immune Checkpoint Inhibitors: A Narrative Review	Am J Nephrol	4.2	236
9	Dimopoulos et al.	2008	Pathogenesis and treatment of renal failure in multiple myeloma	Leukemia	11.4	221
10	Fudaba et al.	2006	Myeloma responses and tolerance following combined kidney and nonmyeloablative marrow transplantation: *in vivo* and *in vitro* analyses	Am J Transplant	8.8	218

therapy, also cause kidney injury. Kidney injury in CAR-T most commonly occurs as AKI, which is related to hypoperfusion and the inflammatory effects of released cytokines [14]. In addition, agents for targeted therapies, including anaplastic lymphoma kinase inhibitors (e.g., brigatinib and ceritinib), cyclin-dependent kinase 4/6 inhibitors (e.g., abemaciclib and ribociclib), BRAF inhibitors (e.g., vemurafenib) and poly(ADP–ribose) polymerase inhibitors (e.g., olaparib), have been found to cause pseudo-AKI by reducing renal tubular secretion of creatinine, resulting in increased serum creatinine [15].

4.2.2. Chemotherapeutic nephrotoxicity

Anticancer agents are increasingly recognized as major causes of acute and chronic kidney injury. Clinically, the manifestations of onconephrology take multiple forms, such as AKI, CKD, electrolyte disturbances, Fanconi's syndrome, onco-hypertension, proteinuria/nephrotic syndrome, renal cysts, and TMA. Our bibliometric analysis of keywords suggests that AKI appears to receive the most attention.

Epidemiologic studies have suggested that the incidence of AKI in cancer patients ranges from 24% (95% CI 17–30%) to 52% (95% CI 34–70%) [16], highlighting that the incidence of AKI in cancer patients may be closely related to coexisting comorbidities, cancer type and stage, and specific treatment regimens. A nationwide cohort study of 3,120 children, 16,310 adults, and 3,802 hospitalized elderly patients revealed that the overall incidence of AKI was 4.9%, most commonly observed in genitourinary, hematological, and neuro-musculoskeletal cancers and caused by purine analogs, folic acid analogs, and combinations of antineoplastic agents [17]. The causes of AKI in cancer patients are heterogeneous and can be broadly categorized into cancer-related factors and therapy-related factors. Cancer-related factors include urinary tract obstruction, metabolic disorders, glomerular disease, and hemodynamic alterations, whereas therapy-related factors include radiation, immunotherapy, targeted therapy, chemotherapy, hematopoietic stem cell transplantation, tumor lysis syndrome, and TMA. A retrospective study of 67,986 cancer patients revealed that stages 1, 2, and 3 AKI were

Table 2. The top 10 most cited references in the field of onconephrology between 1 January 2000 and 27 April 2024.

Rank	First author	Publication year	Document title	Journal	Impact factor	Citation number
1	Cockcroft et al.	1976	Prediction of creatinine clearance from serum creatinine	Nephron	2.5	146
2	Bladé et al.	1998	Renal failure in multiple myeloma: presenting features and predictors of outcome in 94 patients from a single institution	Arch Intern Med	NA	133
3	Knudsen et al.	2000	Renal failure in multiple myeloma: reversibility and impact on the prognosis. Nordic Myeloma Study Group	Eur J Haematol	3.1	124
4	Levey et al.	2009	A new equation to estimate glomerular filtration rate	Ann Intern Med	39.2	122
5	Levey et al.	1999	A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group	Ann Intern Med	39.2	106
6	Kyle et al.	2003	Review of 1027 patients with newly diagnosed multiple myeloma	Mayo Clin Proc	8.9	105
7	Dimopoulos et al.	2010	Renal impairment in patients with multiple myeloma: a consensus statement on behalf of the International Myeloma Working Group	J Clin Oncol	45.3	102
8	Launay-Vacher et al.	2007	Prevalence of Renal Insufficiency in cancer patients and implications for anticancer drug management: the renal insufficiency and anticancer medications (IRMA) study	Cancer	6.2	88
9	Dimopoulos et al.	2008	Pathogenesis and treatment of renal failure in multiple myeloma	Leukemia	11.4	80
10	Clark et al.	2005	Plasma exchange when myeloma presents as acute renal failure: a randomized, controlled trial	Ann Intern Med	39.2	76

