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Antibodies everywhere: The spectrum of IgG4-related disease—A case series and literature review

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ABSTRACT

Introduction: IgG4-related disease (RD) is now known to affect multiple organs, including the gastrointestinal system, kidneys, lungs, and retroperitoneum. Diagnosis of IgG4-RD involves a combination of clinical, radiological, serological, and histopathological findings.

Case Series: This case series underscores the importance of recognizing IgG4-RD as a potential etiology of diseases of unclear pathology with patient presentations, including pancreatitis, cholangitis, renal and mesenteric ischemia, back mass, and interstitial lung disease.

Conclusion: Further, we discuss the histological findings, imaging, serology, other organ involvement and response therapy (HISORt) criterion of diagnosis, and treatment options including steroid-sparing agents.

Keywords: Autoimmune pancreatitis, HISORt criterion, IgG4 pancreatitis, Rituximab, Sclerosing cholangitis

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INTRODUCTION

Immunoglobulin G4-related disease (IgG4-RD) is a systemic fibroinflammatory condition characterized by the infiltration of IgG4-positive plasma cells and tissue fibrosis in various organs, often mimicking malignancy or other inflammatory disorders. The development of tumor-like masses, organ enlargement, and the presence of IgG4-secreting plasma cells—which can be confused with cancer cells—are some of the ways that IgG4-related illness (IgG4-RD) might mimic cancer. Furthermore, IgG4-RD frequently affects organs such as the kidneys, salivary glands, and pancreas, where mass-like growths may be mistaken for tumors.

While initially recognized in autoimmune pancreatitis (AIP), IgG4-RD is now known to affect multiple organs, including the gastrointestinal system, kidneys, lungs, and retroperitoneum. This case series explores the diverse clinical manifestations of IgG4-RD within the gastrointestinal system, highlighting the challenges in diagnosis and management.

The spectrum of IgG4-related gastrointestinal involvement encompasses autoimmune pancreatitis, sclerosing cholangitis, and inflammatory bowel disease-like presentations, among others. Patients may present with abdominal pain, jaundice, diarrhea, or acute pancreatitis, often necessitating a comprehensive diagnostic approach to differentiate IgG4-RD from other conditions.

Diagnosis of IgG4-RD involves a combination of clinical, radiological, serological, and histopathological findings. Elevated serum IgG4 levels, characteristic imaging features, and histological evidence of lymphoplasmacytic infiltrates, storiform fibrosis, and obliterative phlebitis are hallmark features. However, distinguishing IgG4-RD from malignancy or other

inflammatory disorders can be challenging, requiring careful consideration of clinical context and ancillary testing.

Treatment of IgG4-RD typically involves glucocorticoids as first-line therapy, with many patients experiencing rapid symptom improvement and disease control. However, relapse rates are high, necessitating long-term management strategies, including steroid-sparing agents such as azathioprine or rituximab.

This case series underscores the importance of recognizing IgG4-RD as a potential etiology of gastrointestinal pathology and highlights the need for a multidisciplinary approach involving gastroenterologists, rheumatologists, and pathologists for accurate diagnosis and optimal management. Through a better understanding of the clinical spectrum and diagnostic criteria of IgG4-RD, clinicians can facilitate timely intervention and improve outcomes for affected patients.

CASE SERIES

Case 1

A 69-year-old female patient presented with complaints of acute epigastric pain over the last few hours. On presentation, the patient was tachycardic at 110 beats per minute and hypotensive with a pressure of 100/56 mm of Hg. As a part of the workup, lab tests were ordered which showed normal liver, and kidney function tests, but an elevated lipase of 3000. Right upper quadrant ultrasound did not demonstrate any biliary dilation. The patient's triglyceride and calcium were within normal limits. The patient did not endorse any alcohol use history. No culprit toxins were noticed on medicine reconciliation.

The patient was resuscitated with 3 L of intravenous fluids leading to an improvement in heart rate and blood pressure. Then gastroenterology was consulted and an IgG4 panel was ordered. IgG4 levels were elevated at 1057 mg/dL, at least seven times the normal range. Computed tomography (CT) abdomen and pelvis with intravenous (IV) contrast showed acute interstitial pancreatitis and a mass-like opacity at the right lower lung zone (Figure 1). This finding prompted a CT lung with IV contrast which demonstrated the lung node again but also highlighted a suspicious nodule in the thyroid gland as well. Interventional radiology (IR)-guided thyroid and lung biopsies demonstrated an abundance of plasma cells and storiform fibroblasts which consolidated the diagnosis of IgG4-related autoimmune disease with multi-system involvement. The patient was started on prednisone 40 mg once daily with improvement in two days after initiation. A plan was made to continue for 4–6 weeks followed by a taper. At the end of treatment, CT-proven resolution of thyroid, lung and pancreatic changes was observed.

