

# Succinate Dehydrogenase-Deficient Renal Cell Carcinoma: A Case Report and Brief Review of the Literature

## —SDH-Deficient RCC

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## Abstract

Succinate dehydrogenase (SDH) is a critical enzyme that connects the tricarboxylic acid cycle and the mitochondrial electron transport chain, and pathogenic germline variants in SDH-related genes may be inherited in an autosomal dominant pattern, unlike other mitochondrial complexes, and predispose to SDH-deficient RCC. A 24-year-old male presenting with hematuria was found to have a mass in the left kidney. A radical nephrectomy was performed. Gross examination showed an exophytic mass at the upper pole of the left kidney. The tumor was well-circumscribed with a tan-brown, red-cut surface. Microscopically, the tumor had a pushing border, entrapping benign tubules at the periphery. The neoplastic cells demonstrated a variably solid or nested architecture and had eosinophilic to clear wispy cytoplasmic vacuoles or inclusions representing the large mitochondria. Immunohistochemically, complete loss of expression for SDH-B in the tumor cells was evident. SDH-deficient renal cell carcinoma is described as a rare entity with fewer than 75 case reports.

## Keywords

Succinate Dehydrogenase Deficiency, Renal Cell Carcinoma, SDHB, SDHA

## 1. Introduction

Succinate dehydrogenase (SDH), also known as complex II, is a vital component

located in the inner mitochondrial membrane. It plays a pivotal role in cellular energy metabolism, bridging the Krebs cycle and oxidative phosphorylation. Understanding its deficiency is key to comprehending the pathogenesis of SDH-deficient renal cell carcinoma [1].

It was a groundbreaking moment when the 2016 World Health Organization (WHO) classification of tumors of the urinary system and male genital organs recognized SDH-deficient renal cell carcinoma (RCC) as a distinct entity, not just a provisional one [2]. This is a rare tumor, with an incidence ranging from a mere 0.05% to 0.2% [3]. The immunohistochemical absence of SDHB staining is a crucial requirement for diagnosing SDH-deficient RCC [2].

Here, we present a compelling case of SDH-deficient renal cell carcinoma at a young age. This report is not only about a rare neoplasm; it also presents an intact gross pathologic picture that may capture your interest and raise awareness of this condition.

## 2. Materials and Methods

A 24-year-old male, a key demographic for this case, presented to the urology clinic with hematuria. Urinalysis showed 12 red blood cells per high-power field. A computed tomography (CT) scan of the upper abdomen revealed a large, exophytic, heterogeneous mass at the upper pole of the left kidney. No metastatic disease was seen. His past medical history was unremarkable, without a family history of malignancy. His bloodwork showed a hemoglobin of 13.8 g/dL, a creatinine of 1.16 mg/dL, and an eGFR of 88 mL/min/1.73 m<sup>2</sup>. Radical nephrectomy was performed two months after the presentation.

## 3. Results

The gross examination revealed an exophytic mass at the upper pole of the left kidney. The cut surface showed a unique, well-circumscribed tumor with expansive growth and a pseudocapsule, measuring 5.8 × 5.7 × 5.3 cm. The tumor was distinct, with brownish-tan to red color, focal hemorrhage, and partial cystic change (**Figure 1**). There was no macroscopic invasion into the renal sinus, the renal vein, or perinephric fat.

The microscopic examination revealed a fascinating, predominantly eosinophilic, solid-cystic neoplastic lesion at low power. Small amounts of the hyalinized stroma separated cystic spaces. Additionally, focal myxoid changes were in close contact with cystic spaces. Hemorrhage and focal coagulative necrosis were present (**Figure 2(a)**). Tubular entrapment by tumor cells was present at the lesion's periphery (**Figure 2(b)**). The tumor showed cuboidal to polygonal cells arranged in nests, solid sheets, or tubules. The neoplastic cells had eosinophilic cytoplasm with granularity and a flocculent appearance due to characteristic intracytoplasmic clear vacuoles and eosinophilic inclusions (**Figure 3**). The nuclei were round, without prominent nucleoli, and had a low nuclear grade (WHO/ISUP grade 1). No sarcomatoid or rhabdoid differentiation was identified. Mitoses were infre-

quent. The surgical margin was clear. Lymphovascular invasion or perineural invasion was not determined histopathologically. The pathological tumor stage was pT1b.

