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Case Report

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A Case Report of Anti-glomerular Basement Membrane Disease Mimicking Malignancy with Pyrexia as Initial Presentation

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Abstract

Immunoglobulin G (IgG) deposits in the glomerular basement membrane (GBM) are linear in anti-glomerular basement membrane (anti-GBM) illness, which is characterized by crescentic necrotizing glomerulonephritis. Clinically, fast progressing glomerulonephritis with or without pulmonary bleeding is a feature of classic anti-GBM illness. Atypical anti-GBM illness is a subset of anti-GBM disease that has milder symptoms and a better prognosis for the kidneys than conventional anti-GBM disease. The capillary beds of the kidneys and lungs are affected by the small vessel vasculitis known as anti-GBM disease. Although the triggers that set off the autoimmune response are not fully understood, it is an autoimmune illness brought on by the formation of directly pathogenic autoantibodies that target a well-characterized autoantigen produced in the basement membranes of these organs. The confirmation of spatial and temporal clustering of cases in recent years raises the possibility that environmental variables, including infection, can cause disease in people with a genetic predisposition. 40% to 60% of patients will also experience concurrent alveolar bleeding as they develop broad glomerular crescent formation, which typically presents with signs of fast progressing GN. Rapid removal of pathogenic autoantibody is the goal of treatment, which frequently involves plasma exchange, steroids, and cytotoxic therapy to stop continued autoantibody formation and tissue inflammation. However, patients who present with oligoanuria, a high percentage of glomerular crescents, or kidney failure requiring dialysis have a poor prognosis for renal function. Retrospective cohort studies suggest that when this combination of treatments is initiated early, the majority of patients will have good renal outcomes. Although de novo anti-GBM disease after transplant for Alport syndrome is a reported event, relapse and recurring disease after kidney transplantation are both infrequent. Atypical anti-GBM illness presentations are also more frequently observed, and co-presentation with other kidney disorders such ANCA-associated vasculitis and membranous nephropathy seems to occur more frequently than would be predicted by chance alone. These findings emphasize the need for further research to clarify the immunopathogenic mechanisms behind anti-GBM illness and how to more effectively refine and enhance treatments, especially for patients who present with poor prognostic indicators.

Keywords: Immunoglobulin G, Glomerular basement membrane, Autoimmune illness, Pulmonary bleeding

Introduction

A small vessel vasculitis called anti-GBM illness can damage either or both pulmonary capillaries and glomerular capillaries, which can lead to crescent-shaped glomeruli and glomerular necrosis. Autoantibodies against the GBM induce the necrotizing and crescentic glomerulonephritis that characterizes anti-GBM illness, which frequently results in pulmonary bleeding. IgG autoantibodies against the GBM, most frequently the non-collagenous domain of the alpha-3 chain (3NC1) of type IV collagen, are the cause of the anti-GBM glomerulonephritis [1, 2]. According to the Revised International Chapel Hill Consensus Conference, it is defined by the presence of circulating and deposited antibodies directed against basement membrane antigens, and as a result is categorized as an immune-complex small vessel vasculitis.

The Australians Stanton and Tange initially coined the term "Goodpasture disease" to characterize this syndrome in 1958 [2] when they reported on nine cases of GN accompanied by pulmonary hemorrhage. In his 1919 study detailing a fatal case of GN and lung hemorrhage that was at the time attributed to an atypic influenza infection, American pathologist Ernest Goodpasture is credited with providing the first description of the illness [3]. The ability to identify anti-GBM antibodies in kidney tissue and to show their pathogenic potential upon elution and transfer to nonhuman primates were not available until the development of immunofluorescence techniques in the 1960s, so we are unsure

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if any of these patients had anti-GBM disease as we understand it today. Patients' circulating anti-GBM antibodies were promptly found [4-6], and Wilson and Dixon provided the first in-depth clinical description of "anti-GBM antibody induced GN" [7]. While "Goodpasture syndrome" can be used to describe co-presentation with GN and pulmonary hemorrhage of any source, the term "Goodpasture disease" has remained and is typically reserved for individuals with demonstrable anti-GBM antibodies.