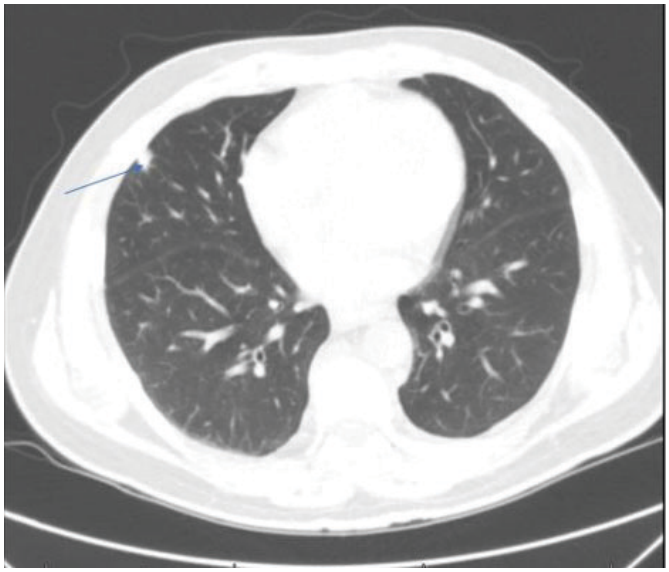

Figure 1: Right lower lobe lung nodule.

Case 2

A 56-year-old male patient with chief complaints including left flank pain and diarrhea for four days. The patient was afebrile and hemodynamically stable. The elevated levels of creatinine (1.6 mg/dL) and lactate dehydrogenase (LDH >400 U/L) were notable abnormalities in the laboratory results. Creatinine elevation suggested compromised renal function, corroborating the clinical suspicion raised by the flank pain. Elevated LDH levels indicated cellular damage and further supported the presence of an inflammatory or ischemic process. The patient had a history of atrial fibrillation raising suspicion for an embolic phenomenon. Computed tomography angiography led to the identification of acute/subacute renal infarcts, patchy gastrointestinal ischemia, and inflammatory changes in the aorta and iliac arteries suggesting vasculitis.

At this point, rheumatology was consulted. Immunologic workup showed elevated IgG levels of 203, pointing towards IgG4-related vasculitis. The initiation of oral steroid therapy proved to be a turning point in the patient's clinical course. The rapid improvement in symptoms following steroid administration underscored the inflammatory nature of the condition and validated the appropriateness of this therapeutic approach.

Case 3

A 60-year-old female with a history of Roux-en-Y procedure and recurrent episodes of pancreatitis was admitted for right upper quadrant pain. On presentation, she was afebrile, hemodynamically stable with a heart rate of 88, and blood pressure of 134/88 mmHg.

After an extensive workup including calcium levels, triglyceride levels, negative alcohol use history, elimination of any medicines implicated in pancreatitis

in the past, and absence of gall stones on ultrasound, the patient was labeled with a diagnosis of possible IgG4 pancreatitis as per 2020 guidelines with an elevated IgG4 level of 167 mg/dL during one of the pancreatitis episodes, radiological features of pancreatic nodular lesions. This was further aided by the patient's excellent response to steroid therapy.

During this hospitalization, she underwent CT imaging suspecting pancreatic pseudocyst given a recent episode two months ago but it demonstrated pancreatic edema, peripancreatic fluid collection, and dilated pancreatic duct with slight dilation of the common bile duct as well. This prompted a magnetic resonance cholangiopancreatography (MRCP) demonstrating 2–3 areas of stricturing without complete obstruction at the level of the lower bile duct (Figure 2).

Furthermore, the patient underwent endoscopic ultrasound directed transgastric ERCP (EDGE-guided ERCP) after pancreatitis resolution to evaluate the pancreatic duct, showing main pancreatic ductal dilation and some strictures at the common bile duct.

