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June 2025

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JCR Year 2024

VIRCHOWS ARCHIV

ISSN

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EISSN

1432-2307

JCR ABBREVIATION

VIRCHOWS ARCH

ISO ABBREVIATION

Virchows Arch.

Journal information

EDITION

Science Citation Index Expanded (SCIE)

CATEGORY

PATHOLOGY

LANGUAGES	REGION	1ST ELECTRONIC JCR YEAR
English	GERMANY (FED REP GER)	1997

Publisher information

PUBLISHER	ADDRESS	PUBLICATION FREQUENCY
SPRINGER	ONE NEW YORK PLAZA, SUITE 4600 , NEW YORK, NY 10004, UNITED STATES	12 issues/year

Journal's performance

Journal Impact Factor

The Journal Impact Factor (JIF) is a journal-level metric calculated from data indexed in the Web of Science Core Collection. It should be used with careful attention to the many factors that influence citation rates, such as the volume of publication and citations characteristics of the subject area and type of journal. The Journal Impact Factor can complement expert opinion and informed peer review. In the case of academic evaluation for tenure, it is inappropriate to use a journal-level metric as a proxy measure for individual researchers, institutions, or articles. Learn more

2024 JOURNAL IMPACT FACTOR	JOURNAL IMPACT FACTOR WITHOUT SELF CITATIONS
3.1	2.9
View calculation	View calculation

Journal Impact Factor Trend 2024

Export

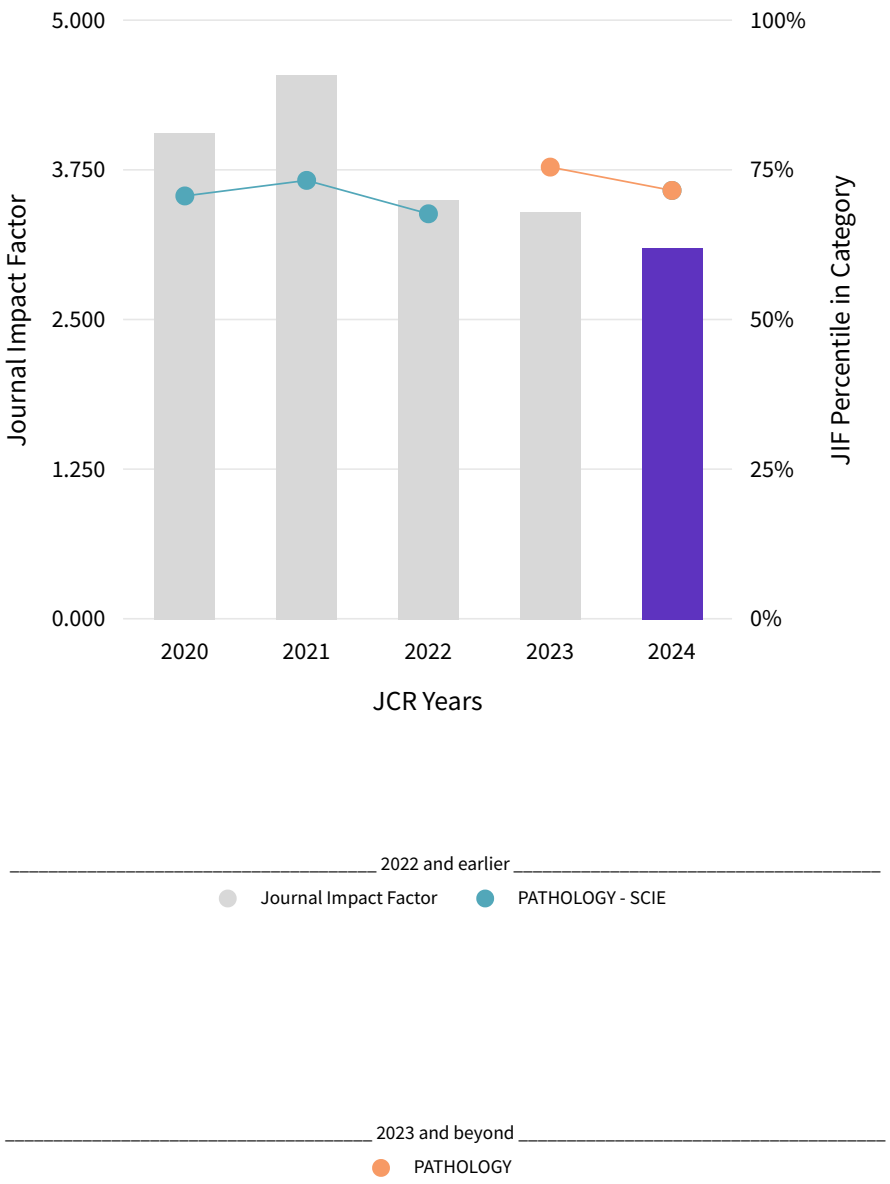
Journal Impact Factor contributing items

Export

Citable items (395) Citing Sources (491)

TITLE	CITATION COUNT
New concepts in EBV-associated B, T, and NK cell lymphoproliferative disorders	30
International Consensus Classification of acute lymphoblastic leukemia/lymphoma	27

15



View all years

The International Consensus Classification of acute myeloid leukemia	22
Standardized pathology report for HER2 testing in compliance with 2023…	21
Artificial intelligence-assisted cancer diagnosis improves the efficiency of…	20
Third International Consensus Conference on lesions of uncertain…	20
Updates on breast biomarkers	20
Plasma cell neoplasms and related entities-evolution in diagnosis and…	17
Comparison of HercepTest™ mAb pharmDx (Dako Omnis, GE001) with…	15
Diagnostic approaches and future directions in Burkitt lymphoma and hig…	14

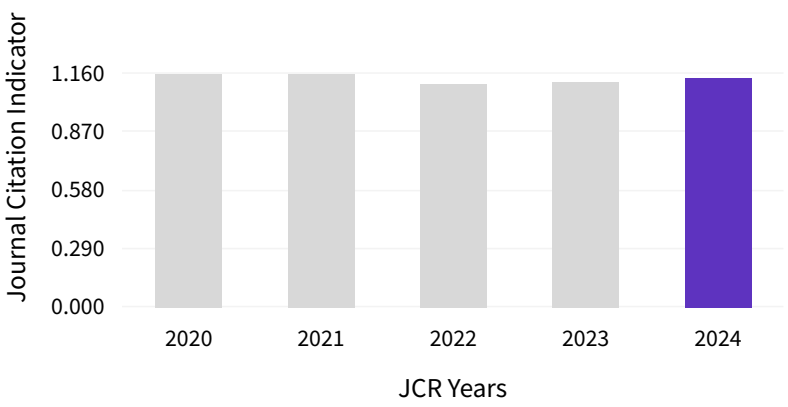
View All in Web of Science

Journal Citation Indicator (JCI)

Export

1.14

The Journal Citation Indicator (JCI) is the average Category Normalized Citation Impact (CNCI) of citable items (articles & reviews) published by a journal over a recent three year period. The average JCI in a category is 1. Journals with a JCI of 1.5 have 50% more citation impact than the average in that category. It may be used alongside other metrics to help you evaluate journals. Learn more



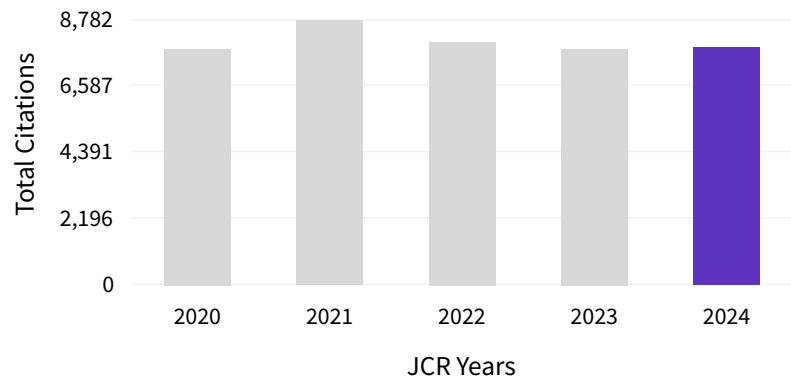
View all years

Total Citations

Export

7,888

The total number of times that a journal has been cited by all journals included in the database in the JCR year. Citations to journals listed in JCR are compiled annually from the JCR years combined database, regardless of which JCR edition lists the journal.