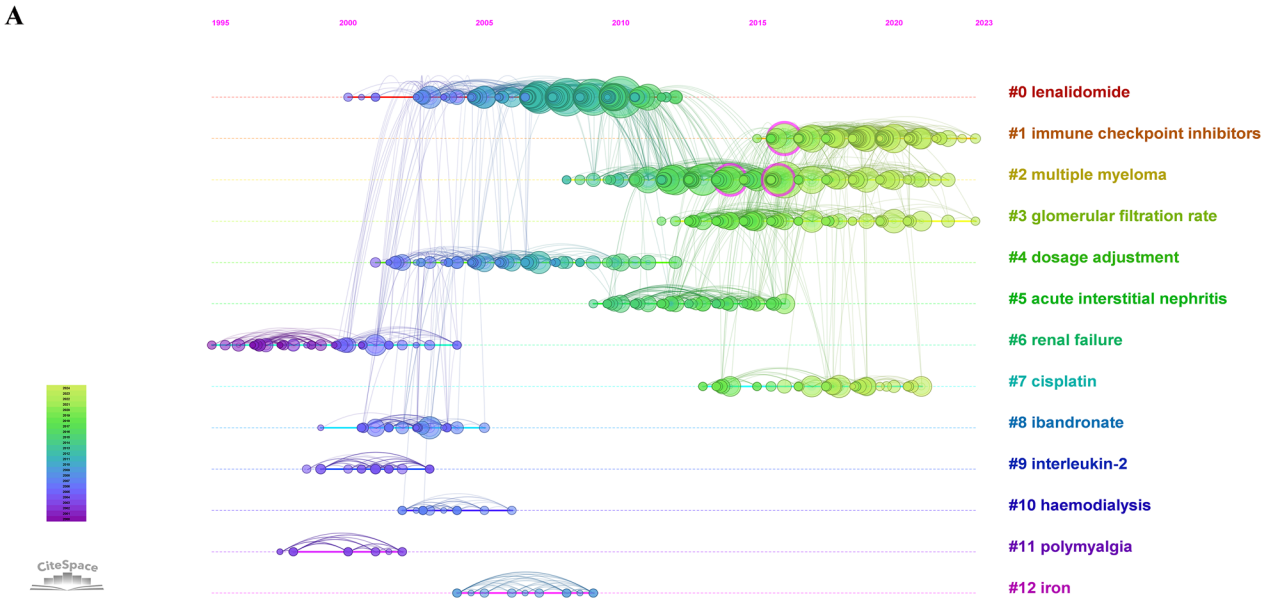
associated with 18.3% (95% CI 1.145–1.221), 71.0% (95% CI 1.629–1.796), and 100.0% (95% CI 1.910–2.095) increased risk of all-cause mortality, respectively [18]. Therefore, sensitive biomarkers for the timely detection and diagnosis of AKI in cancer patients are urgently needed for early intervention to improve patient outcomes.

4.2.3. Kidney function assessment

Accurate assessment of kidney function in cancer patients is important for risk stratification and appropriate dosing of chemotherapeutic agents, antibiotics, opioid analgesics, and other medications. Dose adjustment on the basis of kidney function is often required for a variety of common chemotherapeutic agents, such as alkylating agents, antimicrotubule agents, platinum agents, antimetabolites, immunomodulatory agents, and proteasome inhibitors. Although assessment with radiopharmaceuticals, such as chromium-51-ethylenediaminetetraacetic acid (^{51}Cr-EDTA, not available in the US) or technetium-99m-diethylenetriaminepentaacetic acid (^{99}mTc-DTPA) is considered the gold standard for kidney function, these methods are time-consuming, costly, and labor-intensive, making them impractical to perform on a regular basis. In comparison, estimation of the kidney glomerular filtration rate by serum creatinine and/or cystatin C-based formulas, such as the Cockcroft-Gault equation, the Modification of Diet in Renal Disease equation, the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, and the race-free formula calculated on the basis of both creatinine and cystatin C [19], appears to be more convenient and practical. Nonetheless, the clinical use of these equations may be biased by various confounding factors in cancer patients, such as anorexia, muscle wasting, and nutritional status. Moreover, validation of these equations in cancer patients is limited by the fact that most involve specific types of cancer in a limited number of patients. In the largest validation cohort of 2471 survivors with various types of cancer, Janowitz et al. validated these formulas against the ^{51}Cr-EDTA method and reported that the body surface area-adjusted CKD-EPI formula is the most accurate [20]. More recently, cancer-specific formulas have been proposed and developed. For example, Williams and colleagues developed an eGFR formula called 'CamGFR v2' based on either standardized or nonstandardized serum creatinine, which outperformed currently available formulas in 7,240 cancer patients [21]. Currently, there is no consensus regarding the best formula for cancer patients, and a prospective clinical trial is needed. In cases where significant discrepancies (differences >10% or >10 mL/min/1.73 m^2) were noted when different formulas were used, dose adjustment based on the lower estimated glomerular filtration rate result may be considered, especially for chemotherapeutic agents with dramatic nephrotoxicity with a narrow therapeutic range or in vulnerable patient populations.

4.2.4. CKD in cancer patients and chemotherapy dosing

The reported prevalence of CKD in patients with cancer ranges from 12 to 25%, depending on the exact cancer



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Top 25 References with the Strongest Citation Bursts