Despite high linear IgG staining, some patients have better renal outcomes and lower symptoms than those with classic anti-GBM illness. Additionally, they lack the circulating antibodies to 3NC1 that may be found using an enzyme immunoassay (EIA) that is readily available in the market. The EIA approach uses chemiluminescence enzyme immunoassays (CLEIA), fluorescence enzyme immunoassays (FEIA), and enzyme-linked immunosorbent assays (ELISA). Atypical anti-GBM disease is thought to be brought on by circulating pathogenic immunoglobulin, which differs from classic anti-GBM sickness in its recognition of GBM epitopes [8-10].

Traditional anti-GBM illness frequently progresses more quickly than other forms of glomerulonephritis because anti-GBM antibodies directly destroy multiple glomeruli at once. Early diagnosis and prompt treatment commencement are crucial. Plasma apheresis, cyclophosphamide, and high-dose steroids are frequently used to treat patients with typical anti-GBM nephritis [11-13]. It is uncertain if the advantages of aggressive treatment outweigh the hazards when it comes to the treatment of atypical anti-GBM.

Pulmonary bleeding results from lung involvement, and crescent-shaped glomerulonephritis causes renal involvement [1]. It occurs between 0.5 and 1 times per million people every year. All age groups are susceptible, but young men experience the peak incidence in the third decade, followed by a second peak in the sixth and seventh decades that equally affects men and women.

Case Report

The patient was presented with complaints of persistent low-grade fever for about 15 days, evening rise of temperature, fever more severe for 1 week associated with loss of appetite, loss of weight, generalized body pains. History of decreased urine output. Investigated outside raised CRP, raised ESR. USG abdomen was s/o hepatomegaly, cholelithiasis. Patient was admitted here for further management. The patient was admitted with provisional diagnosis of Acute Pyrexia/PUO, 2 Erythema Nodosum and treatment was started with IV fluids, PI's, antiemetics, folic acid supplements and other supportive treatment. Meanwhile necessary investigations were done, WIsh revealed low Hb (8.20 g%), raised TLC (12,810), raised serum creatinine (3.10), raised spot urine P/C ratio. CUE showed pus cells - 28, bacteria cocci - 42. CA - 19-9 (7.62). CEA - 1.20.

Nephrologist consultation was taken in view of deranged RFT with history of decreased urine output, opined to have AKI due to sepsis, underwent relevant investigations, advised continuation of treatment. General physician's consultation was taken in view of history of fever on & off, cough, underwent relevant, investigations - Malarial fever ruled out. Viral markers, Leptospira, brucella were negative. Blood and urine culture were negative, advice followed. Infectious Disease Specialist consultation was taken in view of above history - Further investigated - ANA with IF, C-ANCA, P-ANCA, RA factor, anti CCP, Complement - C3, C4, IgG4, LDH, ACE, Gene-expert MTB, sr. Electrophoresis were not conclusive. PET CT whole body was done for assessment of malignancy which showed diffuse cortical retention in both kidneys with multiple paraaortic nodes as described, infective, bulky pharyngeal tonsils with hypermetabolic right cervical nodes as described, infective, low grade focal hyperactivity in small bones of right foot as described -infective (in view of recent cellulitis), diffuse hyperactivity in bone marrow - reactive, advised followed accordingly.

In view of low Hb, multiple blood and blood product transfusions were given. Bone marrow aspiration and biopsy was done which revealed Bone marrow with Myeloid prominence and Lymphoplasmacytosis suggestive of a Reactive Bone marrow change. In view of PET CT scan findings, Surgical Oncologist consultation was taken, underwent Right Level II Cervical Lymph Node Biopsy Under General Anesthesia, sent for HPE, which was s/o reactive lymphadenitis. Repeat investigations were done as per need which revealed raised serum creatinine, advised, and underwent hemodialysis with monitoring of RPT/urine output. Infectious disease specialists reviewed the patient in view of raised TLC, started on IV antibiotic coverage (Inj. Linezolid). Plastic surgeon's consultation was taken in view of swelling left upper limb, opined to have cellulitis left elbow, HRUS left elbow was done which LASs s/o Basilic vein acute thrombophlebilis with adjacent inflammation/cellulitis and treated accordingly.