View all years

15

Citation distribution

Export

The Citation Distribution shows the frequency with which items published in the year or two years prior were cited in the JCR data year (i.e., the component of the calculation of the JIF). The graph has similar functionality as the JIF Trend graph, including hover-over data descriptions for each data point, and an interactive legend where each data element's legend can be used as a toggle. You can view Articles, Reviews, or Non-Citable (other) items to the JIF numerator. Learn more

ARTICLE CITATION MEDIAN

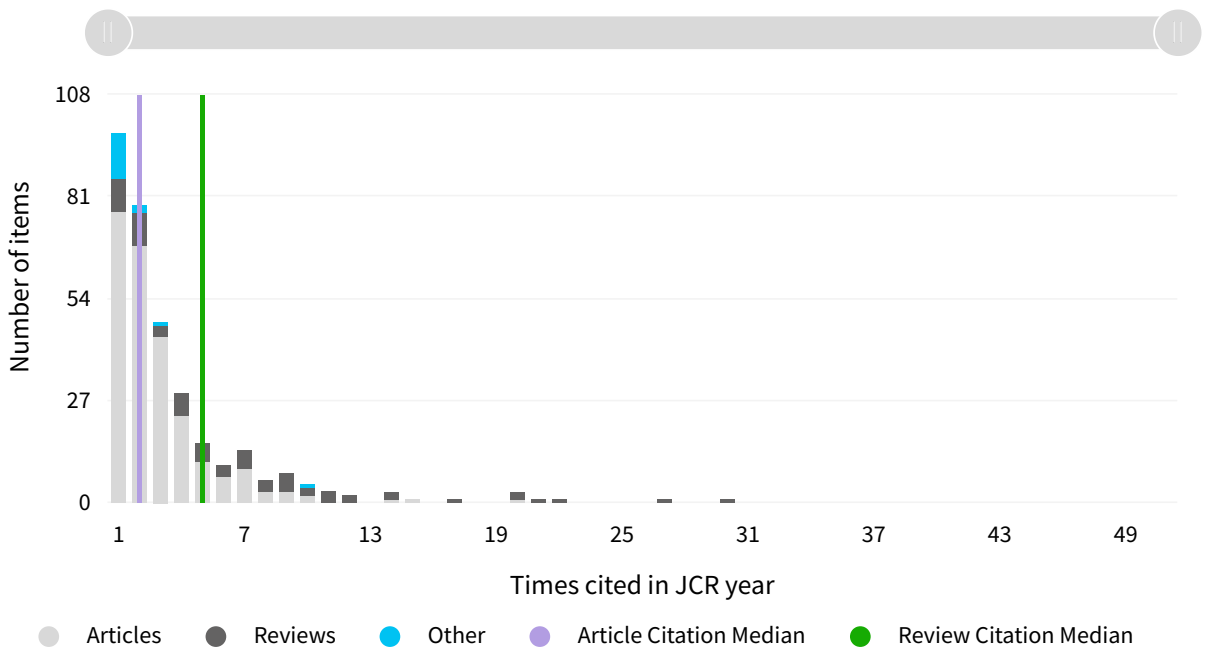
2

REVIEW CITATION MEDIAN

5

UNLINKED CITATIONS

16



TIMES CITED

0

ARTICLES

73

REVIEWS

8

OTHER

1,226

Open Access (OA)

Export

The data included in this tile summarizes the items published in the journal in the JCR data year and in the previous two years. This three-year set of published items is used to provide descriptive analysis of the content and community of the journal. Learn more

Items

TOTAL CITABLE	% OF CITABLE OA
640	40.63%

Citations*

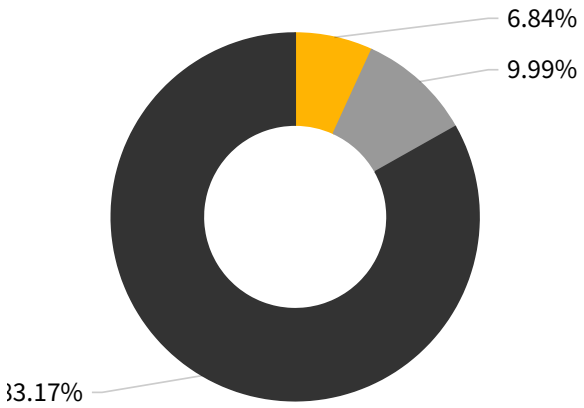
TOTAL CITABLE	% OF CITABLE OA
1,328	53.46%

15

CITABLE

GOLD OPEN ACCESS
260 / 6.84%

SUBSCRIPTION AND FREE TO READ
380 / 9.99%



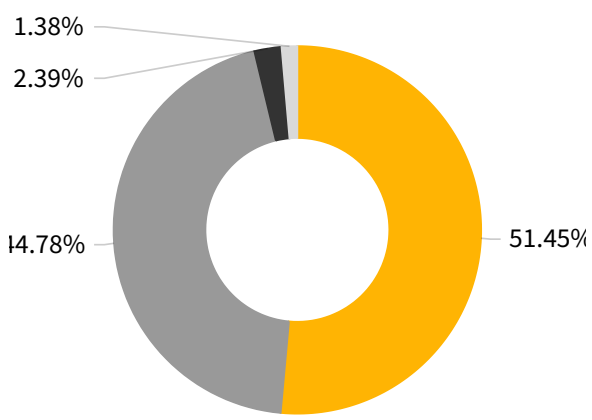
NON-CITABLE

OTHER (NON CITABLE ITEMS)
3,162 / 83.17%

CITABLE

GOLD OPEN ACCESS
710 / 51.45%

SUBSCRIPTION AND FREE TO READ
618 / 44.78%



NON-CITABLE

OTHER (NON CITABLE ITEMS)
33 / 2.39%

UNLINKED CITATIONS
19 / 1.38%

*Citations in 2024 to items published in [2022 - 2024]

Rank by Journal Impact Factor

Journals within a category are sorted in descending order by Journal Impact Factor (JIF) resulting in the Category Ranking below. A separate rank is shown for each category in which the journal is listed in JCR. Beginning in 2023, ranks are calculated by category.
Learn more

CATEGORY
PATHOLOGY

26/90

JCR YEAR	JIF RANK	JIF QUARTILE	JIF PERCENTILE
2024	26/90	Q2	71.7
2023	22/88	Q1	75.6

Rank by JIF before 2023 for PATHOLOGY

EDITION
Science Citation Index Expanded (SCIE)

JCR YEAR	JIF RANK	JIF QUARTILE	JIF PERCENTILE
2022	25/76	Q2	67.8
2021	21/77	Q2	73.38
2020	23/77	Q2	70.78
2019	24/78	Q2	69.87

Rank by Journal Citation Indicator (JCI)

15

Journals within a category are sorted in descending order by Journal Citation Indicator (JCI) resulting in the Category Ranking below. A separate rank is shown for each category in which the journal is listed in JCR. Data for the most recent year is presented at the top of the list, with other years shown in reverse chronological order. Learn more

CATEGORY

PATHOLOGY

19/90

JCR YEAR	JCI RANK	JCI QUARTILE	JCI PERCENTILE
2024	19/90	Q1	79.44
2023	17/88	Q1	81.25
2022	24/87	Q2	72.99
2021	20/89	Q1	78.09
2020	19/90	Q1	79.44
2019	22/89	Q1	75.84
2018	21/89	Q1	76.97

Citation network

Cited Half-life

6.7 years

The Cited Half-Life is the median age of the items in this journal that were cited in the JCR year. Half of a journal's cited items were published more recently than the cited half-life.