References	Year	Strength	Begin	End	2000 - 2024
Chanan-Khan AA, 2007, BLOOD, V109, P2604, DOI 10.1182/blood-2006-09-046409, DOI	2007	13.82	**2008**	2012	
Kastritis E, 2007, HAEMATOLOGICA, V92, P546, DOI 10.3324/haematol.10759, DOI	2007	12.51	**2008**	2012	
Ludwig H, 2007, HAEMATOLOGICA, V92, P1411, DOI 10.3324/haematol.11463, DOI	2007	9.47	**2008**	2012	
Dimopoulos MA, 2008, LEUKEMIA, V22, P1485, DOI 10.1038/leu.2008.131, DOI	2008	16.78	**2009**	2013	
San-Miguel JF, 2008, LEUKEMIA, V22, P842, DOI 10.1038/sj.leu.2405087, DOI	2008	10.86	**2009**	2013	
Eleutherakis-Papaiakovou V, 2007, LEUKEMIA LYMPHOMA, V48, P337, DOI 10.1080/10428190601126602, DOI	2007	9.06	**2009**	2012	
Hutchison CA, 2009, CLIN J AM SOC NEPHRO, V4, P745, DOI 10.2215/CJN.04590908, DOI	2009	15.23	**2010**	2014	
Leung N, 2008, KIDNEY INT, V73, P1282, DOI 10.1038/ki.2008.108, DOI	2008	10.13	**2010**	2013	
Dimopoulos MA, 2009, CLIN LYMPHOMA MYELOM, V9, P302, DOI 10.3816/CLM.2009.n.059, DOI	2009	10.13	**2010**	2013	
Ludwig H, 2010, J CLIN ONCOL, V28, P4635, DOI 10.1200/JCO.2010.28.1238, DOI	2010	11.16	**2011**	2015	
Roussou M, 2010, LEUKEMIA RES, V34, P1395, DOI 10.1016/j.leukres.2010.04.024, DOI	2010	9.08	**2011**	2015	
Haynes RJ, 2010, NEPHROL DIAL TRANSPL, V25, P419, DOI 10.1093/ndt/gfp488, DOI	2010	9.03	**2011**	2014	
Dimopoulos MA, 2010, J CLIN ONCOL, V28, P4976, DOI 10.1200/JCO.2010.30.8791, DOI	2010	19.98	**2012**	2015	
Hutchison CA, 2012, NAT REV NEPHROL, V8, P43, DOI 10.1038/nrneph.2011.168, DOI	2012	10.24	**2012**	2017	
Hutchison CA, 2011, J AM SOC NEPHROL, V22, P1129, DOI 10.1681/ASN.2010080857, DOI	2011	9.91	**2012**	2016	
Dimopoulos MA, 2013, LEUKEMIA, V27, P423, DOI 10.1038/leu.2012.182, DOI	2013	13.23	**2013**	2018	
Hutchison CA, 2012, NEPHROL DIAL TRANSPL, V27, P3823, DOI 10.1093/ndt/gfr773, DOI	2012	9.06	**2013**	2017	
Dimopoulos MA, 2014, ANN ONCOL, V25, P195, DOI 10.1093/annonc/mdt483, DOI	2014	10.29	**2015**	2019	
Rajkumar SV, 2014, LANCET ONCOL, V15, PE538, DOI 10.1016/S1470-2045(14)70442-5, DOI	2014	9.72	**2016**	2019	
Dimopoulos MA, 2016, J CLIN ONCOL, V34, P1544, DOI 10.1200/JCO.2015.65.0044, DOI	2016	15.7	**2017**	2021	
Cortazar FB, 2016, KIDNEY INT, V90, P638, DOI 10.1016/j.kint.2016.04.008, DOI	2016	10.9	**2017**	2021	
Bridoux F, 2017, JAMA-J AM MED ASSOC, V318, P2099, DOI 10.1001/jama.2017.17924, DOI	2017	9.8	**2018**	2022	
Rosner MH, 2017, NEW ENGL J MED, V376, P1770, DOI 10.1056/NEJMra1613984, DOI	2017	9.8	**2018**	2022	
Cortazar FB, 2020, J AM SOC NEPHROL, V31, P435, DOI 10.1681/ASN.2019070676, DOI	2020	12.11	**2020**	2024	
Seethapathy H, 2019, CLIN J AM SOC NEPHRO, V14, P1692, DOI 10.2215/CJN.00990119, DOI	2019	9.77	**2021**	2024	

Figure 5. (A) Timeline view of the most highly cited references in the field of onconephrology. Each node on the horizontal axis represents a highly cited reference, and the size of each node reflects the frequency of that reference. References with the same cluster are positioned on the same horizontal line, and different colors denote different reference clusters. (B) Top 25 references with the strongest citation bursts in the field onconephrology. The line represents the time axis, with the red part indicating the start year, end year, and duration of the reference burst.

type and associated demographic factors [22]. In an analysis of 5,831 biopsy-proven cancer patients, Ciorcan et al. demonstrated that cancers of the kidney, urinary tract, and pancreas had the highest prevalence of CKD, defined as an estimated glomerular filtration rate <60 mL/min/1.73 m^2 [22]. Although the majority of CKD cases in cancer patients are modest in degree, the presence of diabetes and hypertension also synergistically worsens patient survival. It has been unequivocally demonstrated that the presence of CKD, especially stage 3–5 CKD, is related to a 27% (95% CI 1.00–1.60) increased mortality rate compared with those with estimated glomerular filtration rate >60 mL/min/1.73 m^2 [23].