Biopsies were obtained demonstrating reactive changes with microabscesses. There was a concern if the cholangiopathy was in the setting of recurrent pancreatitis versus the IgG4-induced cholangiopathy which was contributing to episodes of pancreatitis. At this point, the gastric stent was left in place for possible future intervention.

However, given IgG-4 disease, the patient was started on prednisone 40 mg daily with a taper and an MRCP after four weeks of glucocorticoids demonstrating resolution of obstruction hinting at IgG4 cholangiopathy.

The patient was then started on mercaptopurine due to the development of steroid dependence given the recurrent episodes. However, the patient failed therapy and ultimately azathioprine titrated at 100 mg was started with good results.

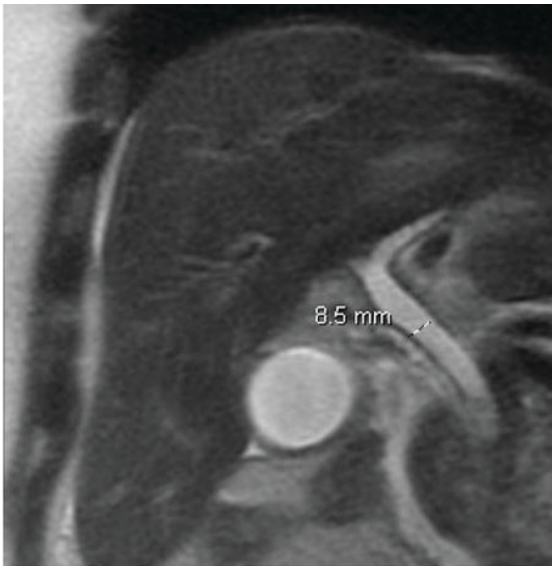

Figure 2: Common bile duct dilation with filling defect at a lower level.

Case 4

A 53-year-old male with chronic back pain failing supportive management underwent a CT showing a paraspinal mass and retroperitoneal nodules (Figure 3) which on subsequent biopsy showed IgG4 positive cells. This prompted serum IgG4 levels to be checked which were significant at 1290 mg/dL. The back pain responded significantly with prednisone 60 mg followed by a 6-week taper. This led to the resolution of symptoms. After around two years, the patient developed skin lesions that showed IgG4-dominant plasma cells, and elevated serum levels to similar levels as before. The patient further developed shortness of breath despite being on glucocorticoids for skin disease. A CT showed changes of the honeycomb fibrosis consistent with interstitial lung disease (Figure 4). The patient was then started on Rituximab therapy.

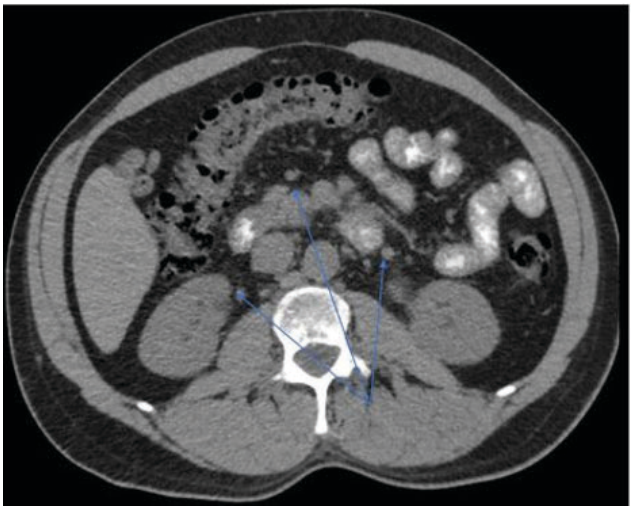

Figure 3: Retroperitoneal nodules.

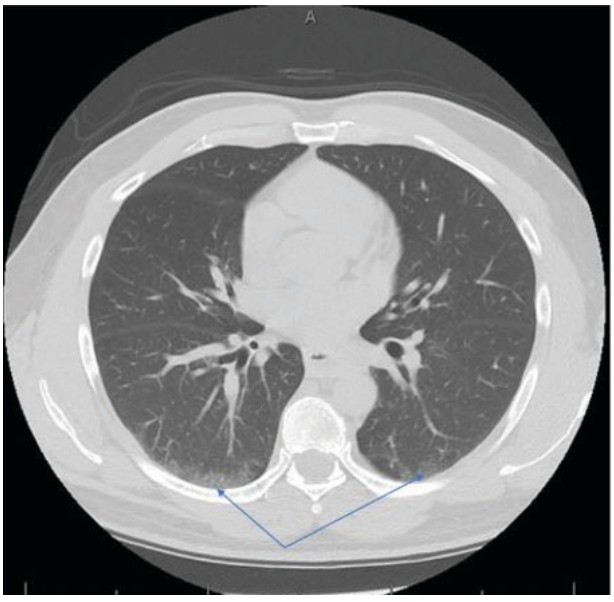

Figure 4: Ground glass opacities in posterior segments of lungs, indicating early interstitial lung disease.