TOTAL NUMBER OF CITES

7,888

NON SELF-CITATIONS

7,623

SELF-CITATIONS

265

Citing Half-life

6.4 years

The Citing Half-Life is the median age of items in other publications cited by this journal in the JCR year.

TOTAL NUMBER OF CITES

8,453

NON SELF-CITATIONS

8,188

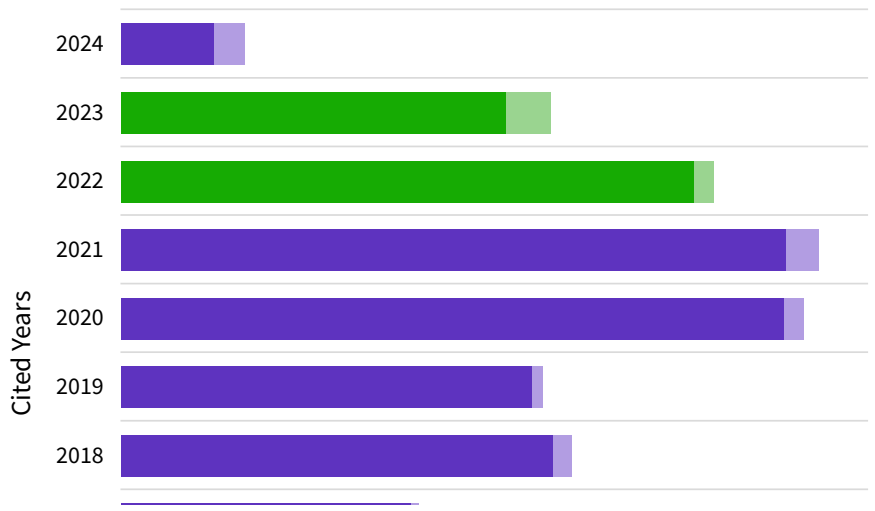
SELF-CITATIONS

265

Cited Half-life Data | Citing Half-life Data

Export

15

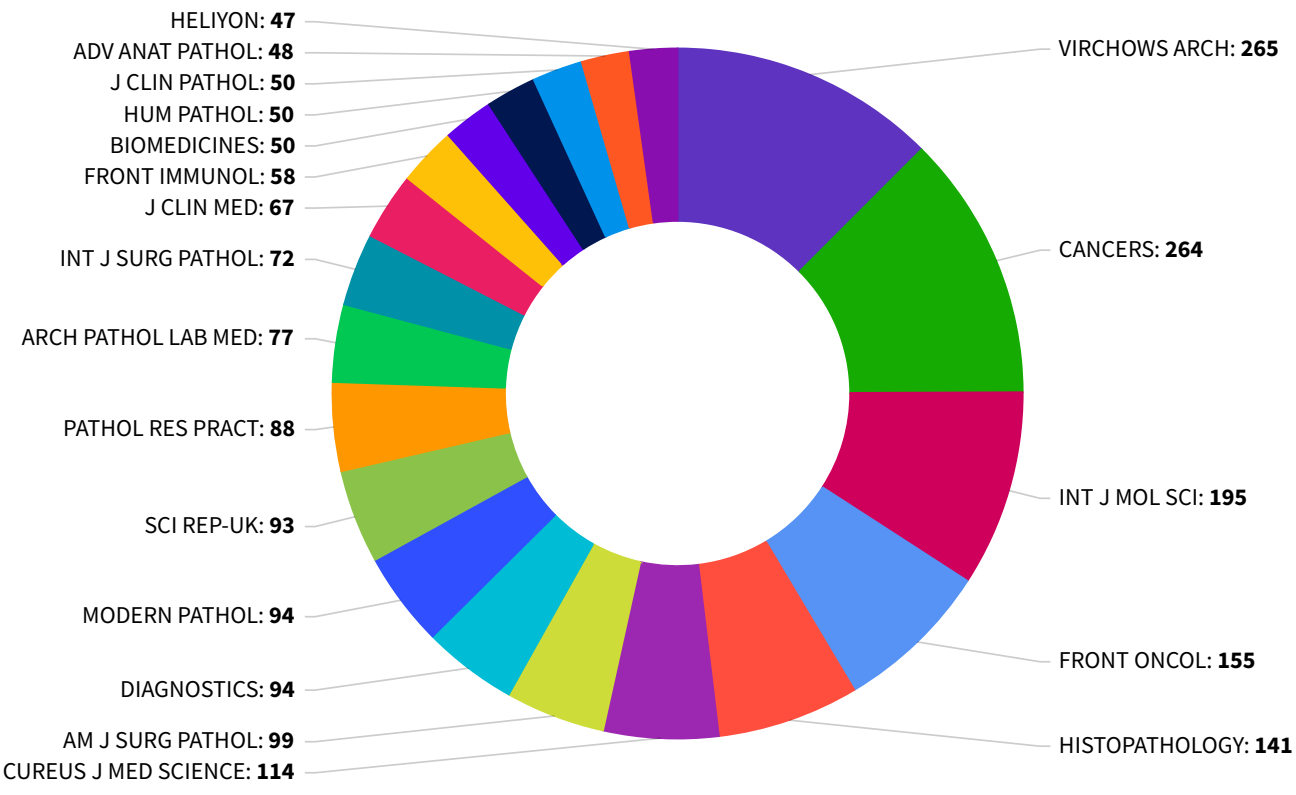


CITED YEAR	# OF CITES FROM 2024	CUMULATIVE %	# OF CITING SOURCES
All years	**7,888 citations**	**100.00%**	**1,801 sources**
2024	149 citations	1.89%	71 sources
2023	517 citations	8.44%	244 sources
2022	714 citations	17.49%	356 sources
2021	839 citations	28.13%	419 sources
2020	822 citations	38.55%	412 sources
2019	507 citations	44.98%	318 sources
2018	542 citations	51.85%	314 sources

Journal Citation Relationships

Cited Data | Citing Data

Top 20 journals citing VIRCHOWS ARCH by number of citations



Content metrics

Source data

This tile shows the breakdown of document types published by the journal. Citable Items are Articles and Reviews. For the purposes of calculating JIF, a JCR year considers the publications of that journal in the two prior years. Learn more

Average JIF Percentile

Export

The Average Journal Impact Factor Percentile takes the sum of the JIF Percentile rank for each category under consideration then calculates the average of those values. Learn more

15

245 total citable items

	ARTICLES	REVIEWS	COMBINED(C)	OTHER DOCUMENT TYPES(O)	PERCENTAG
NUMBER IN JCR YEAR 2024 (A)	215	30	245	1,920	11%
NUMBER OF REFERENCES (B)	6,272	2,071	8,343	110	99%
RATIO (B/A)	29.2	69.0	34.1	0.1	

ALL CATEGORIES AVERAGE
71.7

PATHOLOGY
71.7

Contributions by organizations

Export

Organizations that have contributed the most papers to the journal in the most recent three-year period. Learn more

RANK	ORGANIZATION	COUNT
1	UNIVERSITE DE TUNIS-EL-MANAR	100
2	CHARLES UNIVERSITY PRAGUE	73
3	UNIVERSITY OF ERLANGEN NUREMBERG	55
4	UNIVERSIDADE DE COIMBRA	52
5	UNIVERSITY OF BARCELONA	47
6	UNIVERSITE DE SFAX	43

