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Tumor Discovery

CASE REPORT

A rare clinical observation of ureteral IgG4-related disease in urological practice: A case report

Ekaterina Anikanova[1,2]*, Daniel Yagudaev[1,3], Konstantin Firsov[1,3], Nina Kalyagina[4,5]*, and Arina Plotnikova[5]

[1]Department of Urology and Oncology, Central Clinical Hospital "Russian Railways-Medicine," Moscow, Russia

[2]Department of Oncology and Radiology, Medical Faculty, A.I. Evdokimov Moscow State University of Medicine and Dentistry, Moscow, Russia

[3]Department of Endoscopic Urology, Faculty of Further Education, the Peoples' Friendship University of Russia, Moscow, Russia

[4]Prokhorov General Physics Institute of the Russian Academy of Sciences, Moscow, Russia

[5]Engineering Physics Institute of Biomedicine, National Research Nuclear University MEPhI, Moscow, Russia

Abstract

IgG4-related disease (IgG4-RD) is a systemic immune-dependent pathology marked by infiltration of lymphocytes and plasma cells expressing IgG4 in affected tissues, leading to phlebitis and fibrosclerosis. In urological practice, diagnosing IgG4-RD of the ureter, which may resemble a malignant tumor, presents challenges. We present a unique case: a 64-year-old patient (Patient P.) experienced left lumbar discomfort and a 4 kg weight loss over 2 months. A computed tomography scan revealed a 111 mm tumor obstructing the left ureter, causing hydronephrosis and regional lymphadenopathy. Suspecting ureteral urothelial cancer, the patient underwent a left nephroureterectomy with lymphadenectomy. Microscopic analysis revealed fibrosis and inflammation infiltration (lymphocytes, plasma cells, and eosinophils) in the ureter wall, with no evidence of tumor growth. Immunohistochemistry confirmed IgG4-positive plasma cells. Serum IgG4 rose to 149 mg/dL. Morphological findings led to a diagnosis of IgG4-RD of the ureter. Clinically, it is crucial to recognize IgG4-RD in ureteral neoplasms for early detection, to prevent unnecessary surgical intervention.

Keywords: IgG4-related diseases; IgG4; Ureter; Pseudotumor inflammation; Urothelial cancer

***Corresponding authors:**
Ekaterina Anikanova (Anikanova1801@gmail.com)
Nina Kalyagina (Nina.Kalyagina@gmail.com)

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1. Introduction

IgG4-related disease (IgG4-RD) is a systemic immune-mediated pathology characterized by diffuse or focal infiltration of affected tissues by lymphocytes and plasma cells expressing IgG4, with the absence of neutrophils (a moderate amount of eosinophils may be present). Subsequently, the condition leads to the development of phlebitis and fibrosclerosis, accompanied by an increase in IgG4 content in serum, serving as a laboratory sign[1-3].

Currently, IgG4-RD is defined by systemic involvement, IgG4-association, and common morphological manifestations, including lymphoplasmacytic infiltration and fibrosis. In 2003, a Japanese research group made a pivotal discovery, establishing IgG4-RD as an independent disease with a diverse array of clinical and morphological expressions. These expressions were previously regarded as distinct diseases[4].

In 2011, diagnostic criteria and a three-level classification for IgG4-RD were proposed for the first time[5]. The following criteria are considered essential: (i) The clinical presentation comprises local or multiple lesions with tumor-like inflammatory infiltration of focal or diffuse nature; (ii) the serum IgG4 concentration is elevated above 135 mg/dL; (iii) a histopathological examination reveals lymphoplasmacytic infiltration with fibrosis and obliterative phlebitis and infiltration of IgG4-positive cells (with more than 10 cells visible in the field of view at 400× magnification and a ratio of IgG4/IgG-plasma cells exceeding 40%). To establish a reliable diagnosis of IgG4-RD, it is necessary to combine clinical criteria with laboratory and histological studies. The presence of clinical and histological criteria indicates a probable IgG4-RD, while a combination of clinical and laboratory criteria indicate a possible IgG4-RD. In 2012, the first international nomenclature for this disease was proposed[6].

IgG4-RD affects various organs. Currently, IgG4-RD has been described in the pancreas, biliary tract, liver, stomach, fiber of the retroperitoneal space, mammary, lacrimal and salivary glands, prostate and thyroid glands, and skin[7-9].