DISCUSSION

Several reported cases of IgG4-RD focus only on pancreatitis or autoimmune pancreatitis. Our case series highlights the true systemic nature and variability of IgG4-RD. Each case here presents with diverse primary symptoms including acute pancreatitis, flank pain due to renal infarcts, recurrent pancreatitis with cholangiopathy, and chronic back pain with a paraspinal mass which later presented with interstitial lung disease. All cases demonstrate elevated IgG4 serum levels, but more importantly, biopsy confirmation played a crucial role in establishing the diagnosis in at least three cases. Steroid response was a key diagnostic clue in majority of the cases. Of note, case 3 and 4 highlight the challenge of steroid dependence leading to the need for second-line immunosuppressive agents such as azathioprine and Rituximab. Case 4 required Rituximab after progression to interstitial lung disease, suggesting that fibrotic progression may occur despite initial steroid responsiveness. Our case series expands the understanding of IgG4-RD by emphasizing its protean manifestations, diagnostic pitfalls, and evolving treatment paradigms.

As mentioned earlier, IgG4-RD is frequently associated with multi-organ involvement extending beyond the pancreas. 2020 Revised guidelines to diagnose IgG4 pancreatitis include (1) clinical or radiological features—one or more organs show diffuse or localized swelling or mass or nodule characteristic of IgG4-related disease, (2) Serological diagnosis—serum IgG4 level of more than 135 mg/dL, (3) Pathological diagnosis—2/3 of following criterion—dense lymphocytic and plasma cell infiltration, IgG4 positive plasma cells at greater than 10 per field, typical storiform fibrosis or obliterative phlebitis. Diagnosis is definite if all criteria are met, probable if the first and third criteria are met, and possible if the first and second are met [1].

The exact etiology of IgG4-RP remains unclear, but it is believed to involve an aberrant immune response possibly triggered by environmental factors in genetically susceptible individuals [2–4]. Clinical presentation for pancreatitis varies but abdominal pain, weight loss, and jaundice are common manifestations. Imaging studies, such as computed tomography (CT) and magnetic resonance imaging (MRI), reveal diffuse pancreatic enlargement and a hypodense appearance, mimicking pancreatic malignancy. Distinguishing IgG4-RP from pancreatic cancer is crucial due to the differing management approaches and prognosis.

Clinical diagnostic criteria are used to diagnose IgG-associated sclerosing cholangitis (IgG4-SC), which is based on a similar combination of imaging, serological, and histological findings, with the coexistence of additional IgG4-related disorders, and sometimes aided by the efficacy of steroid therapy [5]. Based on the stricture regions, four categories of the distinctive cholangiography features of IgG4-SC have been proposed [5]. Only the distal common bile duct (CBD) exhibits stenosis in Type 1 IgG4-SC, a condition frequently seen in pancreatic cancer. It is important to distinguish Type 2 IgG4-SC from primary sclerosing cholangitis (PSC) because it involves stenosis that is dispersed across the intrahepatic and proximal bile ducts. In IgG4-SC Types 3 and 4, hepatic hilar cholangiocarcinoma-like stenosis in the hilar hepatic bile duct is seen. A few cases with IgG4-SC underwent resection due to possible cholangiocarcinoma. Primary sclerosing cholangitis is a chronic, progressive illness that does not improve with steroid treatment and has a dismal prognosis. As a result, IgG4-SC needs to be distinguished from the other two illnesses. Serum IgG4 cut-off levels greater than × 2 ULN may be helpful in more accurately distinguishing IgG4-SC from PSC or cholangiocarcinoma [5]. Between 80% and 90% of IgG4-SC cases are linked to autoimmune pancreatitis (AIP). Other than AIP, symmetrical dacryoadenitis/sialadenitis and IgG4-related retroperitoneal fibrosis are two systemic IgG4-RD that are occasionally linked to IgG4-SC [6–8]. In contrast to PSC, individuals with IgG4-SC seldom experience inflammatory bowel disease (IBD) [8].