A critical and practical clinical issue related to CKD is dose adjustment for most chemotherapeutic agents. The prerequisite is accurate measurement of the glomerular filtration rate, which can have a dramatic impact on chemotherapeutic pharmacokinetics and drug toxicity. Studies have shown that the incidences of AKI or AKI transition to CKD are significantly increased in those receiving higher doses of preconditioning chemotherapy [24]. A comprehensive review of the exact dosing regimens of anticancer drugs

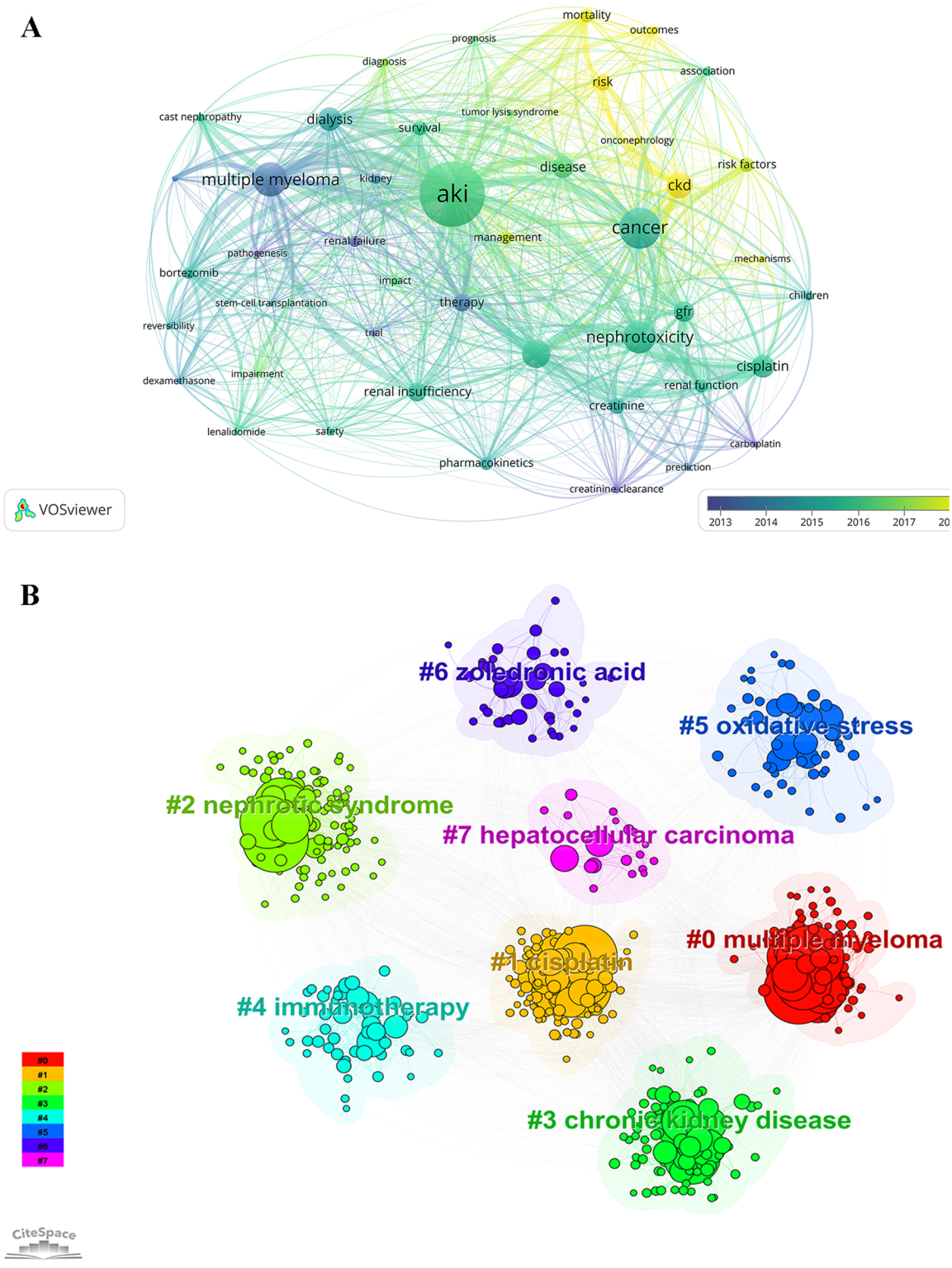

Figure 6. (A) Most frequent keywords in the field of onconephrology analyzed with the VOSviewer software. The size of the nodes and width of the connecting line represent the frequency of the keywords and the color of the node denotes the time frame. (B) Cluster analysis of keywords in the field of onconephrology. Keywords with similar meanings were grouped into clusters.

is beyond the scope of this study, but a brief discussion of the dosage adjustment of chemotherapeutic medications with different renal functions is essential to highlight their nuanced relationship. For example, reducing the dose of cisplatin by 50–70% in patients with a creatinine clearance rate <50 mL/min/1.73 m^2, and by 75% for capecitabine in patients with a creatinine clearance rate between 30 and 50 mL/min/1.73 m^2, is recommended [25]. Nonetheless, no dose adjustments are required for vascular endothelial growth factor inhibitors, epithelial growth factor receptor inhibitors,