Rheumatologist consultation was taken in view of HRUS left elbow findings, underwent relevant investigations - Anti CCP, RA factor, HR U/S both hands, advice followed. After stabilization, under aseptic conditions, US guided Renal Biopsy done, sent for Biopsy which showed Kidney (needle] biopsy shows features of a crescentic glomerulonephritis with significant peripheral linear continuous deposits of IgG consistent with an anti-GBM antibody disease. Started on IV steroids, Inj. Endoxan and plasmapheresis, continuation of dialytic support. Further sent anti-GBM antibody which was positive (1:10). The patient had complaints of episodes of vomiting and was treated appropriately. Vascular surgeon's consultation was taken in view of swelling of left lower limb, Venous doppler left lower limb was done, which was s/o Intraluminal echogenic contents within common femoral vein and superficial femoral vein with no color uptake - Suggestive of thrombus, mud log perforator along course of superficial femoral vein. Started on LMWH. The patient was shifted to the intensive care unit for Plasmapheresis and 5 sessions of plasmapheresis given in intensive care unit care. The patient also received Inj. Rituximab 500 mg.

The patient was diagnosed and treated as a case of Crescentic Glomerulonephritis, anti-GBM disease and treatment was further continued with nephron-protectives, Inj. Erypro, antiemetics, steroids, antibiotics. PPI, folic acid supplements, LMWH, Inj. Albumin and other essential treatments.

The patient improved gradually and was discharged with the following advice.

- Inj. Erypro 4000 units S/C twice weekly

- Tab. Pantop 40 g once daily
- Tab. Optineuron once daily at 2pm
- Tab. Oxycal 500 mg thrice daily
- Tab. Folvite 5 mg once daily at 2pm
- Tab. Omnacortil 60 mg onoe daily
- Tab. Ganaton 50 mg twice daily 8am - 6 pm
- Cap. Bistla twice daily
- Inj. Clexane 40 mg S/C once daily
- Tab Duonen 200 mg twice All for 5 days

Discussion

"Anti-GBM disease" is characterized by serologically positive anti-GBM antibodies as well as crescentic necrotizing glomerulonephritis in histology. Anti-GBM is characterized pathologically by linear IgG staining that is deposited in the GBM, which is typically accompanied by autoantibodies against the GBM's structural collagen IV components. Classic anti-GBM illness is linked to a decreased life expectancy, fast progressing glomerulonephritis, and frequent pulmonary bleeding [8-10]. The basement membrane of the lungs and kidneys is harmed by infectious disorders (such as influenza), hazardous material inhalation (such as organic solvents and carbon tetrachloride), and smoking. As a result, antigen epitopes on the 3 and 5 chains are exposed and anti-GBM antibodies are produced in response to them [13]. The majority of these antibodies target the protein 3NC1 and bind to epitopes that are often covered by intrachain methionine cross-links (17-31 = EA, 127-141 = EB) [14-16]. However, other collagen IV epitopes, such as 5NC1 and 4NC1, are also reactive with some antibodies. Furthermore, moderate nonprogressive glomerulonephritis has been linked to antibodies that preferentially target the 345NC1 hexamer but not the 3NC1 monomer [17, 18]. When opposed to classic anti-GBM disease, atypical anti-GBM disease has milder symptoms and a better prognosis for the kidneys (Figure 1). The NC1 domain of the type IV collagen 3 chain contains amino acid residues that are recognized as antigen epitopes by anti-GBM antibodies at the N-terminal 17–31 (EA) and C–terminal 127–141 (EB) [11,12].