Tables 1 and 2 show diagnostic criteria. Renal involvement in IgG4-related disease often manifests as tubulointerstitial nephritis [9–11]. Renal artery involvement leading to infarcts is less common. The severe stenosis observed in both renal arteries in the second case, likely contributed to the acute/subacute infarcts seen on imaging. The gastrointestinal involvement, characterized by patchy ischemia and inflammatory changes in the celiac, superior mesenteric, and inferior mesenteric artery regions is an unusual presentation but underscores the systemic nature of this disorder.

Two distinct case reports detailing two individuals with increased serum IgG4 levels and parenchymal lung infiltrates were published in 2004 [12, 13]. Because they affect not only the lung parenchyma but also the intrathoracic lymph nodes, mediastinum, pleura, and airways, the intrathoracic symptoms of (ISD) seem to be quite diverse. Given the growing body of evidence, it appears that it could be mostly responsible for several fibroinflammatory disorders of unclear etiology that are seen in pulmonary disease.

Treatment of IgG4-RP primarily involves glucocorticoids, typically prednisone. The response to glucocorticoids is often dramatic, with rapid improvement in symptoms and imaging findings [3]. The proposed therapeutic regimen entails the administration of prednisone at a dosage of 40 mg per day, to be sustained for an initial duration of four weeks. Subsequently, a meticulously designed 7-week prednisone taper is recommended, wherein the dosage is gradually reduced by 5 mg per week until complete cessation, resulting in an overall treatment duration of 11 weeks. Despite this approach, a staggering 50% of patients experience relapse.

For individuals facing recurrent or refractory disease, particularly those undergoing steroid tapering or

discontinuation, the therapeutic landscape expands to encompass second-line agents. Among these options, azathioprine, Mycophenolate mofetil, methotrexate, and even tacrolimus enter the therapeutic arsenal, although it is crucial to note that the efficacy of these agents lacks confirmation from randomized controlled studies. Recent studies suggest that rituximab, a monoclonal anti-CD20 antibody, may be a promising alternative for patients with relapsing or refractory IgG4-RD. It has been studied only under non-randomized prospective studies however, several case series have demonstrated that rituximab leads to disease remission and corticosteroid sparing effects in a significant subset of patients [14–16].

Additionally, B-cell depletion therapy has shown efficacy and improvement in multi-level organ involvement in IgG4-RD [16]. However, long-term data regarding the safety and relapse rates of rituximab remain scarce, necessitating further prospective studies.

In terms of emerging therapies, novel biologic agents targeting the B-cell axis and inflammatory cytokines are under investigation. A phase III randomized controlled trial is currently assessing inebilizumab, an anti-CD19 monoclonal antibody, for IgG4-RD [16]. Additionally, Bruton's tyrosine kinase inhibitors, such as rilzabrutinib and zanubrutinib, are being studied for their potential role in regulating B-cell activity in IgG4-RD [16]. These targeted therapies may reduce the need for chronic steroid use while providing better disease control with fewer systemic side effects.

Table 3 summarizes therapies being studied for IgG4 disease [16]. The prognosis of IgG4-RD depends largely on early recognition and effective treatment. While many patients achieve remission with glucocorticoid therapy, a significant proportion experience relapse, particularly those with pancreatic and biliary involvement. One systematic review noted that patients with elevated IgG4 (>6.5 g/L), IgG (>20.8 g/L), and a responder index >9 were linked to a higher risk of relapse [17]. As with other autoimmune conditions, the research between IgG4-RD and malignancy has yielded conflicting findings. Some studies indicate that individuals with IgG4-RD have a similar cancer risk to the general population, while others suggest an increased likelihood of malignancies. Autoimmune pancreatitis, in particular, has been linked to a higher risk of various cancers, though one long-term follow-up study found no cases of pancreatic cancer among patients with AIP [15]. On the contrary, a separate study following AIP patients found that 11% developed malignancies, such as liver, biliary, or pancreatic cancers either shortly before or after being diagnosed with IgG4-RD [15]. Further investigation and prospective studies are needed to evaluate the role of malignancy in IgG4-RD progression.