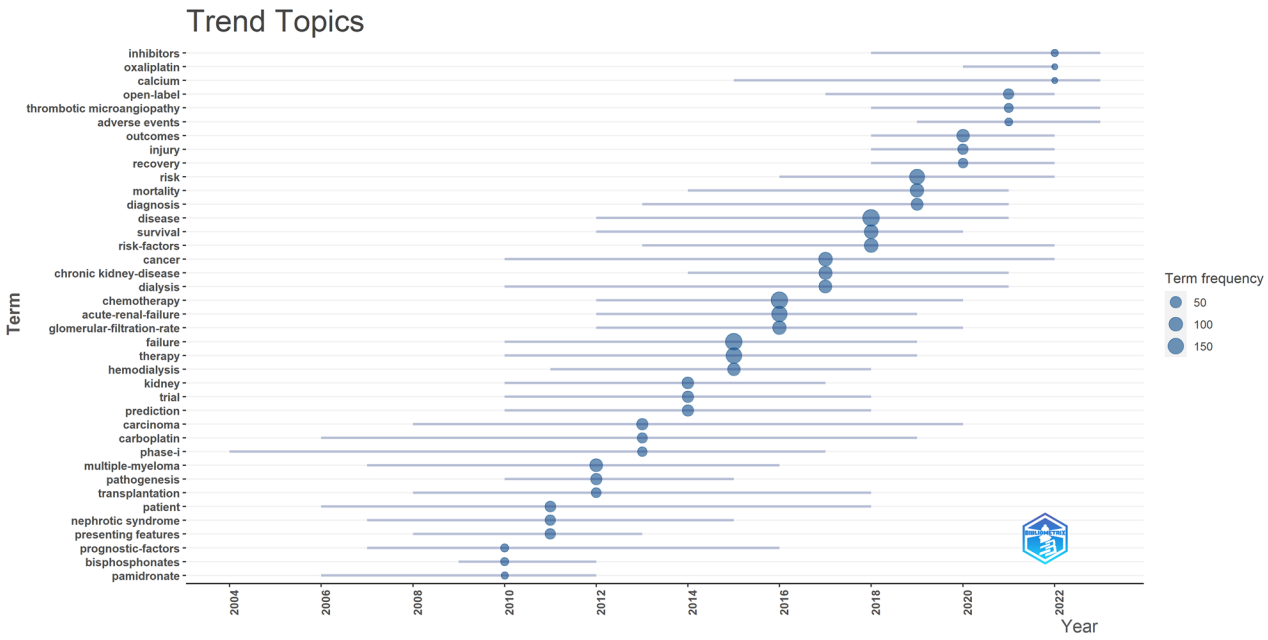

Figure 7. Results of the analysis of trend topics created by the R package bibliometrix. The size of each node corresponds to the frequency of the trend topic, and the horizontal line of each node denotes the duration of that trend topic.

Top 25 Keywords with the Strongest Citation Bursts

Keywords	Year	Strength	Begin	End	2000 - 2024
renal failure	2000	21.91	**2000**	2010	
nephrotic syndrome	2000	12.09	**2000**	2011	
glomerulonephritis	2000	6.13	**2000**	2011	
leukemia	2000	6.08	**2000**	2011	
myeloma	2001	6.75	**2001**	2013	
creatinine clearance	2001	6.08	**2001**	2009	
focal segmental glomerulosclerosis	2002	6.2	**2002**	2014	
patient	2003	7.03	**2003**	2011	
thalidomide	2004	7.81	**2004**	2013	
trial	2000	6.53	**2007**	2014	
bisphosphonates	2007	6.42	**2007**	2012	
pathogenesis	2006	10.78	**2008**	2015	
plasma-exchange	2008	6.31	**2008**	2014	
reversibility	2009	11.45	**2009**	2016	
presenting features	2001	9.73	**2010**	2013	
bortezomib	2006	8.42	**2011**	2014	
stem cell transplantation	2001	6.27	**2012**	2016	
outcm	2016	11.03	**2018**	2024	
onconephrology	2016	7.13	**2018**	2024	
aki	2014	6.15	**2019**	2024	
risk	2007	5.96	**2019**	2022	
immunotherapy	2020	6.43	**2020**	2024	
acute kidney injury	2008	15.23	**2021**	2024	
risk factors	2007	9.55	**2022**	2024	
chronic kidney disease	2007	6.58	**2022**	2024	

Figure 8. Top 25 keywords with the strongest citation bursts in the field of onconephrology created with the CiteSpace software. The red line represents the start year and end year for each keyword. The strength indicates the frequency of citation bursts, with a higher value indicating higher frequency of the burst keyword's appearance during a specific period.

PD-1/PD-L1 antagonists, or cytotoxic T-lymphocyte antigen 4 antagonists. In addition, other strategies in addition to dose adjustment may be used to mitigate nephrotoxicity associated with anticancer agents. For example, teneligliptin, a dipeptidyl peptidase-4 inhibitor, has been shown *in vivo* to attenuate cisplatin-induced nephrotoxicity and improve renal function by accelerating tubular regeneration and reducing injury and fibrosis [26].

4.2.5. Glomerular diseases associated with cancer

Glomerular disease in cancer refers to the concurrent or metachronous occurrence of glomerular disease, most commonly manifested as proteinuria with or without elevated serum creatinine, in cancer. Although various patterns of glomerular injury have been described in the literature, the prototypical example is cancer-associated membranous nephropathy, which is largely mediated by antibodies to thrombospondin type 1 domain-containing 7A [27]. Epidemiologic studies suggest that the incidence of cancer in membranous nephropathy is 10% (95% CI 6.1–14.6%), with cancer detected before or at the time of membranous nephropathy diagnosis in ~80% of cases [28]. The mechanistic link is hypothesized to be subepithelial deposition of tumor antigens associated with an enhanced immune response targeting the tumor. In contrast to idiopathic membranous nephropathy, which is histologically characterized by the IgG4 subclass, cancer-associated membranous nephropathy is characterized predominantly by the IgG1 and IgG2 subclasses. Other reported patterns of glomerular disease in solid tumors include pauci-immune glomerulonephritis, renal vasculitis, podocytopathy, C3 glomerulopathy, rapidly progressive glomerulonephritis, IgA nephropathy, and membranoproliferative glomerulonephritis [29].

