WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.453
Volume 15, Issue 2, 1006-1015. Case Study ISSN 2277–7105

A CASE STUDY ON GOODPASTURE SYNDROME

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Article Received on 15 Dec. 2025,
Article Revised on 05 Jan. 2026,
Article Published on 16 Jan. 2026,

https://doi.org/10.5281/zenodo.18266492

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How to cite this Article: Saheena C.*[1], Vasantha Priya[2], K. Arbiya Ambreen Taj[2], D. Keerthana[3], Dr. Syed MD H.[4]. (2026). A Case Study on Goodpasture Syndrome, World Journal of Pharmaceutical Research, 15(2), 1006–1015.

ABSTRACT

Goodpasture syndrome is a rare autoimmune disorder characterized by circulating anti–glomerular basement membrane (anti-GBM) antibodies that cause inflammatory damage to the kidneys and occasionally, the lungs. Because early clinical manifestations are often nonspecific, diagnosis is frequently delayed, resulting in rapid disease progression and irreversible organ damage. We report a case of a 56-year-old male who presented with altered sensorium and reduced responsiveness without fever or respiratory symptoms. Laboratory investigations revealed anemia, elevated serum creatinine and blood urea levels, hematuria and proteinuria, suggestive of acute renal involvement. Serological testing confirmed positivity for anti-GBM antibodies and ultrasonography demonstrated bilateral grade I renal parenchymal disease. The patient's underlying type 2 diabetes mellitus may have contributed to baseline renal impairment and disease severity. This case highlights the importance of maintaining a high index of suspicion and early testing for anti-GBM antibodies in patients with acute renal dysfunction. Prompt diagnosis and timely initiation of immunosuppressive therapy are essential to prevent disease progression and improve clinical outcomes.

KEYWORDS: Goodpasture syndrome, Anti-GBM antibodies, Crescentic glomerulonephritis, Autoimmune renal disease.

INTRODUCTION

Goodpasture's syndrome (GS) is a rare and organ-specific autoimmune disease. It is mediated by anti-glomerular basement membrane (anti-GBM).[1] It affects both pulmonary capillaries and glomerular capillaries caused by the deposition in alveolar and glomerular basement membrane of circulating autoantibodies.[2] GS has pathology characterized by crescentic glomerulonephritis with linear immunofluorescent staining for IgG on the GBM.[1] Goodpasture syndrome was first described in 1958 by Australian scientists Stanton and Tange, who reported nine cases of rare glomerulonephritis closely linked with unexplained pulmonary hemorrhage. One of their cases involved a man admitted to hospital with anemia, coughing up blood and signs of bronchopneumonia in the right lung, who died after three days. Urinalysis showed traces of albumin, suggesting glomerulonephritis. A postmortem examination revealed red blood cells filling the terminal bronchioles and necrosis of alveolar walls. The disease was named in recognition of similar case reports described by Dr. Ernest Goodpasture in his 1919 publication on the influenza pandemic. At that time, similar clinical presentations of lung hemorrhage linked with glomerulonephritis were attributed to atypical influenza infection.[3] whereby the glomerular basement membrane was first identified as an antigen in the 1950s that the autoimmune mechanism behind Goodpasture syndrome was understood.[4]

Epidemiology

- Anti-GBM disease is a rare small-vessel vasculitis.
- It accounts for 1–2% of acute glomerulonephritis cases and 10–15% of rapidly progressive crescentic glomerulonephritis cases. (Fig-01)
- In Europe and Asia, the incidence is estimated to be 0.5–1.8 cases per million people per year. (Fig-02)
- In the United States, the prevalence is 10 cases per million hospitalized patients. (Fig-02)
- It has been reported mainly in White and Asian individuals and is less common in African individuals.[2]

Etiology

1. **Interaction of Environment + Genetics**

- Goodpasture syndrome occurs due to environmental or infectious triggers acting on a genetically susceptible person.

2. Genetic Predisposition

- Strong genetic involvement is seen, especially with certain HLA types.
- HLA-DR2 is present in up to 80% of patients with anti-GBM disease.

3. Mechanism of Triggering Injury

- Disease likely begins when an initial injury to the renal glomerular basement membrane exposes hidden antigens.
- Environmental exposures cause inflammation and make these antigens accessible to autoantibodies.

4. Environmental / Triggering Factors

Factors linked to the development of anti-GBM disease include

- Smoking / tobacco exposure.
- Exposure to metal dust, organic solvents or hydrocarbons.
- Bacteremia.
- Endotoxemia.
- Influenza A infection.
- Drugs causing T-cell depletion (e.g., alemtuzumab).
- Inhalation of cocaine.
- Exposure to volatile hydrocarbons.[5]

Clinical presentation

➢ Early signs and symptoms of Goodpasture syndrome may include :

- Feeling very weak or tired.
- Nausea (feeling sick to your stomach).
- Vomiting (throwing up).