Contributions by country/region

Export

Countries or Regions that have contributed the most papers to the journal in the most recent three-year period. Learn more

RANK	COUNTRY / REGION	COUNT
1	SPAIN	413
2	ITALY	340
3	USA	301
4	TURKIYE	252
5	PORTUGAL	246
6	TUNISIA	239
7	GERMANY (FED REP GER)	225
8	ROMANIA	168
9	RUSSIA	153

Additional metrics

Eigenfactor Score

0.00833

The Eigenfactor Score is a reflection of the density of the network of citations around the journal using 5 years of cited content as cited by the Current Year. It considers both the number of citations and the source of those citations, so that highly cited sources will influence the network more than

Normalized Eigenfactor

1.86673

The Normalized Eigenfactor Score is the Eigenfactor score normalized, by rescaling the total number of journals in the JCR each year, so that the average journal has a score of 1. Journals can then be compared and influence measured by their score relative to 1. Learn more

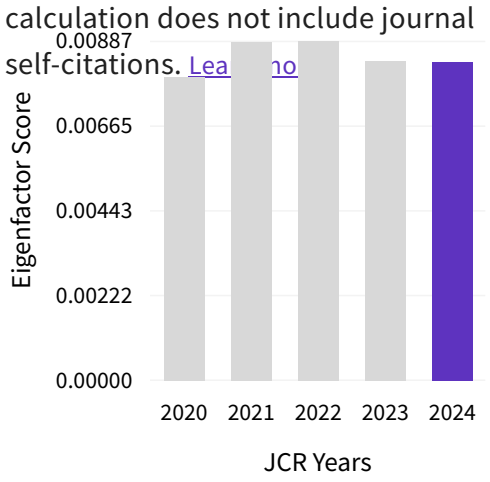
Article influence score

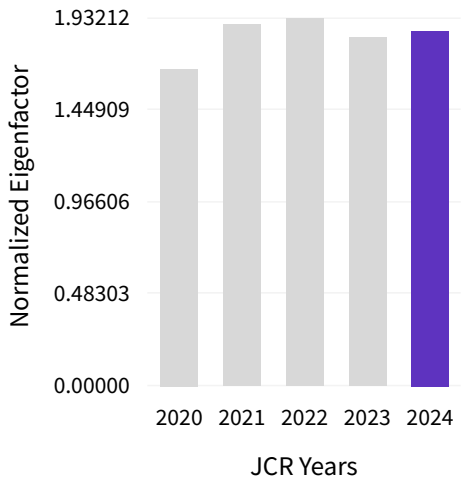
1.010

The Article Influence Score normalizes the Eigenfactor Score according to the cumulative size of the cited journal across the prior five years. The mean Article Influence Score for each article is 1.00. A score greater than 1.00

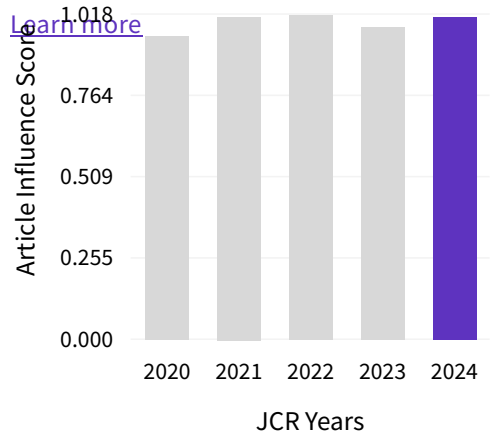
15

less cited sources. The Eigenfactor calculation does not include journal self-citations. Lea[illegible]no





indicates that each article in the journal has above-average influence. Learn more

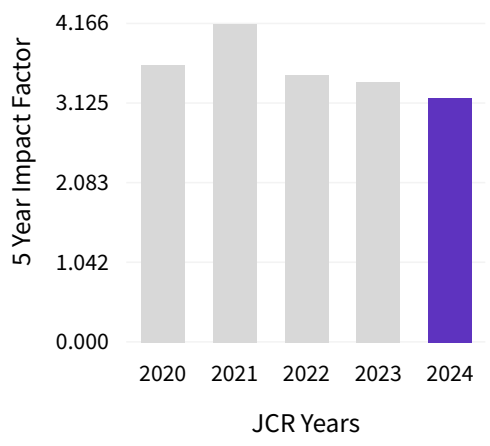


5 Year Impact Factor

3.2

View Calculation

The 5-year Impact Factor is the average number of times articles from the journal published in the past five years have been cited in the JCR year. It is calculated by dividing the number of citations in the JCR year by the total number of articles published in the five previous years. Learn more

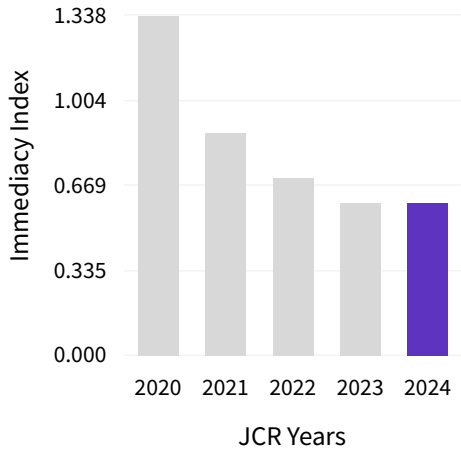


Immediacy Index

0.6

View Calculation

The Immediacy Index is the count of citations in the current year to the journal that reference content in this same year. Journals that have a consistently high Immediacy Index attract citations rapidly. Learn more



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Kocaeli Üniversitesi Explore open access funding | Change institution

Journal menu

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ORIGINAL ARTICLE

Succinate dehydrogenase deficient renal cell carcinoma frequently expresses GATA3 and L1CAM

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Abstract

Succinate dehydrogenase deficient renal cell carcinoma (SDH RCC) is an uncommon, familial RCC that has overlapping morphologic features with other low grade eosinophilic tumors of the kidney. Although the diagnosis of SDH RCC relies on loss of SDHB by immunohistochemistry (IHC), not all laboratories have access to this antibody. GATA3 and L1CAM are increasingly utilized in the diagnosis of eosinophilic renal tumors; however, their expression profile has not been studied in SDH RCC. We evaluated clinical parameters and GATA3 and L1CAM reactivity in a large nephrectomy cohort ($n=40$) of patients with SDH RCC. The male-to-female ratio was 3:1 and the median age was 48 years (range: 17 to 79 years). Specimens included 20 radical resections, 19 partial resections, and 1 unknown procedure. Tumor laterality was nearly equal (right:left = 18:20; one case was bilateral). Tumors in 4 (10%) patients were multifocal. Median tumor size was 2.0 cm (range 1 – 14.8 cm). Tumor stage distribution was: pT1a ($n=16$, 40%), pT1b ($n=7$, 18%), pT2a ($n=5$, 13%), pT2b ($n=4$, 10%), pT3a ($n=6$, 15%), and pT4 ($n=1$, 3%). Most cases ($n=33$, 83%) exhibited classical morphologic features, whereas 2/40 (5%) had sarcomatoid differentiation. GATA3 was positive in 37/39 (95%) tumors, with more than 50% of cells positive in 35/37 (95%), and with moderate-high intensity (2/3 +) in 33/37 (89%). L1CAM was expressed in 13/18 (72%) tumors, with moderate-high intensity (2/3 +) in 10/13 (77%). When both markers were performed, dual GATA3/L1CAM expression was present in 12/17 (71%) tumors. As L1CAM has been postulated to represent a marker of principal cell of the distal nephron, frequent L1CAM expression in SDH RCC suggests that they may originate from the principal cells of the distal nephron.