4.2.6. Electrolyte disorders

Electrolyte abnormalities are common in both solid and hematopoietic malignancies, most commonly as chemotherapy-induced, tumor-related sequelae. Common electrolyte disturbances observed in cancer patients include hyponatremia, hypokalemia/hyperkalemia, hypocalcemia/hypercalcemia, hyperphosphatemia/hypophosphatemia, and hypomagnesemia. Hyponatremia is the most common type of electrolyte disturbance and is most commonly caused by decreased appetite, anorexia, and vomiting, and inappropriate secretion of antidiuretic hormone by many malignancies and chemotherapeutic regimens. Hyperkalemia is mostly caused by tumor lysis syndrome, an oncologic emergency in which tumors lyse spontaneously or secondary to anticancer therapies with sudden release of intracellular ions and metabolites into the systemic circulation. In fact, in addition to hyperkalemia, tumor lysis syndrome may also include hyperphosphatemia, hypocalcemia, hyperuricemia, and AKI. Hypercalcemia in cancer patients is often caused by extensive cytokine-mediated osteolysis or the release of parathyroid hormone-related peptides. The appearance of zoledronic acid as a keyword cluster suggests that attention has been given to the treatment of hypercalcemia, which can often be an onconephrology emergency. Hypophosphatemia is often associated with platinum-based chemotherapy, especially cisplatin. Hypomagnesemia is often associated with the administration of monoclonal antibodies targeting epidermal growth factor receptor, particularly cetuximab, due to increased urinary magnesium loss, as magnesium reabsorption in the distal tubule is partially dependent on the activity of epidermal growth factor receptor proteins located on the basolateral tubular membrane [30].

4.2.7. Immunotherapy

CAR-T cell therapy is a type of immunotherapy that represents a revolutionary treatment for hematological malignancies using genetically engineered host T cells. Although CAR-T cell therapy been shown to induce durable remission with reduced mortality and prolonged survival [31], it can potentially cause cytokine release syndrome, which can be observed in up to 90% of patients receiving CD19 CAR-T cells for B-cell acute lymphoblastic leukemia and non-Hodgkin B-cell lymphoma [32]. A recent meta-analysis indicated that the pooled estimated incidences of AKI and AKI requiring renal replacement therapy following CAR-T cell therapy were 18.6% (95% CI 14.3–23.8%) and 4.4% (95% CI 2.1–8.9%), respectively [33]. The cumulative incidence at day 100 was 21.7% (95% CI 9.7–33.8%) for grade 1 AKI and 8.7% (95% CI 0.4–17%) for grade 2–3 AKI [34], suggesting that AKI in patients receiving CAR-T cell therapy is mostly transient, mild in severity, and associated with rapid recovery [35]. Nevertheless, 75 and 67% of patients who experienced AKI after CAR-T cell therapy still had CKD at the 6- and 12-month follow-ups, respectively [36]. Potential mechanisms for AKI after CAR-T cell infusion include capillary leakage with hypovolemia, tumor lysis syndrome, and immune effector cell-associated neurotoxicity syndrome (ICANS).

Immune checkpoint inhibitors are monoclonal antibodies that selectively block intrinsic down-regulating receptors of the immune system to activate suppressed T cells to enhance anti-tumor-directed immune responses [37]. As reflected in the keywords trend analysis, immune checkpoint inhibitors have become widely used and considered standard of care in the management of many advanced cancers. AKI has been increasingly recognized as an uncommon but important form of kidney toxicity following the use of immune checkpoint inhibitors. A multi-center analysis revealed that AKI typically manifests at a median of 16 weeks following the initiation of immune checkpoint inhibitors, and kidney recovery was observed in 64.3% of patients at a median of 7 weeks following the onset of AKI [38].

The underlying pathophysiology and corresponding treatments for AKI associated with CAR-T therapies and immune checkpoint inhibitors were different. Kidney injury in CAR-T is mostly related to hemodynamic changes and is typically transient and mild in severity, with only a small percentage of patients requiring continuous renal replacement therapy [39]. Interestingly, studies have shown that preexisting CKD

does not appear to have a significant impact on the safety, efficacy, or patient outcomes of CAR-T cell therapy [40]. In comparison, acute tubulointerstitial nephritis is the most common cause of immune checkpoint inhibitor-associated AKI. Manohar et al. reported that prompt withholding of immune checkpoint inhibitors along with steroid use resulted in a complete response in 63% of patients [41]. In addition, rechallenge in 4 patients was successful in 3 patients and the other patient developed recurrent acute allergic interstitial nephritis and fatal pneumonitis [41].