Table 1: HISORt criterion for IgG4 disease

Category	Description
H: Histology	Dense lymphoplasmacytic infiltrate, storiform fibrosis, and obliterative phlebitis; >10 IgG4+ plasma cells/HPF; IgG4+/IgG+ ratio >40%
I: Imaging	Organ enlargement, mass/nodular lesions, or radiologic findings consistent with IgG4-RD (e.g., "sausage-shaped" pancreas in AIP)
S: Serology	Elevated serum IgG4 (>135 mg/dL), though not required for diagnosis
O: Other organ involvement	Involvement of typical IgG4-RD organs (e.g., bile ducts, salivary glands, kidneys, lungs, retroperitoneum)
Rt: Response to therapy	Rapid and sustained response to corticosteroids or immunosuppressive therapy

Table 2: Diagnostic methods per HISORt criteria

Diagnosis	Criteria
Definite IgG4-SC	Characteristic imaging (bile duct strictures, wall thickening) AND elevated serum IgG4 AND histology consistent with IgG4-RD OR other organ involvement typical of IgG4-RD AND response to corticosteroids
Probable IgG4-SC	Imaging features AND elevated serum IgG4 AND steroid responsiveness BUT histology or other organ involvement may be lacking
Possible IgG4-SC	Imaging features AND either elevated IgG4 OR response to steroids WITHOUT confirmatory histology or other organ involvement

Table 3: Alternate treatment methods

Drug	Mechanism	Effectiveness	Relapse rate	Adverse events	Recommendation
Rituximab	Anti-CD20 monoclonal antibody (B-cell depletion)	88–89% remission; effective in steroid-resistant, intolerant, and relapsing cases	21%; higher (36%) in multi-organ disease	Infections: TB, Borrelia skin; limited by ADR profile	Recommended for relapse, steroid resistance, and maintenance
Azathioprine	Purine synthesis inhibitor	81% effective (vs 70% steroids); lower relapse than steroid alone	19% on AZA vs 30% steroids	GI upset, transaminase elevation	Considered in maintenance; more data needed
Cyclophosphamide	Alkylating agent	Improved remission when combined with steroids	12% combo vs 38% steroids alone	Not specified	Useful in combination with steroids
Mycophenolate	Inhibits T and B cell proliferation	Higher remission vs steroids alone (76.5% vs 51.4%)	14.8% (MMF) vs 40% (steroids)	Not specified	Effective; particularly in relapse prevention
Tacrolimus	Calcineurin inhibitor	Effective in autoimmune pancreatitis Type 1 with AIC	Reduced relapse (used after steroid failure)	Not specified	Promising, especially in AIP1 and AIC overlap
Tocilizumab	IL-6 receptor blocker	50% remission at 6 months vs 20% with cyclophosphamide	Not statistically significant	Not specified	Limited data; potential alternative
CD19 Inhibitors	B-cell targeting (CD19+)	Recently approved; no post-marketing data yet	Unknown	Unknown	Not recommended pending further studies

CONCLUSION

In conclusion, the cases presented underscore the diverse clinical manifestations and systemic nature of IgG4-related disease (IgG4-RD) within the gastrointestinal system. From autoimmune pancreatitis to sclerosing cholangitis and beyond, IgG4-RD poses diagnostic challenges due to its variable presentation and multi-organ involvement.

While glucocorticoids remain the cornerstone of treatment for IgG4-RD, the cases discussed highlight the need for further research into alternative and adjunctive therapeutic options. Despite the efficacy of glucocorticoids in inducing remission, relapse rates remain high, and long-term use is associated with significant adverse effects.

Exploring second-line agents such as azathioprine, Mycophenolate mofetil, methotrexate, and even biologic agents like rituximab could offer promising avenues for achieving sustained remission and reducing the burden of steroid-related adverse effects. However, randomized controlled studies are needed to validate the efficacy and safety of these therapeutic modalities in IgG4-RD.

In parallel, further research into the pathogenesis of IgG4-RD is warranted to elucidate the underlying immune dysregulation driving disease progression. By unraveling the molecular mechanisms and immunologic pathways involved in IgG4-RD, novel targeted therapies may emerge, offering more effective and safer treatment options for patients with this complex condition.

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Author Contributions

Bipneet Singh – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Palak Grover – Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Iman El-Feki – Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Jahnavi Ethakota – Design of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Guarantor of Submission

The corresponding author is the guarantor of submission.

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Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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