Keywords Succinate dehydrogenase deficient renal cell carcinoma · GATA3 · L1CAM

Introduction

Succinate dehydrogenase deficient renal cell carcinoma (SDH RCC) is a rare malignant neoplasm, accounting for less than one percent of primary kidney tumors [1]. It is caused by biallelic inactivating mutations in the genes transcribing several subunits (A, B, C or D) of the mitochondrial succinate dehydrogenase (SDH) enzyme [2]. These mutations are virtually always autosomal dominant and germline, and the most commonly affected unit in the SDH complex is SDHB, followed by SDHA, SDHC, and SDHD [2]. Personal or family history of other SDH-deficient tumors, such as pheochromocytoma/paraganglioma or gastrointestinal stromal tumor (GIST) is common [2]. SDH RCC mostly affects young adults with a slight male predominance [3]. While most of these tumors behave in an indolent fashion, aggressive behavior in a small subset of cases is well-documented, with approximately 10% showing distant metastases, sometimes years after primary tumor diagnosis [3].

Microscopically, SDH RCC is typically composed of monomorphic eosinophilic tumor cells with low-nuclear grade, frequent intracytoplasmic vacuoles filled with flocculent material, and a diffuse to nested growth pattern [1, 3]. Although uncommon, high-grade cytomorphology, including sarcomatoid differentiation, have been documented [3]. SDH RCC typically demonstrates an absence

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of keratin 7 (KRT7) and KIT expression by immunohistochemistry (IHC); pankeratin expression can be variable [3–5]. In lieu of disease-defining *SDH* gene mutation, aberrant loss of SDHB expression by IHC in tumor cells is considered sufficient for the diagnosis of SDH RCC [5]. Unlike most other molecularly-defined RCCs, SDH RCC is in the differential diagnosis with low-grade renal eosinophilic neoplasms including oncocytoma, eosinophilic chromophobe RCC, low-grade oncocytic tumor (LOT), eosinophilic vacuolated tumor (EVT), eosinophilic solid and cystic RCC (ESC RCC), and hybrid oncocytic tumors in patients with Birt-Hogg-Dube (BHD) syndrome [6]. Inclusion of relatively novel markers such as GATA3 and L1CAM have gained traction in the genitourinary pathology literature [7–12]. After encountering several cases in which SDH RCC showed positivity for both GATA3/L1CAM, herein we explore the expression profile of GATA3/L1CAM in a large multi-institutional SDH RCC cohort.

Material and methods

Case selection

A multi-institutional cohort of SDH RCC, after the approval of local institutional review boards, was collected from Australia, Canada, Türkiye, and the United States. Ten cases were included from previously published cohorts, but in these prior reports, GATA3/L1CAM were not evaluated [1, 3, 13, 14]. Hematoxylin and eosin-stained slides and IHC assays were reviewed by genitourinary pathologists in the respective participating institutions. Diffuse loss of SDHB expression by IHC in the tumor cells with retained expression in the non-tumoral internal controls was the inclusion criteria. The following clinicopathologic features from all cases were documented: patient age, gender, known germline setting and/or family history, type of resection, tumor characteristics (size, laterality, pathologic tumor staging according to American Joint Committee on Cancer 8th Edition [15]). The presence of additional non-renal neoplasms was also recorded. Microscopically, the classical features of SDH RCC were defined as low nuclear grade and cytologically eosinophilic tumor cells with frequent intracytoplasmic vacuoles forming diffuse sheets or tightly packed nests and/or acini [3]. Additional microscopic features, including cystic changes, tubule formation, papillae, high-grade atypia, and sarcomatoid differentiation were also documented. Tumors were graded using the World Health Organization/International Society of Urological Pathology nuclear grading system [16].

Immunohistochemical assay and additional work-up

As previously indicated, loss of SDHB expression was requisite for the inclusion of cases. Depending on the material availability, IHC for GATA3 and/or L1CAM (see Supplemental Table 1 for more information) was performed on one representative whole-slide section in each contributing institution, and each scored the staining extent (percentage of tumor cells expressing the biomarker) and intensity (ranging from 0 to 3; 0 complete negativity, 1 + weak, 2 + moderate, and 3 + strong expression) and calculated the H-score (percent of cells x intensity, range 0–300). Similar extent and intensity evaluation was performed in cases where other IHC assays were available. We also recorded the results of molecular testing, if performed clinically. Available follow-up data was also documented in a subset of cases.

Results

Table 1 depicts the clinicopathologic features of the 40 patients with SDH RCC. Male-to-female ratio was 3:1 and median age was 48 years (range: 17 to 79 years). All specimens were nephrectomies, including radical ($n = 20$) or partial ($n = 19$), and in one case it was unknown. The right ($n = 18$) and left ($n = 20$) kidneys were similarly involved; one additional patient had bilateral involvement. Four patients (10%) had multifocal involvement. Five of 15 (33%) patients had a known personal/family history of an *SDH* germline mutation related tumor or syndrome. One patient had a prior SDH deficient paraganglioma, and another had a recurrent SDH RCC. Two patients had unrelated tumors including mesothelioma and uterine leiomyomas, respectively. Median SDH RCC tumor size was 2.0 cm (range 1 – 14.8 cm). Most tumors were staged pT1a ($n = 16$, 40%), followed by pT1b ($n = 7$, 17.5%), pT2a ($n = 5$, 12.5%), pT2b ($n = 4$, 10%), pT3a ($n = 6$, 15%) and pT4 ($n = 1$, 2.5%).

Most cases ($n = 33$, 82.5%) exhibited classical morphologic features (Fig. 1A). 16/40 (40%) had focal cyst formation (Fig. 1B). In 9/40 (22.5%) cases, focal edematous stroma was intervening with the diffuse/nested growth pattern, forming clusters of various sizes (Fig. 1C) or lace-like spaces (Fig. 1D), resembling oncocytoma and LOT, respectively. Other morphologic patterns included tubular (Fig. 1E; $n = 5$, 12.5%), and papillary (Fig. 1F; $n = 2$; 5%). Nine cases (22.5%) had focal high-grade atypia, and 2 cases (5%) had sarcomatoid differentiation (Fig. 1G).

IHC and molecular results are summarized in Table 2 (See Supplemental Table 2 for all biomarker data). All tumors showed diffuse loss of SDHB expression, as per the inclusion criteria (Fig. 1H). 37/39 (95%) cases expressed

Table 1 Patient and tumor characteristics of 40 Succinate Dehydrogenase Deficient Renal Cell Carcinoma (SDH RCC)