Within the urinary system, IgG4-RD is infrequently observed, with a higher prevalence noted in the kidneys and bladder[10,11], while occurrences of ureteral lesions are primarily documented through isolated observations[12,13]. Given that IgG4-RD has only recently been classified as an independent disease, ureteral lesions displaying typical clinical and morphological characteristics were previously labeled as "inflammatory pseudotumor" or "idiopathic segmental ureteritis." The first description of such a lesion dates back to 1978 when Bissada and Finkbeiner first published their findings[14].

The clinical manifestations of IgG4-RD are non-specific, posing difficulties in the differential diagnosis process. This difficulty extends to distinguishing IgG4-RD from various infectious, inflammatory diseases, and tumors. The complexity of differentiation contributes to prolonged periods from the onset of initial clinical manifestations to a definitive diagnosis, leading to the potential adoption of incorrect treatment approaches for affected individuals.

The non-specific clinical manifestations of IgG4-RD present a considerable obstacle in achieving an accurate differential diagnosis, given the potential overlap with infectious, inflammatory, and neoplastic conditions. This overlap often results in delayed diagnosis and the implementation of inappropriate treatment plans for affected individuals.

Here, we present a clinical case of IgG4-RD affecting the ureter, initially misinterpreted as urothelial carcinoma and leading to surgical intervention in the patient.

2. Case presentation

Patient P., a 64-year-old individual, sought medical attention at the hospital, presenting complaints of left lumbar region pain and a weight loss of 4 kg over a 2-month period. A hypervascular tumor, exhibiting a circular shape, was identified during computed tomography (CT) imaging of the abdominal cavity and small pelvis. The tumor was located at the junction of the middle and lower thirds of the left ureter, causing lumen stenosis, with a length measuring 111 mm. Extending into the ureteral wall up to 8 mm, the tumor resulted in hydronephrosis on the left side. Further examination revealed an expansion of the upper third of the ureter to 18 mm, and the pelvis of the left kidney measured 27 mm. Additionally, external iliac lymph nodes on the left exhibited enlargement, measuring up to 18 mm in diameter, while obturator nodes reached up to 13 mm (Figure 1A and B).

The patient underwent an ureteroscopy, during which a circular constriction of the ureter to 2/3 of its lumen was observed at the border of the lower and middle sections of the left ureter. This constriction resulted from the proliferation of whitish tissue covered with fibrin. The shaft of the instrument was passed above the constriction to determine the dilatation of the ureter. The length of the constriction was approximately 100 mm. A biopsy was taken from the affected segment of the ureter. Microscopic examination of the biopsy revealed small fragments of the ureteral wall with induced changes, partially covered with urothelium. The composition consisted of fibrous tissue with plethoric vessels and diffused focal lymphoplasmacytic infiltration, including an admixture of histiocytes and eosinophils. Notably, no conclusive signs of tumor growth were identified within the examined tissue (Figure S1).

Laboratory tests revealed a decrease in blood hemoglobin to 110 g/L, erythrocyte values to 3.39 million/μL, and an increase in blood creatinine to 131 μmol/L and urea to 7.1 mmol/L.

The clinical and radiological findings did not permit the exclusion of a malignant neoplasm of the ureter in the

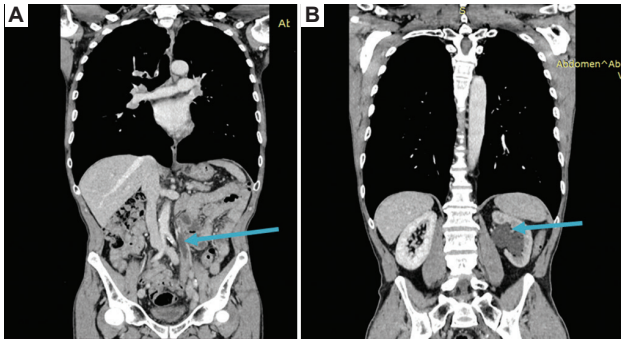


Figure 1. (A) Extended tumor in the middle and lower parts of the left ureter (indicated by the arrow). (B) Pyelokalikoectasis of the left kidney (indicated by the arrow).

patient. Therefore, a laparoscopic left nephroureterectomy with regional lymph node dissection was performed. Following the insertion of working trocars and pneumoperitoneum, the left kidney was mobilized with the ureter to the bladder. Instrumental palpation identified the presence of a whitish-colored, stony formation with a length of up to 100 mm at the junction of the middle and lower thirds of the ureter (Figure 2).