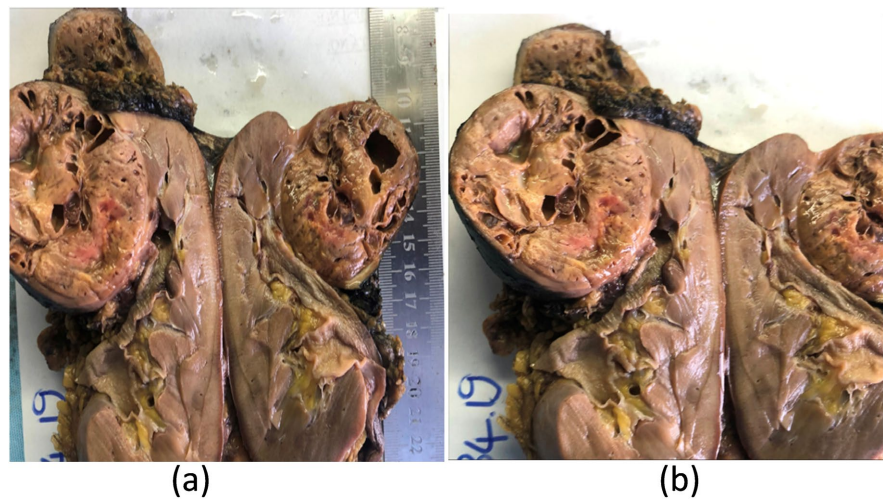


**Figure 1.** (a), (b) An exophytic mass was located at the upper pole of the left kidney. The cut surface revealed a well-circumscribed, tan-brown solid tumor with cystic change.

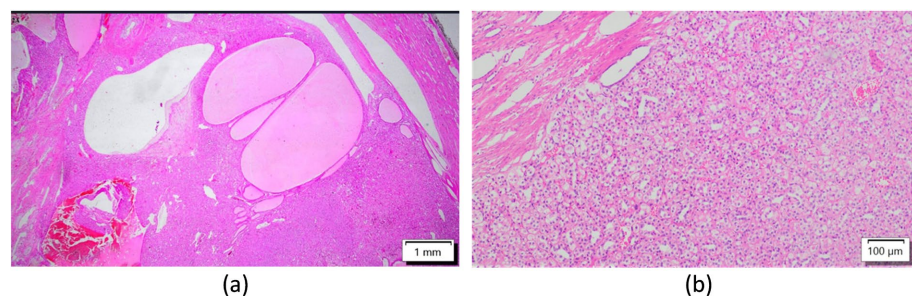


**Figure 2.** (a) Microscopic examination showed a predominantly eosinophilic, solid, and cystic neoplastic, vascularized lesion at low power. Cystic spaces were filled with pale eosinophilic material or blood. (Hematoxylin and eosin (H&E), original magnification  $\times 12.5$ ). (b) Tubular entrapment by the tumor cells was noticed at the periphery of the lesion (H&E, original magnification  $\times 100$ ).

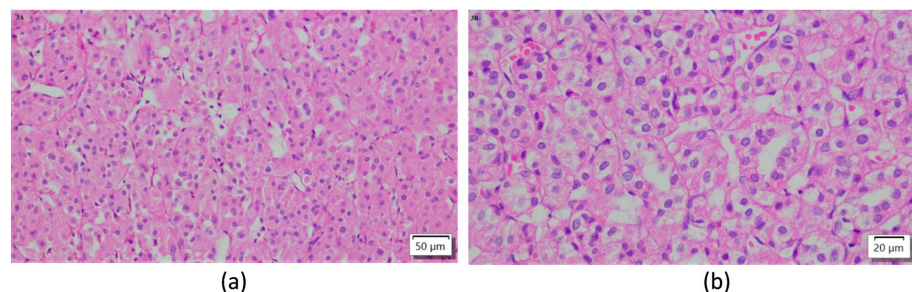


**Figure 3.** (a) The neoplastic cells had eosinophilic cytoplasm with granularity. Pale eosinophilic cytoplasmic inclusions were present (H&E, original magnification  $\times 200$ ). (b) There were occasional clear vacuoles and a flocculent appearance of the cytoplasm (H&E, original magnification  $\times 400$ ).