IIF, Western blot, and the EIA approach, which entails ELISA, CLEIA, and FEIA, are all used to identify anti-GBM antibodies. If anti-GBM antibodies cannot be found using EIA, such as ELISA, CLEIA, or FEIA, identifying the presence of anti-GBM illness using IIF or other techniques may be helpful. IIF or other techniques must be used in situations when anti-GBM antibodies cannot be detected by EIA in order to diagnose anti-GBM illness. A completely automated test can promptly produce the EIA results. The sensitivity and specificity of the EIA approach are very good [20, 21]. Under typical circumstances, the antigen epitopes are located within the basement membrane and are found in a hexamer made of type IV collagen 345 chains. Additionally, EIA does not identify circulating antibodies to 3NC1 in atypical anti-GBM disease. The NC1 domain EA area of the 5 chains has also been described as an antigenic epitope in addition to these epitopes.

The commercial EIA approach may not be able to identify antibodies for a variety of reasons described below. Firstly, there's a chance that anti-GBM antibody titers are too low. Low circulating anti-GBM antibody titers have been linked in certain studies to mild hematuria, proteinuria, and renal impairment. Additionally, a decreased incidence of crescentic glomerulonephritis and a better prognosis have been linked to low-affinity antibodies to the 3NC1 peptide (Figure 2).

Only sensitive assays, such as Western blot or biosensor tests, can detect antibodies with low affinities, as opposed to conventional tech-

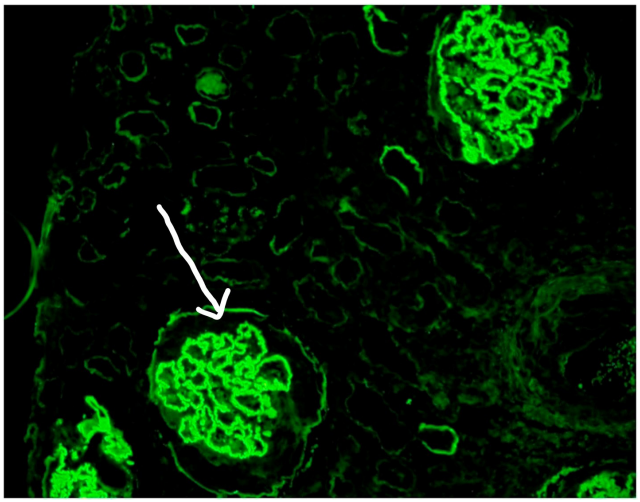

Figure 1: IgG immunofluorescence in kidney biopsy samples revealing GBM linear deposits (Arrow) and weaker Bowman's capsule and tubular basement membrane staining [19].

niques [17]. Second, Other antigens on GBM components besides 3NC1 may be the target of autoantibodies in some cases. Therefore, conventional assays are unable to find the antibodies. In this instance, normal kidney tissue allowed IIF to identify serum anti-GBM antibodies. Third, Antibody synthesis stops after the restoration of immunological homeostasis, and liver-mediated circulating antibody destruction is more pronounced than liver-mediated tissue antibody destruction [16] and this can lead to low titres of antibodies.

The combination of plasma exchange, cyclophosphamide, and corticosteroids appears to be effective in treating lung hemorrhage in >90% of patients treated with it and in maintaining independent kidney function in the majority of patients, including those who initially present with severe kidney dysfunction [15]. Patients with classic anti-GBM are typically treated with high-dose steroids, cyclophosphamide, and plasmapheresis, particularly those who have initial serum creatinine levels < 5 mg/dL and/or pulmonary hemorrhage [14]. It is still unknown, though, whether these strong regimens are beneficial for individuals with atypical anti-GBM who have modest clinical and pathological symptoms and frequently have no pulmonary bleeding. Proteinuria and hematuria can be eliminated by aggressive therapy, which also included high-dose steroids and plasma exchange and helped in improving renal function. Survival was 95% and 94% at 1 and 5 years, respectively, in patients with creatinine values under 500 mol/L. Survival was 82% and 50% at the same time points in patients who initially had creatinine levels over 500 mol/L but did not require urgent dialysis. However, only 8% of patients who initially required dialysis were still alive after a year had passed after recovery of their renal function. The best course of action needs to be determined through additional research. There is controlled evidence for rituximab monotherapy in patients with ESRD who have a live donor selected for transplantation but continue to have anti -GBM antibody positivity.