Electrolyte abnormalities are extremely common in patients receiving immunotherapy and most commonly present as hyponatremia, hypokalemia, hypercalcemia/hypocalcemia, hyperphosphatemia/hypophosphatemia, and hypomagnesemia. A meta-analysis of 48 clinical trials revealed a pooled risk ratio of 1.67% (95% CI 0.89–3.12) for electrolyte disorders in cancer patients receiving PD-1 inhibitors [42]. In a recent real-world study of 2,458 patients, Seethapathy's group reported that 62% of patients had hyponatremia and that 6% had severe hyponatremia with sodium <124 mmol/L [43]. There have been an increasing number of case reports highlighting hypophysitis and secondary adrenal insufficiency as the underlying cause of hyponatremia in patients receiving immune checkpoint inhibitors [44]. Another common electrolyte abnormality in cancer patients receiving immunotherapy is hypercalcemia, which may be caused by immune endocrinopathies, parathyroid hormone-related peptide release, cancer hyperprogression, and sarcoidosis-like granulomas [45].

Notable side effects associated with CAR-T cell therapy include cytokine release syndrome, tumor lysis syndrome, and ICANS. Patients with cytokine release syndrome often have nonspecific symptoms of fever, arthralgia, malaise and fatigue, anorexia, and tachycardia. Therapeutic approaches for cytokine release syndrome include supportive care and pharmacological intervention with the monoclonal anti-interleukin-6 antibody tocilizumab. There are also agents currently under investigation for the prophylaxis of cytokine release syndrome, such as tocilizumab, anakinra, teclistamab, and duvelisib [46]. Tumor lysis treatment is an oncological emergency resulting from the release of intracellular electrolytes and nucleic acids from malignant cells. Treatment and prophylaxis for tumor lysis syndrome are similar and include aggressive intravenous hydration, the use of xanthine oxidase inhibitors and rasburicase, and medical management of associated electrolyte abnormalities. ICANS is a constellation of neuropsychiatric symptoms, including headache, aphasia, seizures, or decreased consciousness, that are observed in 20–70% of CAR-T cell therapy recipients. Risk factors for the occurrence of ICANS include cytokine release syndrome, high tumor burden, advanced age, and a strong inflammatory response [47]. Antiepileptic medications, steroids, and tocilizumab have been the most common treatments for ICANS, especially for those with concurrent cytokine release syndrome. Preliminary studies investigating the interleukin-1 receptor antagonist anakinra for the prophylaxis of ICANS have reported encouraging results [48].

4.2.8. Underlying pathophysiology

The investigation of the molecular mechanisms underlying nephrotoxicity caused by various anticancer drugs or treatments is particularly important, as it may shed light on prevention and intervention strategies. Moreover, the exact underlying pathways for anticancer drug-induced renal injury may differ depending on the exact type of agent. For example, cisplatin, a classic and highly effective chemotherapeutic agent for a variety of malignancies, is well known for its potential risk of inducing nephrotoxicity. Various molecular mechanisms have been reported in the pathogenesis of cisplatin-induced nephrotoxicity, including tubular cell apoptosis under oxidative stress, renal interstitial inflammatory cell infiltration leading to AKI, and extensive production of proinflammatory cytokines [49]. Several lines of evidence also suggest that various signaling pathways are involved, including the toll-like receptor pathway, the NF-κB pathway, and the poly-ADP–ribose polymerase-1 pathway [50]. Currently, the mechanism of action for nephrotoxicity resulting from the majority of traditional chemotherapeutic agents has been described and is largely related to the side effects of the desired anticancer properties of these agents. Specifically, antimetabolites, such as gemcitabine have a vasoconstrictive effect on afferent renal arteries, resulting in a decreased glomerular filtration rate and AKI [51]. However, the mechanisms of action of targeted therapy-induced nephrotoxicity may extend beyond pharmacological action. For example, epidermal growth factor receptor inhibitors, such as cetuximab, cause electrolyte disturbances that can be explained by inhibited signaling at the distal convoluted tubule that regulates transepithelial magnesium transport. However, epidermal growth factor receptor inhibitors may also cause AKI, although the mechanisms of action remain unclear. Similarly, the exact mechanisms by which Her-2 inhibitors, anaplastic lymphoma kinase inhibitors, and BRAF inhibitors cause AKI remain elusive and warrant additional investigations.

4.3. Future research directions

4.3.1. TMA

TMA is a pathological term that is clinically characterized by microangiopathic hemolytic anemia and thrombocytopenia and is associated with elevated lactate dehydrogenase and end-organ failure [52]. The emergency of TMA as a trend topic is consistent with increasing reports suggesting that TMA represents ~5.4% of the findings in kidney biopsies from cancer patients [53]. Unlike primary TMA–which is mostly caused by genetic mutations–cancers and related therapies, especially hematopoietic stem cell transplantation, are important causes of secondary TMA. On biopsy, the renal TMA is characterized by extensive formation of fibrin thrombi in the capillary loops and arterioles, intimal on ionization, fragmented red blood cells, and mesangiolysis. Common solid tumors that can induce TMA include gastrointestinal, lung, genitourinary, and hepatobiliary cancers. In a series of 168 cases of cancer-related TMA, Lechner et al. reported that lymphoma accounted for ~8.3% of all cases [54].