On immunohistochemistry, the neoplastic cells showed diffuse membranous expression of kidney-specific cadherin (KSC). They were negative for cytokeratin 7 (CK7), cytokeratin 20 (CK20), vimentin, carbonic anhydrase IX (CA IX), c-KIT (CD117), transcription factor e3 (TFE3). A modest intratumoral mast cell infiltrate was noted on c-KIT. Histochemically, colloidal iron staining was negative. SDHB staining was lost throughout two different tumor sections, with positive internal controls, including the entrapped benign tubules. In comparison, SDHA staining demonstrated diffuse granular cytoplasmic positivity (mitochondrial pattern) in tumor cells (**Figure 4, Table 1**). The diagnosis was established as SDH-deficient RCC.

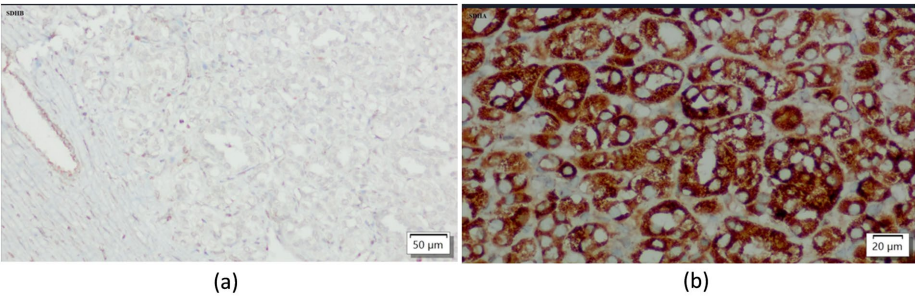


**Figure 4.** (a) SDHB staining was completely lost with positive internal controls including the entrapped benign tubules (original magnification  $\times 200$ ). (b) SDHA staining demonstrated diffuse granular cytoplasmic positivity (mitochondrial pattern) in tumor cells (original magnification  $\times 400$ ).

**Table 1.** Features of Succinate dehydrogenase (SDH) antibodies used for immunohistochemical examinations.

| Antigen | Clone name    | Supplier | Cell distribution | Result   | Dilution | Incubation |
|---------|---------------|----------|-------------------|----------|----------|------------|
| SDHA    | 2E3GC12FB2AE2 | Abcam    | Cytoplasmic       | Positive | 1/250    | 32 minutes |
| SDHB    | 21A11AE7      | Abcam    | Cytoplasmic       | Negative | 1/100    | 44 minutes |

Abcam, Cambridge, UK. Staining platform: Ventana BenchMark Ultra (Roche Diagnostics). Standard EDTA Antigen Retrieval Protocol was performed.

The patient, who did not receive adjuvant treatment, experienced a remarkable recovery. An 18 F-fluorodeoxyglucose (18 FDG) positron emission tomography scan six months after nephrectomy showed no evidence of local or distant recurrence. The genetic test was not performed to the patient. At the 18-month outpatient follow-up, the patient was alive with no evidence of recurrent or metastatic disease.

4. Discussion

Heterozygous mutations of *SDH* not only increase the risk of cancer, particularly hereditary pheochromocytoma/paraganglioma (PC/PGL), gastrointestinal stromal tumor (GIST), pituitary adenoma, and renal cell carcinoma [4] [5], but also lead to a diverse range of nonneoplastic conditions. These include neurodegener-

ation, diabetes, aging, optic atrophy, ischemia-reperfusion injury, and ischemic preconditioning [6]. The reported incidences of SDH-deficient pituitary adenoma, adrenal pheochromocytoma, extra-adrenal paraganglioma, and gastric GIST were 0.3%, 3%, 40%, and 5% - 7.5%, respectively [3].

SDH comprises A, B, C, and D subunits, joined by assembly factors. Researchers have identified four assembly factors (SDHAF1, SDHAF2, SDHAF3, SDHAF4) in humans. Unlike other complexes of the mitochondrial oxidative phosphorylation system, SDH is entirely nuclear-encoded [7] [8].