The degree of renal dysfunction at presentation, the percentage of glomeruli impacted by crescents, and oligoanuria at presentation are all predictors of poor renal outcomes [23]. Withholding therapy (and its associated toxicity) is frequently taken into consideration in these situations since no patient who required hemodialysis and had 100% crescents on a kidney biopsy had restored renal function [24]. It is important to take into account all instances for treatment, paying close attention to any other characteristics that can predict renal recovery on biopsy (such as concurrent acute tubular damage) and the tolerance of patients for each therapy component. If there is no sign of renal improvement after 2-4 weeks, a brief trial of early therapy may be taken into consideration and quickly discontinued. Additionally, in eligible individuals, the possible advantage of a period of immunosuppression to hasten the elimination of autoantibodies and enable kidney transplantation sooner should be taken into account.

The long-term survival of 449 patients with ESRD caused by anti-GBM disease was analyzed in a recent retrospective study (an-Australia and New Zealand Dialysis and Transplant Registry [ANZDATA] study), and it was discovered that their survival was comparable to patients with ESRD caused by other causes, whether they continued on dialysis or underwent kidney transplantation [25].

Alveolar bleeding rarely results in chronic respiratory consequences [26]. Anti-GBM illness relapses are uncommon. Avoiding precipitants is a crucial component of long-term therapy of these patients since it is typically linked to continued exposure to lung irritants such cigarette smoke and hydrocarbons [26, 27]. In situations of relapse involving the kidneys, we advise repeating the kidney biopsy in order to confirm the diagnosis and rule out associated illnesses including AAV and membranous nephropathy (described below). Standard retreatment with cytotoxins and corticosteroids is typically recommended in confirmed cases. Rituximab therapy has proven to be effective in a patient with multiple recurrent alveolar hemorrhages.

Conclusion

Coordination of big, prospective trials in anti-GBM illness is difficult since it is an uncommon condition that needs prompt therapy. Fur-

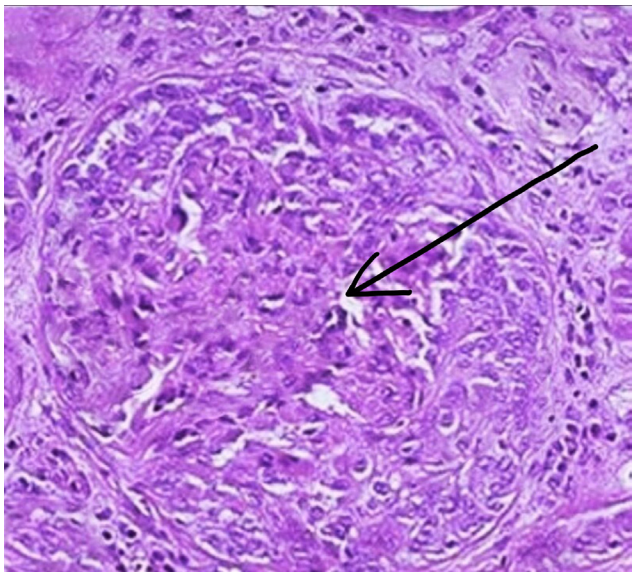

Figure 2: Goodpasture syndrome renal biopsy reveals crescentic glomerulonephritis (Arrow) [22].

thermore, when initiated at an early enough stage, the effectiveness of current treatment regimens is largely acknowledged. Future therapeutic research may thus concentrate on locating "add-on" therapies that might enhance outcomes in severe illness. In order to start the best therapy as soon as possible if anti-GBM antibodies cannot be found using EIA, such as ELISA, CLEIA, or FEIA, identifying the existence of anti-GBM illness using IIF or other techniques may be helpful.