CASE#	Age/Gender	Known germline setting / Family history?	Procedure/ Laterality	Unifocal or multifocal?	Tumor size (cm, largest if multifocal)	WHO/ ISUP grade	Pathological stage/Extent of tumor ıf pT3a or higher	Extrarenal tumors	Follow up (months)
1	72/F	Unknown	Radical/Left	Unifocal	3	2	pT1a	none	23, NED
2	79/M	Unknown	Partial/Left	Unifocal	4	2	pT1a	none	12, NED
3	35/M	Unknown	Partial/Right	Unifocal	1.5	2	pT1a	none	NA
4	48/M	Yes, Father has SDH RCC	Partial/Left	Unifocal	9	2	pT2a	none	NA
5	22/M	Unknown	Radical/Right	Unifocal	1	2	pT1a	none	NA
6	51/M	Unknown	Radical/Right	Unifocal	6.5	3	pT1b	none	NA
7	43/F	Unknown	Partial/Right	Unifocal	2.9	2	pT1a	none	6, NED
8	54/F	Unknown	Partial/Left	Unifocal	2.5	2	pT1a	Uterine Leiomyomas	21, NED
9	29/M	Unknown	Radical/Right	Unifocal	12	4	pT3a	none	1, NED
10	72/F	Unknown	Partial/Bilateral	Multifocal	5	3	pT1b	none	72, NED
11	58/F	Unknown	Partial/Left	Unifocal	3.8	2	pT1a	none	0.5, NED
12	NA/M	Unknown	NA/NA	Unifocal	NA	4	NA	none	NA
13	66/F	Unknown	Radical/Left	Unifocal	4.5	4	pT4; renal sinus, and focal perineural invasion	none	1, NED
14	17/M	No	Radical/Right	Unifocal	5	2	pT1b	none	84, NED
15	33/M	No	Radical/Left	Unifocal	13	2	pT2b	none	26, NED
16	70/M	No	Partial/Left	Unifocal	2.6	2	pT3a; perinephric fat	none	4, NED
17	45/M	No	Partial/Left	Unifocal	3.1	2	pT1a	none	2, NED
18	56/M	No	Radical/Right	Unifocal	11	3	pT2b	none	5, NED
19	48/M	No	Radical/Left	Unifocal	3.5	3	pT3a; perinephric fat	none	25, NED
20	51/M	No	Radical/Left	Unifocal	4.7	2	pT3a; perinephric fat	none	11, NED
21	67/M	No	Radical/Right	Unifocal	4.5	2	pT1b	none	2, NED
22	62/M	No	Radical/Left	Unifocal	14.8	2	pT2b	NA	124, NED
23	39/M	Yes, personal	Partial/Left	Unifocal	3.5	2	pT1a	Kidney recurrence	1, NED
24	32/F	Yes, personal	Partial/Right	Unifocal	1.5	2	pT1a	NA	40, NED
25	20/F	Unknown	Partial/Right	Unifocal	5	2	pT1b	none	NA
26	46/M	Yes, personal	Radical/Left	Unifocal	9.9	2	pT2a	Paraganglioma	12, NED
27	40/M	Unknown	Partial/Right	Unifocal	2	2	pT1a	none	7, NED
28	62/M	Yes, personal	Radical/Right	Unifocal	9.5	2	pT2a	none	81, NED
29	58/M	Unknown	Partial/Right	Unifocal	5.4	2	pT1b	none	24, NED
30	47/M	Unknown	Radical/Left	Unifocal	6.5	2	pT1b	none	6, NED
31	62/M	Unknown	Partial/Left	Unifocal	1.8	2	pT1a	none	NA
32	64/M	Unknown	Partial/Left	Unifocal	2.7	2	pT1a	none	NA
33	70/M	Unknown	Partial/Left	Unifocal	1.5	2	pT1a	none	NA
34	65/M	Unknown	Radical/Left	Multifocal	9.5	3	pT3a; renal vein, renal sinus fat	none	47, DOD

Table 1 (continued)

CASE#	Age/Gender	Known germline setting / Family history?	Procedure/ Laterality	Unifocal or multifocal?	Tumor size (cm, largest if multifocal)	WHO/ ISUP grade	Pathological stage/Extent of tumor ıf pT3a or higher	Extrarenal tumors	Follow up (months)
35	48/M	Unknown	Radical/Right	Unifocal	10.1	3	pT3a; renal sinus fat	none	NA
36	73/M	Unknown	Radical/Right	Multifocal	7.5	2	pT2a	Mesothelioma	NA
37	26/F	Unknown	Radical/Right	Unifocal	11.5	2	pT2b	none	NA
38	36/F	Unknown	Partial/Right	Unifocal	3.5	2	PT1a	none	NA
39	40/M	No	Radical/Left	Multifocal	10	2	PT2a	none	NA
40	39/M	Unknown	Partial/Right	Unifocal	1.8	2	pT1a	none	72, NED

DOD Died of disease; *NA* Not applicable; *NED* No evidence of disease; *WHO/ISUP* World Health Organization/International Society of Urological Pathology

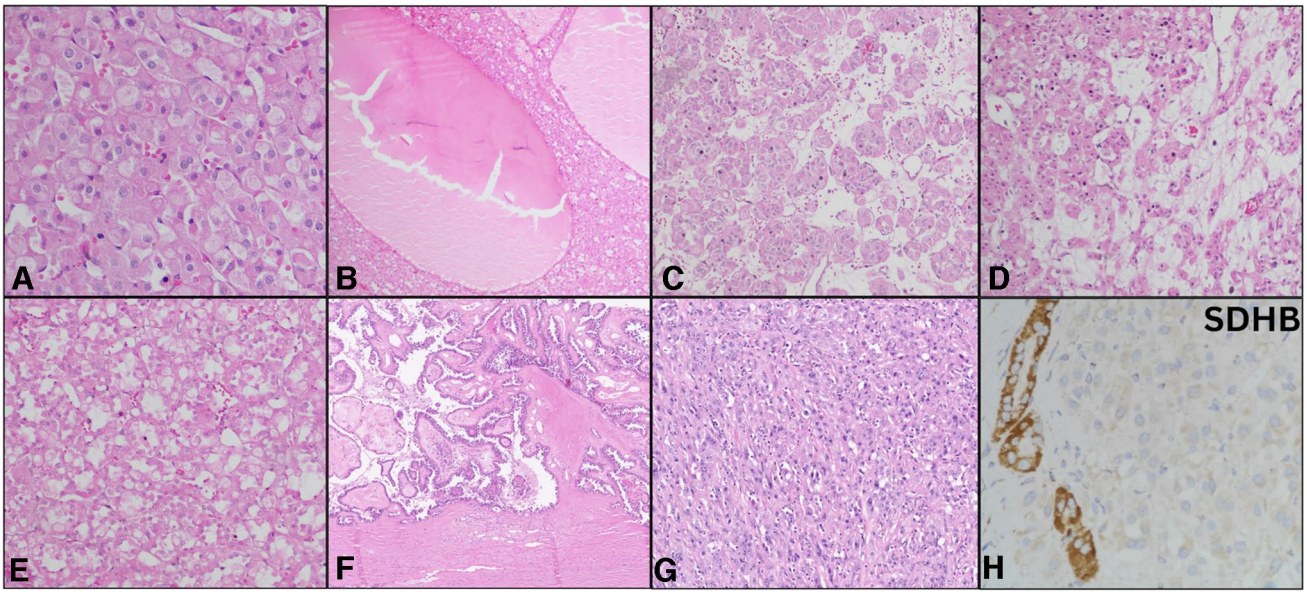


Fig. 1 **A** The majority of the cases in the cohort demonstrated diffuse/nested growth by tumor cells with eosinophilic cytology and intracytoplasmic vacuoles (200x). **B** Focal cystic changes were not uncommon (40x). **C-D** Focal intervening edematous stroma with clusters of tumor cells or "lace-like" edema with cord-like formations resembled oncocytoma and low-grade oncocytic tumor, respectively (each 100x). Other architectural patterns included pseudo-tubular growth (**E**; 100x) and papilla (**F**; 50x). **G** Rarely, sarcomatoid differentiation was seen (100x). **H** SDHB immunohistochemical assay showed loss of expression in all casess with positive internal controls (200x); weak cytoplasmic expression was present in some cases as has been previously described for SDH-RCC

GATA3; 35/37 (95%) exhibited reactivity in more than 50% of tumor cells (Fig. 2A), and 33/37 (89%) showed moderate-high intensity (2/3 +). Of note, GATA3 showed decreased staining intensity in the low-grade foci (Fig. 2B) or in the high-grade tumor regions, when present (Fig. 2C). L1CAM was expressed in 13/18 (72%) of cases, with moderate-high intensity (2/3 +) in 10/13 (77%) cases (Fig. 2D). L1CAM showed more focal/patchy staining when compared to GATA3 (Fig. 2E). Similar to GATA3, it also showed decreased expression in the high-grade tumor regions (Fig. 2F). In cases where both GATA3 and L1CAM were performed, dual expression was present in 12/17 (71%) of cases. In addition, other IHC stains, such as Cathepsin K (0/13), CA9 (0/14), KRT20 (0/9) and KIT (0/30) were negative in all evaluated cases. Only 2/22 (9%) cases had focal (5%) and weak (1 +) KRT7 expression, and only 1/20 (5%) case expressed focal weak pankeratin. Next generation sequencing was performed for clinical purposes in 6/40 tumors; 5/6 (83%) had pathogenic mutations in *SDHB* gene.