When SDH function is impaired, succinate accumulates in the mitochondria and is then transported to the cytosol. There, it plays a pivotal role by inhibiting prolyl hydroxylase, a key enzyme responsible for the degrading of hypoxia-inducible factor (HIF). This direct inhibition increases HIF levels, which in turn promote angiogenesis, resistance to apoptosis, and pseudohypoxic signaling that favors glycolysis. Elevated succinate levels also cause epigenetic dysregulation such as histone methylation (*i.e.*, hypermethylation). These conditions facilitate tumorigenesis [8], highlighting the direct impact of succinate on cellular processes.

In 2004, Vanharanta *et al.* [9] first reported *SDH*-deficient RCC in three patients from two families with hereditary paraganglioma-pheochromocytoma syndrome. All three cases were in their third decade of life and had a germline heterozygous mutation in *SDHB*. *SDH*-deficient RCC has distinctive morphologic features. *SDH*-deficient RCC is a malignant epithelial neoplasm composed of vacuolated cells with eosinophilic to clear cytoplasm with immunohistochemically complete lack of SDH subunit B staining [2]. Presentation is typically in young adulthood (22 to 72 years) [1] [3] [9]-[11].

Macroscopically, the tumor has distinct boundaries, often with pushing margins. However, it's important to note that some cases have shown unclear boundaries and renal capsule infiltration, as reported [12]. The cut surface is typically tan to brown, solid, and sometimes accompanied by hemorrhagic and cystic areas [1]-[3] [13]. Under the microscope, the neoplastic cells are mostly uniform, round to oval, and form solid, nested, or tubular patterns [2] [13]. Interestingly, papillary structures were observed in three patients with *SDHA* mutation, coinciding with immunohistochemical loss of expression for *SDHA* and *SDHB* [14]-[16]. Benign tubules are commonly found entrapped around the tumor periphery [2] [15] [17]. The most intriguing finding is the presence of intracytoplasmic clear vacuoles and/or inclusions containing eosinophilic or wispy pale material, which represent enlarged mitochondria [1] [2] [9] [18]. Nuclei are typically cytologically bland. Coagulative necrosis [16] [19] [20]; sarcomatoid or rhabdoid changes are rare. However, tumors with a rarely observed high-grade transformation may exhibit atypical cells with prominent nucleoli, loss of cytoplasmic inclusions, and sarcomatoid transformation [3] [11] [21].

Immunohistochemically, *SDHB*-, *SDHC*-, and *SDHD*-deficient renal cell carcinomas exhibit loss of *SDHB* expression but not of *SDHA* [22]. In contrast, *SDHA*-deficient RCC shows negative staining for both *SDHA* and *SDHB* [14].

Double-hit inactivation of any *SDH gene* results in negative immunohistochemical staining for SDHB in SDH-deficient RCC, making immunohistochemistry a valuable tool for screening syndromic disease [2] [3] [14] [22]. One of the two hits is almost always in the germline [22] [23]. Other immunohistochemical features of SDH-deficient RCC include consistently positive paired box gene 8 (PAX8) and KSC, lack of CD117 reactivity, and frequent absence of weak, focal cytokeratin 7 immunoreactivity [24].

Genetic studies have confirmed that most reported cases lacking SDHB staining have pathogenic *SDHx* mutations [3]. However, Ugarte-Camara *et al.* [17] reported a male patient with metachronous bilateral RCCs with an interval of 6 years between the two tumors. His family history included PGL and clear cell RCC, affecting his mother and maternal aunt, respectively. When the second tumor arose, SDH-deficient RCC was suspected. Although the staining was less intense than in the nonneoplastic area, neoplastic cells showed diffuse granular cytoplasmic SDHB staining in the first recognized renal tumor. The second diagnosed renal tumor also exhibited a bluish cytoplasmic granular positivity with SDHB staining. However, a significant finding emerged from the genetic test: a pathogenic deletion in the *SDHB* gene was identified, shedding light on the genetic basis of this rare case.