Such investigations of anti-GBM illness and its experimental correlates are anticipated to shed new light on the processes underlying renal autoimmunity because it is the prototypical autoimmune condition. The alarming clinical presentation, however, and the need for emergency care, frequently in critically ill patients, highlight the necessity for clinicians to be aware of this rare condition, the difficulties in diagnosing it, particularly in atypical and variant presentations, and the early and appropriate use of immunosuppressive and extracorporeal therapies, in order to prevent morbidity and to improve survival.

Acknowledgements

None.

Conflict of Interest

None.

References

1. Jennette JC, Falk RJ, Bacon PA, Basu N, Cid MC, et al. (2013) 2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides. Arthritis Rheum 65: 1-11. https://doi.org/10.1002/art.37715
2. Stanton MC, Tange JD (1958) Goodpasture's syndrome (pulmonary haemorrhage associated with glomerulonephritis). Australas Ann Med 7: 132-144. https://doi.org/10.1111/imj.1958.7.2.132
3. Goodpasture E (1919) The significance of certain pulmonary lesions in relation to the etiology of influenza. Am J Med Sci 158: 863-870. https://doi.org/10.1111/imj.1958.7.2.132
4. Scheer RL, Grossman MA (1964) Immune aspects of the glomerulonephritis associated with pulmonary hemorrhage. Ann Intern Med 60: 1009-1021. https://doi.org/10.7326/0003-4819-60-6-1009
5. Lerner RA, Glassock RJ, Dixon FJ (1967) The role of anti-glomerular basement membrane antibody in the pathogenesis of human glomerulonephritis. J Exp Med 126: 989-1004. https://doi.org/10.1084/jem.126.6.989
6. McPhaul JJ, Dixon FJ (1969) The presence of anti-glomerular basement membrane antibodies in peripheral blood. J Immunol 103: 1168-1175. https://doi.org/10.4049/jimmunol.103.6.1168
7. Wilson CB, Dixon FJ (1973) Anti-glomerular basement membrane antibody-induced glomerulonephritis. Kidney Int 3: 74-89. https://doi.org/10.1038/ki.1973.14
8. Hellmark T, Johansson C, Wieslander J (1994) Characterization of anti-GBM antibodies involved in Goodpasture's syndrome. Kidney Int 46: 823-829. https://doi.org/10.1038/ki.1994.338
9. Koyama A, Yamagata K, Makino H, Arimura Y, Wada T, et al. (2009) A nationwide survey of rapidly progressive glomerulonephritis in Japan: etiology, prognosis and treatment diversity. Clin Exp Nephrol 13: 633-650. https://doi.org/10.1007/s10157-009-0201-7
10. Jennette JC (2003) Rapidly progressive crescentic glomerulonephritis. Kidney Int 63: 1164-1177. https://doi.org/10.1046/j.1523-1755.2003.00843.x
11. Netzer KO, Leinonen A, Boutaud A, Borza DB, Todd P, et al. (1999) The goodpasture autoantigen. Mapping the major conformational epitope(s) of alpha3(IV) collagen to residues 17–31 and 127–141 of the NC1 domain. J Biol Chem 274: 11267-11274. https://doi.org/10.1074/jbc.274.16.11267
12. Borza DB, Netzer KO, Leinonen A, Todd P, Cervera J, et al. (2000) The goodpasture autoantigen. Identification of multiple cryptic epitopes on the NC1 domain of the alpha3(IV) collagen chain. J Biol Chem 275: 6030-6037. https://doi.org/10.1074/jbc.275.8.6030
13. Pedchenko V, Bondar O, Fogo AB, Vanacore R, Voziyan P, et al. (2010) Molecular architecture of the goodpasture autoantigen in anti-GBM nephritis. N Engl J Med 363: 343-354. https://doi.org/10.1056/NEJMoa0910500
14. Cui Z, Zhao J, Jia XY, Zhu SN, Jin QZ, et al. (2011) Anti-glomerular basement membrane disease: outcomes of different therapeutic regimens in a large single-center Chinese cohort study. Medicine 90: 303-311. https://doi.org/10.1097/md.0b013e31822f6f68
15. Dammacco F, Battaglia S, Gesualdo L, Racanelli V (2013) Goodpasture's disease: a report of ten cases and a review of the literature. Autoimmun Rev 12: 1101-1108. https://doi.org/10.1016/j.autrev.2013.06.014
16. Yang R, Hellmark T, Zhao J, Cui Z, Segelmark M, et al. (2009) Levels of epitope-specific autoantibodies correlate with renal damage in anti-GBM disease. Nephrol Dial Transplant 24: 1838-1844. https://doi.org/10.1093/ndt/gfn761
17. Salama AD, Dougan T, Levy JB, Cook HT, Morgan SH, et al. (2002) Goodpasture's disease in the absence of circulating anti-glomerular basement membrane antibodies as detected by standard techniques. Am J Kidney Dis 39: 1162-1167. https://doi.org/10.1053/ajkd.2002.33385
18. Olaru F, Wang XP, Luo W, Ge L, Miner JH, et al. (2013) Proteolysis breaks tolerance toward intact α345(IV) collagen, eliciting novel anti-glomerular basement membrane autoantibodies specific for α345NC1 hexamers. J Immunol 190: 1424-1432. https://doi.org/10.4049/jimmunol.1202204
19. Jia XY, Qu Z, Cui Z, Yang R, Zhao J, et al. (2012) Circulating anti-glomerular basement membrane autoantibodies against α3(IV)NC1 undetectable by commercially available enzyme-linked immunosorbent assays. Nephrology 17: 160-166. https://doi.org/10.1111/j.1440-1797.2011.01511.x
20. Mahler M, Radice A, Sinico RA, Damoiseaux J, Seaman A, et al. (2012) Performance evaluation of a novel chemiluminescence assay for detection of anti-GBM antibodies: an international multicenter study. Nephrol Dial Transplant 27: 243-252. https://doi.org/10.1093/ndt/gfr203
21. Alchi B, Griffiths M, Sivalingam M, Jayne D, Farrington K (2015) Predictors of renal and patient outcomes in anti-GBM disease: clinicopathologic analysis of a two-centre cohort. Nephrol Dial Transplant 30: 814-821. https://doi.org/10.1093/ndt/gfu399
22. Laczika K, Knapp S, Derfler K, Soleiman A, Hörl WH, et al. (2000) Immunoadsorption in goodpasture's syndrome. Am J Kidney Dis 36: 392-395. https://doi.org/10.1053/ajkd.2000.8993