Cancer therapy-related TMA is also relatively common and can be observed in those treated with conventional chemotherapy, targeted therapy, and immunotherapy, especially in those with underlying genetic defects in the alternative complement cascade. Commonly used conventional chemotherapy drugs include mitomycin-C, gemcitabine, bleomycin, and platinum-based agents, whereas epidermal growth factor monoclonal antibody agents are the most common targeted therapy used to induce TMA [55]. Compared with conventional chemotherapy-induced TMA, which is often fulminant, systemic, and lethal, TMA caused by targeted therapy is generally confined to the kidney and has more favorable outcomes [56]. However, discontinuation of stimulating agents may not lead to renal recovery, and additional therapeutic maneuvers, such as plasma exchange and rituximab administration, may have variable clinical efficacy [57]. However, currently, all available evidence comes from case reports or small case series. The American Society for Apheresis guidelines consider therapeutic plasma exchange to be ineffective for treating chemotherapy-related TMA [58]. In the largest cohort of gemcitabine-associated TMA, Daviet et al. reported that 54.7% of patients eventually died and that treatment with therapeutic plasma exchange did not improve outcomes compared with glucocorticoids and was associated with more adverse events [59]. Eculizumab, a monoclonal antibody targeting terminal complement C5, has been shown in several case reports to promote renal recovery in cancer therapy-associated TMA [60]. In a retrospective analysis of 12 cases of gemcitabine-induced TMA, Grall et al. showed that 17 and 67% achieved complete and partial recovery of renal function, respectively, after treatment with eculizumab [61]. These increasing results encourage the conduct of prospective studies in the future to conclusively determine the efficacy of eculizumab in the treatment of cancer-related TMA.

4.3.2. Clinical trials

Our trend topic analysis revealed that clinical trials are likely to be a hot topic in onconephrology in the future. We believe this raises 2 considerations. First, this may imply the recruitment of patients with impaired renal function into cancer clinical trials. Clearly, obtaining accurate information on the safety and effective dosing of chemotherapeutic agents in cancer patients with CKD is critical for formulating appropriate treatment regimens. Unfortunately, the majority of cancer clinical trials have enrolled patients with normal or mildly impaired renal function, thus underrepresenting patients with severely impaired renal function. For example, in a recent meta-analysis of 11,066 participants from 32 clinical trials receiving combination therapy with vascular endothelial growth factor pathway inhibitors and immune checkpoint inhibitors, Elyan et al. reported that all trials excluded patients with advanced CKD, and few trials included people with proteinuria [62]. Butrovich et al. reported that pharmacokinetic analyses in patients with CKD stages 4–5 and hemodialysis were performed for only 29 and 6% of the 55 drugs approved between 2015 and 2019, respectively [63]. The reasons for the exclusion of this specific patient population are complex, including sponsor concerns, safety concerns, and the lack of a robust nephrology clinical trial infrastructure [64].

Another consideration is the design and application of specific clinical trials for specific onconephrology conditions or issues. Eculizumab or other novel treatments for cancer-associated TMA appear to be good examples of such clinical trials. Another area of interest is the design of prospective and controlled trials to evaluate the efficacy of renoprotective agents during cancer treatment. Successful trials of renoprotective agents would not only provide effective chemotherapy with minimized risk of renal toxicity but also offer chemotherapeutic options for cancer patients with preexisting renal impairment. For example, sirtuins, which are NAD+-dependent deacetylases with important antioxidant activity, have been demonstrated in animal studies to exert cytoprotective and renoprotective effects in various forms of nephrotoxicity [65]. Thus, clinical trials evaluating their ability to reduce nephrotoxicity during cancer treatment are very meaningful.

5. Study limitations

The present study has several limitations that should be acknowledged. First, we only used the SCIE-WOS database for bibliometric analysis, which may have missed important studies indexed in other databases, thus limiting the comprehensiveness of the findings. Previous studies have indicated that the Scopus database has more citations per article and better coverage than the SCIE-WOS database [66]. Extending the search for articles to other databases, such as Scopus and Google Scholar, may provide a more comprehensive overview of progress in the field of onconephrology. Second, the inclusion of English language-only documents obviously discriminated against countries, institutions, or authors publishing research in other languages. Third, we did not evaluate literature quality, which is often done in meta-analyses. Finally, the concept of onconephrology is constantly evolving, and some scholars have included an increased risk of cancer in kidney transplant patients in the field of onconephrology [67], a topic that was not covered in the present study.

6. Conclusions

In summary, this bibliometric study revealed that the field of onconephrology is gradually gaining momentum in terms of both scientific output and the number of citations. The United States, University of Texas MD Anderson Cancer Center, Meletios A. Dimopoulos, and Nephrology Dialysis Transplantation were the most productive country, institution, author, and journal, respectively. Current research hotspots in onconephrology include chemotherapeutic nephrotoxicity, assessment of kidney function in cancer patients, CKD in cancer patients and chemotherapy dosing, glomerular diseases in cancer patients, electrolyte disorders, immunotherapy, and basic studies of the underlying molecular mechanisms of nephrotoxicity. Future directions in this field may include TMA and clinical trials.

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Author contributions

WW: data analysis, idea conception, manuscript writing, and editing; YWW: data collection, data analysis, manuscript writing; SLF: data analysis.

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Data availability statement

Data are provided within the Supplementary Files.

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