In our case, the first curious feature was the gross appearance, and the second was the patient's young age. Gross examination was remarkable, as it did not show the yellow-golden appearance of clear cell carcinoma, nor did it demonstrate intracystic protrusions suggestive of papillary RCC. There was also no central scar, as in oncocytoma. Microscopically, we did not identify a perinuclear halo or wrinkled nuclei, as in chromophobe RCC. No papillary architecture was present. The overall differential diagnosis primarily included succinate dehydrogenase deficient renal cell carcinoma, an eosinophilic variant of clear cell carcinoma, and eosinophilic solid and cystic RCC [25]. Histochemical and immunohistochemical stainings were performed for the most common differential diagnoses. The results excluded an eosinophilic variant of clear cell RCC [Vimentin (–), CAIX (–)], chromophobe RCC [colloidal iron stain (–); CK7, c-KIT (–)], eosinophilic solid and cystic RCC [CK20 (–)], oncocytoma [CK7, c-KIT (–)]. The final diagnosis was SDH-deficient RCC based on complete loss of SDHB expression. There was no accompanying TFE3 positivity, as reported previously [26].

The main rarely observed discriminating features in the present case, compared with those reported, included myxoid changes, hyalinized areas, and focal necrosis. Myxoid change or hyalinized stroma was seldom described [1] [3] [9]. Our case report had a lower WHO/International Society of Urological Pathology (ISUP) or Fuhrman nuclear grade than some reported SDH-deficient RCCs [1] [3] [14]–[16]. The main limitations of our case are unknown genetic status and limited surveillance related to neuroendocrine-specific markers.

Prognosis is usually favorable [17] [24] [27]. However, high-grade transformation features are associated with poor prognosis [3]. In addition, metastatic

disease to lymph nodes [16] or distant sites [28] may occur [3] [15]. Li *et al.* [11] also reported an SDHB-deficient RCC with pleomorphism and extensive necrosis that presented with bone metastasis. The prognostic outlook in our case is favorable, as there was no sarcomatoid change, pleomorphism, or high nuclear grade. However, focal necrosis and rarely observed (1/10 HPF) mitoses were exceptional for long-term follow-up.

Targeted therapies against vascular endothelial growth factor (VEGF), mammalian target of rapamycin (mTOR), and tyrosine kinase have been considered for patients with adverse presentations. McEvoy *et al.* [15] reported an SDH-deficient RCC with mixed solid, papillary, and microcystic architecture, patchy coagulative necrosis, sarcomatoid dedifferentiation, and ISUP grade 4 nuclear features. Immunohistochemically, tumor cells were negative for both SDHA and SDHB. Signs of recurrent disease were determined ten months after surgery. A PET-CT scan identified peritoneal, retroperitoneal, subcutaneous, and pulmonary metastases. Two variants were identified in *SDHA*. The patient started with a programmed cell death-1 (PD-1) inhibitor (nivolumab), and treatment was changed to different kinds of monoclonal antibodies due to the side effects [15]. However, there is no evidence-based guideline for chemotherapeutic treatment of disseminated disease [10] [28].

## 5. Main Points

1) SDH-deficient RCC is a condition that should be considered in eosinophilic-appearing renal cell carcinoma in patients presenting at a young age.

2) A detailed family history and genetic consultation should be recommended for long-term patient surveillance and for detecting other neoplastic processes as early as possible.

3) The prognosis was favorable in most reported cases. However, tumors with adverse prognostic features should be monitored for local or distant recurrence. In such a scenario, targeted therapies might be considered based on current literature.

4) The accumulation of cases of SDH-deficient RCC, a task that the healthcare community can undertake, may further highlight the clinicopathologic features for diagnosis, patient management, and screening of other family members for an accompanying hereditary tumor syndrome.

5) Biochemical and imaging surveillance is also necessary for the early diagnosis of SDH-deficiency-related neoplasms.

## Ethics Statement

Written Informed Consent for publication was obtained from the patient and added to the submission.

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ciety of Pathology and XXXIII International Congress of the International Academy of Pathology.

## Author Contributions

Conceptualization, S.K., A.O. and O.E.; methodology, S.K.; validation, S.K., S.C., and O.E.; formal analysis, O.E.; investigation, S.K., A.O., S.C.; resources, S.K., A.O., F.P., A.A., S.C., I.I.G., O.E.; data curation, S.K., A.O., S.C., O.E.; writing—original draft preparation, S.K., O.E.; writing—review and editing, S.K., O.E.; supervision, O.E.; funding acquisition, S.K. All authors have read and agreed to the published version of the manuscript.

## Conflicts of Interest

The authors declare that they have no potential conflicts of interest.

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