At this level, the ureter was intimately fused with nearby tissues, and enlarged lymph nodes were observed in the left iliac area (Figure S2).

The macroscopic sample consisted of a removed left kidney and left ureter with a tumor and a resected orifice of the left ureter (Figure S3).

Upon macroscopic evaluation, the gross specimen comprised a removed left kidney measuring 100×40×45 mm. The capsule proved challenging to remove. Incisions revealed an expanded calyx-pelvic system with a smooth, pinkish-cyanotic mucous membrane. The boundaries between cortical and medullary regions were indistinct. The left ureter measured 210 mm, exhibiting a significant narrowing of the lumen exceeding 110 mm in the middle and lower thirds, accompanied by up to a 10 mm thickening of the wall. The ureteral wall at this level presented as whitish-gray dense tissue. The distance from the narrowing site to the distal edge of the resection was 30 mm and from the kidney hilum was 90 mm. The removed lymph node varied in diameter from 6 mm to 18 mm (Figure 3A and B).

Microscopic examination revealed a tumor-like thickening of the ureter wall due to extensive moire (storiform) fibrosis (Figure S4). Additionally, there was moderately pronounced, diffuse infiltration of lympho-plasma cells with an admixture of eosinophils (Figure 4).

The muscle wall exhibited fraying, and the muscle fibers displayed hypertrophy. The mucous membrane was

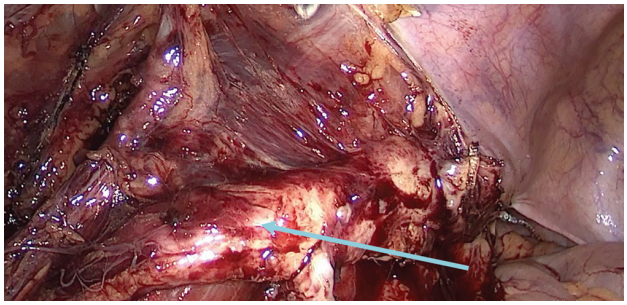


Figure 2. A tumor on the border of the middle and lower parts of the left ureter (indicated by an arrow).

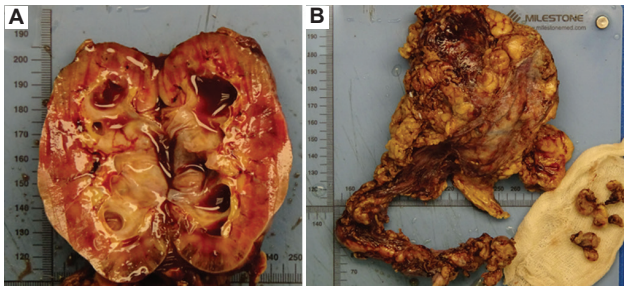


Figure 3. (A) Left kidney. (B) Left ureter dissected with lymph nodes.

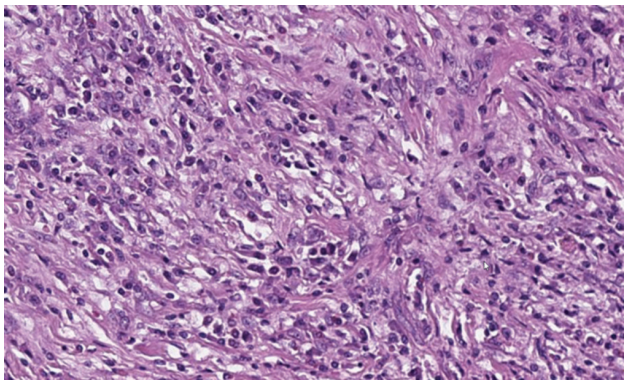


Figure 4. Ureteral wall with diffuse infiltration by lymphocytes, plasmocytes, and eosinophils (hematoxylin end eosin staining, magnification: ×400).

covered with urothelium of normal histological structure, devoid of signs of atypia (Figure S5).

An increased number of venous-type vessels was noted, exhibiting both inflammatory infiltration of the walls without obliteration of the vein lumens and areas with complete obliteration (non-obliterating and obliterating phlebitis). Furthermore, inflammatory infiltration and fibrosis affected the ureteral adventitia and surrounding adipose tissue.

The extracted kidney tissue exhibited typical architectonics with a relatively moderate degree of interstitial infiltration by lympho-plasma cells, interstitial

fibrosis, and sclerosis of individual glomeruli. The renal tubule lumens were dilated, accompanied by dystrophic changes in the tubular epithelium.