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23. Tang W, McDonald SP, Hawley CM, Badve SV, Boudville NC et al. (2013) Anti-glomerular basement membrane antibody disease is an uncommon cause of end-stage renal disease. Kidney Int 83: 503-510. https://doi.org/10.1038/ki.2012.375

24. Lazor R, Bigay-Gamé L, Cottin V, Cadranel J, Decaux O, et al. (2007) Alveolar hemorrhage in anti-basement membrane antibody disease: a series of 28 cases. Medicine 86: 181-193. https://doi.org/10.1097/md.0b013e318067da56

25. Liu P, Waheed S, Boujelbane L, Maursetter LJ (2016) Multiple recurrences of anti-glomerular basement membrane disease with variable antibody detection: can the laboratory be trusted? Clin Kidney J 9: 657-660. https://doi.org/10.1093/ckj/sfw038

26. Gu B, Magil AB, Barbour SJ (2016) Frequently relapsing anti-glomerular basement membrane antibody disease with changing clinical phenotype and antibody characteristics over time. Clin Kidney J 9: 661-664. https://doi.org/10.1093/ckj/sfw048

27. McAdoo SP, Pusey CD (2017) Anti-glomerular basement membrane disease. Clin J Am Soc Nephrol. 12: 1162-1172. https://doi.org/10.2215/cjn.01380217