Follow-up data was available for 25 patients with median length of 12 months (range 0.5–124 months). One patient died of disease at 47 months, and one had had a recurrent disease in the kidney at 11 months; remaining patients had no evidence of disease recurrence and progression.

Table 2 Immunohistochemical profile and molecular ınformation of 40 succinate dehydrogenase deficient renal cell carcinoma

Case #	Predominant architecture	Additional architectural features and comments	High-grade atypia		KRT7	KIT	GATA3	L1CAM	NGS performed?	
			High-grade atypia (%)	Sarcomatoid (% if any)	Extent (%), ıntensity (0–3)	Extent (%)	Extent (%), ıntensity (0–3)	Extent (%), ıntensity (0–3)	Yes (List pathogenic mutations)	No
1	Diffuse/Nested	none	none	0	5, 1	negative	**> 95,2**	**ND**		No
2	Diffuse/Nested	none	none	0	negative	negative	**> 95,3**	**ND**		No
3	Diffuse/Nested	none	none	0	negative	negative	**> 95,3**	**ND**		No
4	Diffuse/Nested	5% cystic	none	0	ND	negative	**> 95,3**	**ND**		No
5	Diffuse/Nested	none	none	0	ND	negative	**> 95,3**	**ND**	SDHB; c.293G > C, p.(Cys98Ser) in Exon 4 genu SDHB	
6	Diffuse/Nested	5% cystic	yes	0	ND	negative	**> 95,3**	**ND**	No detected mutation	
7	Diffuse/Nested	none	none	0	ND	negative	**> 95,3**	**ND**		No
8	Diffuse/Nested	5% cystic	none	no	ND	negative	**> 95,3**	**ND**	SDHB; c.725G > A, p.Arg242His inv exon 7	
9	Diffuse/Nested	predominantly solid with nested, tubular, and papillary features, 10% cystic	yes	5	ND	negative	**50,2**	**ND**		No
10	Papillary	predominantly papillary features	yes	none	ND	negative	**> 95,3**	**ND**		No
11	Diffuse/Nested	5% cystic	none	0	ND	negative	**> 95,3**	**ND**		No
12	Diffuse/Nested	5% cystic, focal dedifferentiation	yes	0	ND	negative	**> 95,3**	**ND**		No
13	Papillary	Unusual tubulocystic morphology, 20% cystic	yes	10	5, 1	negative	**negative**	**ND**		No
14	Tubular	Central scar, 5% cyst formation	none	none	negative	negative	**> 95,1**	**negative**		No
15	Diffuse/Nested	1% tubular	none	none	negative	negative	**> 95,2**	**ND**		No
16	Diffuse/Nested	5% cyst formation	none	none	negative	negative	**negative**	**ND**		No
17	Diffuse/Nested	5% cyst formation	none	none	negative	negative	**95,3**	**ND**		No
18	Tubular/Acinar	10% cystic	yes	none	negative	negative	**> 95,3**	**60, 3**		No
19	Diffuse/Nested	10% cystic	yes	none	ND	ND	**95,3**	**ND**		No
20	Diffuse/Nested	none	none	none	ND	ND	**> 95,2**	**ND**		No
21	Diffuse/Nested	none	none	none	negative	negative	**70,2**	**ND**		No
22	Nested and Tubular	none	none	none	negative	negative	**10,1**	**negative**		No
23	Diffuse/Nested	none	none	none	ND	ND	**80,1**	**20, 1**		No
24	Diffuse/Nested	none	none	none	negative	negative	**90,3**	**negative**	SDHB gene c.725C > A (p.Arg242His)	
25	Diffuse/Nested	10% tubular	none	none	ND	ND	**> 95,3**	**40, 2**		No

Table 2 (continued)

Case #	Predominant architecture	Additional architectural features and comments	High-grade atypia		KRT7	KIT	GATA3	L1CAM	NGS performed?	
			High-grade atypia (%)	Sarcomatoid (% if any)	Extent (%), intensity (0–3)	Extent (%)	Extent (%), intensity (0–3)	Extent (%), intensity (0–3)	Yes (List pathogenic mutations)	No
26	Diffuse/Nested	20% edematous areas, LOT-like	none	none	ND	ND	**> 95,3**	**90, 3**	germline SDHB c.278G > T (p.Cys93Phe) and PMS2 c.873del (p.Phe291Leufs*16) – germline testing not tumor	
27	Diffuse/Nested	60% edematous areas resembling LOT	none	none	ND	ND	**> 95,3**	**> 95, 3**		No
28	Diffuse/Nested	30% hemorrhagic areas resembling LOT, 5% cystic	none	none	ND	ND	**> 95,3**	**50, 2**	germline deleterious mutation, c.286 + 1G > A in SDHB as well as the low penetrance CHEK2 mutation c.470 T > C (p.Ile157Thr) – germline testing not tumor	
29	Diffuse/Nested	20% edematous areas	none	none	negative	ND	**> 95,3**	**10, 2**		No
30	Tubular	20% tubulopapillary features	none	none	ND	ND	**> 95,3**	**5, 2**		No
31	Diffuse/Nested	10% edematous and hemorrhagic areas	none	none	ND	ND	**ND**	**10, 1**		No
32	Diffuse/Nested	80% edematous and hemorrhagic areas resembling LOT	none	none	negative	negative	**> 95,3**	**80, 3**		No
33	Diffuse/Nested	20% tubular and 20% edematous areas resembling oncocytoma	none	none	negative	negative	**> 95,3**	**negative**		No
34	Tubular	none	yes	none	negative	negative	**> 95,1**	**negative**		No
35	Diffuse/Nested	20% tubular	yes	none	negative	negative	**90,3**	**90, 3**		No
36	Diffuse/Nested	30% tubular, 10% cystic, 10% edematous areas resembling LOT	none	none	negative	ND	**80,2**	**70, 2**		No
37	Diffuse/Nested	5% edematous areas	none	none	negative	negative	**> 95,2**	**5, 1**		No
38	Diffuse/Nested	5% cystic	none	none	negative	negative	**80,2**	**ND**		No
39	Diffuse/Nested	none	none	none	negative	negative	**> 95,3**	**ND**		No
40	Diffuse/Nested	10% cystic	none	none	negative	negative	**> 95,2**	**ND**		No