Within the lymph node tissue, slight fibrosis was noted with plasticization of the interfollicular spaces and sinuses. Morphological changes were most consistent with tumor-like fibroinflammatory lesions of the ureter, chronic interstitial nephritis, and sinus histiocytosis of the lymph nodes.

For the differential diagnosis of IgG4-RD, immunohistochemistry (IHC) was performed using antibodies against CD138, anaplastic lymphoma kinase (ALK), kappa and lambda light chains, and IgG4. IHC specimens revealed a normal kappa/lambda ratio in the infiltrate, and no ALK expression was observed. Staining with antibodies against CD138 confirmed the presence of an admixture of plasma cells in the infiltrate (Figure 5), the majority of which were IgG4-positive (Figure 6).

The CD138+/IgG4+ ratio exceeded 80%, with over 200 IgG4-positive plasma cells observed in the field of view at 400× magnification.

Morphological and IHC changes corresponded to IgG4-related ureteritis (so-called segmental ureteritis) and IgG4-related interstitial nephritis. No evidence of malignant tumor growth was found in the examined tissue. According to the morphological and IHC results, the patient exhibited a serum IgG4 concentration of 149 mg/dL (reference range: 10 – 135 mg/dL). Thus, based on the clinical, morphological, and laboratory findings, we established the diagnosis of IgG4-RD of the left ureter. The patient is currently under dynamic follow-up, and no signs of disease recurrence have been observed.

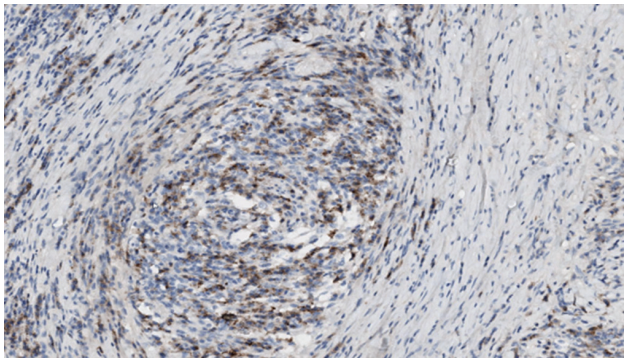


Figure 5. Immunohistochemical image showing periphlebitis. There is a positive reaction with antibodies to CD138 in the cytoplasm of plasma cells. Magnification: ×200.

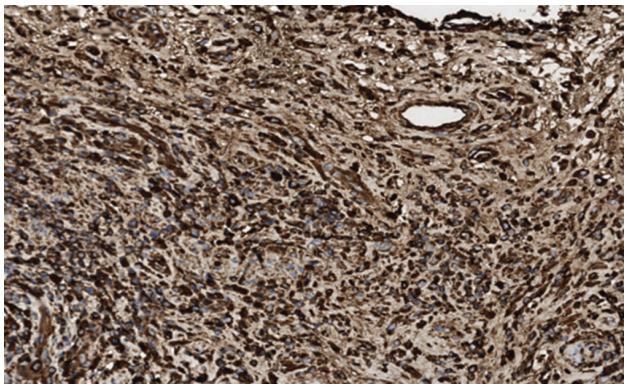


Figure 6. Immunohistochemical study showing positive staining with IgG4 antibodies in the tumor. Magnification: ×400.

3. Discussion

IgG4-RD has recently been identified as an independent disease, characterized by the presence of lymphoplasmacytic infiltration of affected tissues accompanied by hyperproduction of IgG4[1-3]. This condition is observed in various organs, exhibiting a diverse range of clinical manifestations, occasionally mimicking malignant neoplasms[7,8]. While IgG4-related nephritis and retroperitoneal fibrosis have been described in detail in the literature, instances of IgG4-related ureteral diseases have been documented in only 15 clinical cases[12,13,15]. The management of patients with ureteral IgG4-RD remains challenging, given the complexities associated with both diagnosis and treatment.

The complexity of the diagnosis largely stems from the subtle clinical course of the disease and the absence of specific clinical manifestations. Notably, Williams *et al.* describe in their clinical observation the presence of pain in the right quadrant of the abdomen[16], while Lei *et al.* reported in their observation the manifestation of IgG4-related ureteral disease with pain in the left lumbar region[12]. Additionally, Zhong *et al.* describe the manifestations of the disease, including lumbar and abdominal pain, fever, myalgia, and weight loss[13]. In a comprehensive study analyzing treatment outcomes in 65 patients with IgG4 genitourinary diseases, Teng *et al.* reported that at the time of the initial presentation, all patients exhibited varying degrees of renal failure due to impaired urodynamics and damage to the tubular apparatus of the kidneys[10]. In our observation of IgG4-RD of the ureter, the patient's clinical manifestations included left lumbar pain and anorexia, which were also non-specific. Laboratory tests revealed an increase in blood creatinine to 131 μmol/L and urea to 7.1 mmol/L, corresponding to the initial stage of chronic kidney disease.

The complexity of the diagnosis is further compounded by the presence of radiological signs typically associated with ureteral tumor lesions. Lei *et al.* provide a radiographic description of an IgG4-related ureteral lesion

resembling a tumor, characterized by active contrast agent accumulation along the thickened ureteral wall and the presence of enlarged regional lymph nodes[12]. Similar radiological images obtained through multi-slice CT (MSCT) of abdominal organs are presented in the clinical observation by Williams *et al.*[16], which also reported the CT progression of the mass, noting a 1.6-fold increase in size over a 6-week observation period. In addition, through the evaluation of positron emission tomography (PET) with ^{18}F-fluorodeoxyglucose (FDG), Williams *et al.*[16] demonstrated high metabolic activity in the lesion identified through MSCT, as well as in the regional lymph nodes. Analyzing FDG PET-CT results, Ueki *et al.* also reported elevated metabolic activity in the ureteral formation, with up to an 8-fold excess accumulation of the radiopharmaceutical[15]. In our observation, we encountered challenges in interpreting CT results, as the patient presented with an extensive ureteral tumor exhibiting signs of active contrast agent accumulation and an increase in regional lymph nodes. The obtained radiological results did not permit the exclusion of a malignant neoplasm of the ureter.

The question of performing a tumor biopsy for morphological confirmation of the diagnosis remains debatable and unresolved. According to Ueki *et al.*, endoscopic biopsy of lesions should always be performed in controversial clinical situations. In such complicated clinical scenarios, where the outcome of the disease may be affected, Ueki *et al.* suggest performing a biopsy through laparoscopic access or resection of the tumorous ureter with an urgent morphological study[15]. At the same time, the question of endoscopic and intraoperative biopsy remains open, as, in the case of a malignant process, it may lead to its further spread. In our pre-operative clinical observation, the patient underwent ureteroscopy with a biopsy of the pathologically modified parts of the ureter. However, morphological examination revealed fragments of the ureteral wall represented by fibrous tissue with signs of chronic inflammation but without reliable signs of tumor growth. Therefore, pre-operative biopsy is not always informative and may not reveal specific signs of IgG4-RD.

According to established criteria, an important diagnostic parameter for identifying IgG4-related ureteral disease is an elevation in serum IgG4 beyond specified reference values (>135 mg/dL)[6]. In this regard, in contentious clinical scenarios, most authors deem it necessary to examine serum IgG4 levels. In the clinical observation by Ueki *et al.*, the median serum IgG4 level was 384 mg/dL (206 – 965 mg/dL). Zhong *et al.* reported a serum IgG4 increase exceeding 3 times the reference values[13]. In an analysis of a survey involving 65 patients with IgG4-RD of the genitourinary system, Teng *et al.* discovered that, for patients with IgG4-RD affecting the kidney, the mean IgG4 serum level was 9510 mg/L (4295 – 15050 mg/L), while for those with ureteral IgG4-RD, the mean serum IgG4 was 7920 mg/L (3058 – 38150 mg/L). In cases of renal IgG4-RD combined with retroperitoneal fibrosis, mean serum IgG4 levels were maximal, remaining at 14100 mg/L (3200 – 22000 mg/L). Teng *et al.* also reported an established direct correlation between the level of blood serum creatinine and the level of serum IgG4[10]. In our observation, the examination of serum IgG4 was performed after surgical treatment and histological examination. However, we noted a serum IgG4 increase to 149 mg/dL after the primary source was removed.

The pre-operative diagnosis of IgG4-RD is exceptionally challenging due to non-specific clinical manifestations, such as lumbar and abdominal pain, coupled with a radiological image that is frequently misinterpreted as indicative of a malignant neoplasm in the ureter. The determination of serum IgG4 is extremely rare in practice. For this reason, urologists often misdiagnose these diseases as malignant neoplasms, leading to the inadvertent treatment of patients with nephroterectomy. Early diagnosis is imperative to prevent overtreatment.