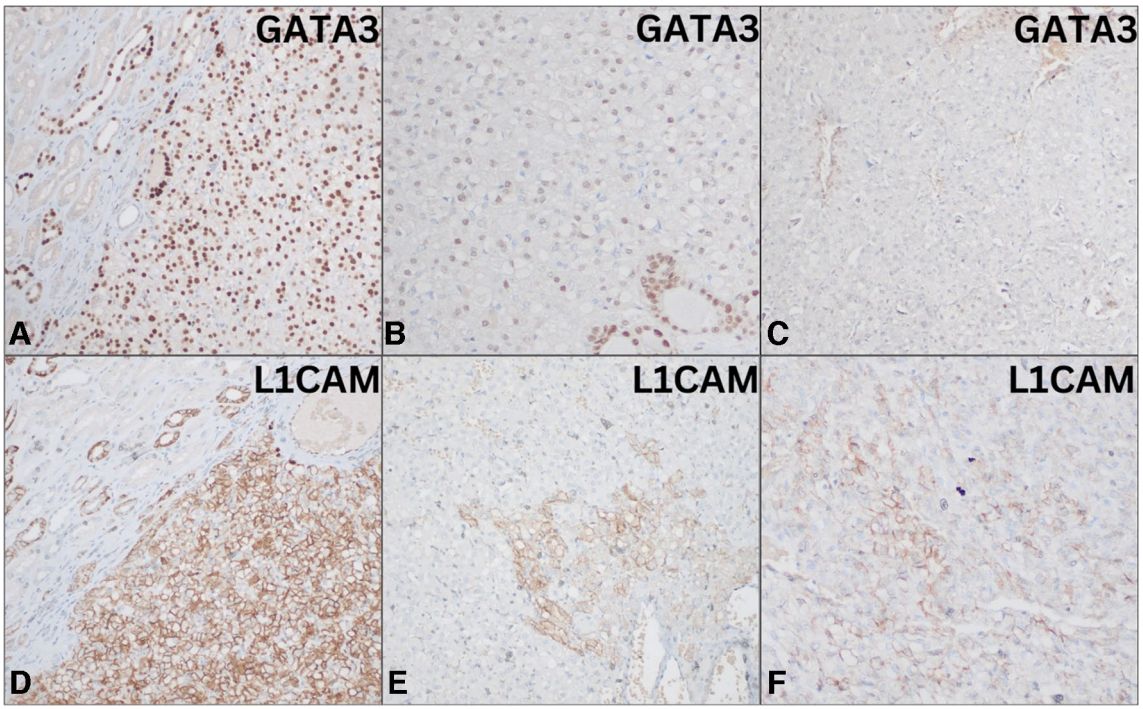


Fig. 2 **A** Diffuse and strong GATA3 expression was present in the vast majority of the SDH RCC (100x), although decreased intensity (**B**; 200x), and high-grade tumor regions (**C**; 100x) also had decreased GATA3 expression. **D** Similarly, L1CAM was diffusely and strongly positive when performed (100x); although focal expression was more common than GATA3 (**E**; 100x). L1CAM expression appeared to be decreased in high-grade tumor regions (**F**; 100x)

Discussion

Eosinophilic renal neoplasms represent one of the most dynamic fields in genitourinary pathology and include a wide spectrum of tumors that demonstrate distinct morphologic features, immunophenotype, and molecular alterations. For decades, IHC work-up of these tumors mostly included KRT7, KIT, vimentin, as well as special stain for colloidal iron [17], however, many tumors that did not demonstrate prototypical features of either oncocytoma or chromophobe RCC were diagnosed either descriptively or as "RCC, unclassified with low-grade features" [18]. Based on increased morphologic, immunophenotypic, and molecular understanding of kidney tumors, the diagnostic spectrum of renal oncocytic/eosinophilic tumors now includes several additional tumor entities, such as ESC RCC, hybrid oncocytic tumors in patients with BHD, SDH RCC, fumarate hydratase deficient RCC, ALK RCC, and two emerging entities (LOT, EVT) (6). The expanding spectrum of these renal entities has consequently altered the well-established IHC work-up algorithms, by incorporating biomarkers that can be applied as diagnostic surrogates for molecular testing (for example, SDHB, fumarate hydratase, ALK) as well as several relatively contemporary biomarkers, such as GATA3, L1CAM [10], cathepsin K [19], and GPNMB [20]. In this study, we explored the expression profile of two such markers, GATA3 and L1CAM, in a large multi-institutional cohort of SDH RCC.

Three distinct observations emerged from this study. First, and perhaps of foremost clinical relevance, SDH RCC can show morphologic overlap with LOT, including low-grade nuclei, nested architectural growth and areas of loose stroma with scattered or fine reticular cell arrangement. LOT, however, exhibits more prominent and extensive perinuclear halos, and more frequent edematous stromal areas compared to SDH RCC. Despite this morphological overlap, IHC work-up is critically important: LOT has diffuse KRT7 and retained SDHB expression, whereas SDH RCC has almost always absent KRT7 (as confirmed with our current findings) and SDHB loss.

Secondly, several recent studies highlight the utility of GATA3 as a useful diagnostic marker for LOT [8, 21–23]. As GATA3 appears to be consistently positive in SDH RCC, this may pose diagnostic challenges, particularly in a renal mass biopsy with scant available tissue. A recent large study also highlighted L1CAM as a highly sensitive marker for LOT, akin to GATA3 (10). However, as demonstrated in this study, SDH RCC can readily show positivity for GATA3 (95%), L1CAM (72%), or both GATA3/L1CAM (71%). It is therefore essential to use SDHB IHC when considering this differential.

The third observation from this study pertains to a potential role in understanding the ontogeny of SDH RCC. While prior studies have shown broader GATA3 expression within the cells of the distal nephron [10, 24], given the seemingly restricted expression of L1CAM to principal cells (and not intercalated cells) of the collecting duct [10, 25–28] in conjunction with the notable number of our SDH RCC tumors that showed L1CAM expression (72%, with strong staining in 77% of these positive cases), it may be plausible that akin to what has been reported for LOT [10], the principal cell may be cell of origin for SDH RCC.

While we recognize that the multi-institutional nature of our study resulting in GATA3 and L1CAM IHC being performed in different laboratories and using differing protocols as a study limitation, this setup simply reflects a real-life practice in a common diagnostic scenario. Nonetheless, in this study we highlight that GATA3 and L1CAM are reactive in great majority of SDH RCCs, which may be a diagnostic pitfall.

In summary, we report that a significant number of SDH RCC tumors demonstrate morphologic overlap with other low grade renal neoplasms, as well as positivity for GATA3 and L1CAM, which (to our knowledge) has not been previously described. Although in most cases prototypical SDH RCC tumor morphology will prompt ordering diagnostic SDHB IHC testing, in the setting of limited biopsy tissue or in cases showing imperfect morphology, SDH RCC may not be included as a top differential diagnostic consideration. Ourstudy demonstrates that in the latter scenarios, the expression of GATA3 and/or L1CAM should not be considered specific for LOT (a benign neoplasm) as they are can also be expressed in SDH RCC (a malignant neoplasm with potential familial associations). Accordingly, a panel of immunostains that also include KRT7 and KIT, and SDHB is recommended. Lastly, while an in-depth evaluation of the principal cell of the distal nephron as a cell of origin for SDH RCC using single-cell technology was beyond the scope of this study, our future studies are needed to corroborate this hypothesis.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00428-025-04078-6.

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Author contributions ARS, SRW, MA, and SKM designed the project. MO, AJG, FM, KT, LAG, DEB, CC, YYK, DB, GC, BYB, LK, BP, LC, and MSH contributed cases. MA devised the full manuscript draft. XZ performed GATA3 and L1CAM immunohistochemical assays. MA and MAA created the figures. ARS, SRW, KT, AJG, MSH, and LC revised the final manuscript.

Data availability All de-identified data is available in the manuscript as well as in the supplemental tables.

Declarations

In this retrospective study, all participating institutions contributed archival material under the approval of each institution's review board, and the completely de-identified information does not interfere with the patients' treatment.

Conflict of interest The authors did not receive support from any organization for the submitted work. The authors have no competing interests to declare that are relevant to the content of this article.

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CORRECTION

Correction to: Succinate dehydrogenase defcient renal cell carcinoma frequently expresses GATA3 and L1CAM

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In the published paper, the author's name should be corrected to Dilek Ertoy Baydar, not Deniz Ertoy Baydar.

The original article has been corrected. The scientific content of this manuscript has no error and remains unchanged.

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