Treatment strategies for IgG4-associated disease are not well-developed, mainly relying on expert opinions and the results of retrospective studies. While the first prospective studies have been published in recent years, they are mostly small and non-randomized[17].

Glucocorticosteroids (GCS) serve as the primary first-line treatment for IgG4, with most patients responding well to induction therapy. In cases where there is a high risk of recurrence, identified by risk factors such as multiorgan damage, elevated concentrations of IgG4 and IgE, and peripheral blood eosinophilia, additional immunosuppressive drugs (azathioprine, methotrexate, tacrolimus, and cyclophosphamide) may complement the initial GCS therapy. However, their effectiveness has not been convincingly proven. Genetically engineered biologics are usually prescribed when conventional immunosuppressive drugs prove ineffective. Ebbo *et al.* used rituximab in 33 patients diagnosed with IgG4-associated disease[18], achieving a 93.5% clinical response rate and allowing discontinuation of GCS in half of the cases. However, within an average of 19 ± 11 months post-treatment, disease recurrence was observed in 41.9% of patients. Maintenance therapy demonstrated prolonged disease-free survival (P = 0.02). However, it should be noted that rituximab therapy is often associated with the development of severe infections and hypogammaglobulinemia[19]. The

relatively high complication rate associated with rituximab therapy significantly limits its applicability in the treatment of IgG4-associated disease, particularly for maintenance indications. Despite existing recommendations for treating IgG4-associated disease, many experts advocate for a cautious approach in specific clinical situations[20]. In our clinical observation, considering the radical nature of surgical treatment and the absence of disease-related clinical manifestations, the decision was made to opt for dynamic observation. This approach helped minimize the drug burden of the patient and postponed the initiation of drug therapy.

The question of optimal diagnostic methods for actively monitoring patients after treatment is still not fully resolved. PET-CT is considered a promising method for the diagnosis and dynamic monitoring of IgG4-associated disease. An evaluation of 46 PET-CT scans in 21 patients diagnosed with IgG4-associated disease[21] revealed pathological accumulation of FDG in organs commonly affected by IgG4-associated disease at the time of diagnosis or recurrence.

In general, PET-CT has demonstrated greater sensitivity than standard imaging in diagnosing lesions in various organs, particularly in arteries, salivary glands, and lymph nodes. However, in certain cases (e.g., in the presence of small lesions), false-negative results were observed. PET-CT findings before and after treatment generally correlated with the response to therapy and disease activity.

In presenting this clinical observation, our objective is to raise awareness among urologists regarding the potential presence of IgG4-RD in patients with suspected urothelial carcinoma of the ureter. We propose incorporating the determination of serum IgG4 and, in specific cases, biopsy of the formation into the diagnostic algorithm.

4. Conclusion

This uniquely rare clinical case emphasizes the importance for urologists to consider IgG4-RD in the differential diagnosis of ureteral neoplasms. Given that IgG4-RDs can be manifested in various organs and often resemble malignant neoplasms, we advocate for the inclusion of serum IgG4 level determination in the diagnostic algorithm for such polysemantic clinical scenarios. Accurate and timely recognition of IgG4-RD of the ureter facilitates a conservative cure with GSC drugs, potentially averting the need for aggressive surgical treatment.

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Conflict of interest

The authors declare that they have no competing interests.

Author contributions

Conceptualization: Ekaterina Anikanova, Daniel Yagudaev
Data curation: Konstantin Firsov
Formal analysis: Nina Kalyagina, Arina Plotnikova
Investigation: Nina Kalyagina, Daniel Yagudaev
Methodology: Daniel Yagudaev, Ekaterina Anikanova, Konstantin Firsov
Resources: Daniel Yagudaev
Supervision: Daniel Yagudaev
Validation: Ekaterina Anikanova, Daniel Yagudaev
Visualization: Konstantin Firsov
Writing – original draft: Ekaterina Anikanova, Nina Kalyagina
Writing – review & editing: Daniel Yagudaev, Arina Plotnikova

Ethics approval and consent to participate

The study was conducted according to the guidelines of the Declaration of Helsinki. Ethical review and approval were waived for this study due to the retrospective nature of the study and the use of de-identified data.

Consent for publication

Informed consent was obtained from the patient involved in the study.

Availability of data

All information that grounded this case report is available within the main text, tables, and figures. Further data are available from the corresponding author upon request.

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