Kidney-related symptoms include

- Blood in the urine (hematuria).
- Protein in the urine (proteinuria).
- Edema (swelling).
- High blood pressure.[6]

Pathophysiology

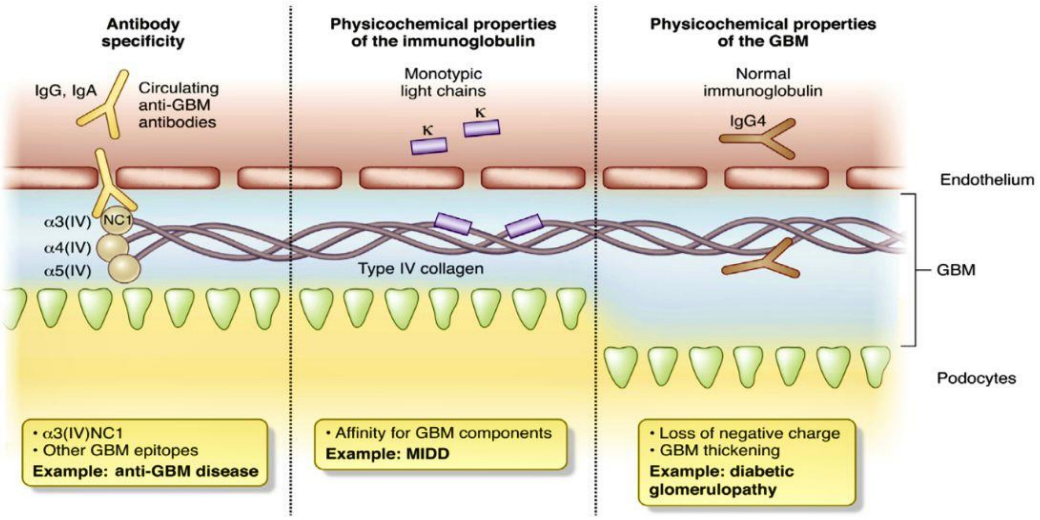

Fig. 1: Basic Structure of GBM Type IV Collagen

1. Basic Structure of Type IV Collagen

- Type IV collagen is made of three α-chains that form a triple helix.
- Each chain has

✓ An NC1 (non-collagenous) domain at the end.

✓ A long collagenous region with interruptions.

✓ A 7S domain at the other end.

2. Type IV Collagen in the Glomerular Basement Membrane (GBM)

- In GBM, collagen uses five types of α-chains (α1–α5) and possibly α6.
- These α-chains combine to form three main triple-helical molecules

✓ $(\alpha1)_2\alpha2$

✓ $(\alpha3)_2\alpha4$

✓ $(\alpha5)_2\alpha?$

3. NC1 Domains Form Higher-Order Structures

- Triple helices are linked through their NC1 domains.
- NC1 monomers pair to form NC1 dimers, and then dimers join to form NC1 hexamers.[7]

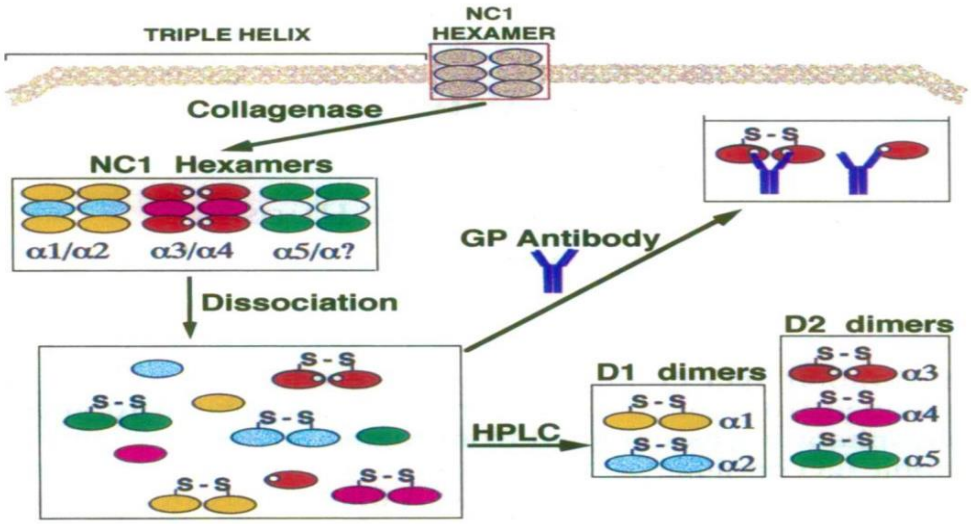

Fig. 2: Structure of GBM non-collagenous domain 1 (NC1) immunogens.

4. How NC1 Hexamers Are Studied

- Collagenase digestion releases NC1 hexamers from the collagen structure.
- These hexamers differ depending on which α-chain NC1 subunits they contain.

5. Goodpasture (GP) Antibody Binding

- The GP epitope is located on the α3 NC1 domain.
- In the intact hexamer, this epitope is hidden/sequestered.
- When the hexamer is disrupted (by denaturation), the epitope becomes exposed making it accessible to circulating anti-GBM antibodies.
- Antibodies bind to the exposed α3 NC1 domain that causes immune attack on the GBM leading to Goodpasture's disease.

➢ Color Coding in the Figure 05

- α1 = yellow
- α2 = light blue
- α3 = red
- α4 = magenta
- α5 = green[8]

Diagnosis

1. Physical examination

Physical examination findings in patients with anti-GBM disease include the following

- Tachypnea (rapid breathing)
- Inspiratory crackles heard over the lung bases
- Cyanosis
- Hepatosplenomegaly
- Hypertension
- Skin rash.

2. Blood tests

- Blood tests may show elevated waste products, suggesting kidney dysfunction.
- BUN (Blood Urea Nitrogen) and serum creatinine levels may be increased due to impaired renal function.
- Blood tests may detect anti-GBM antibodies, confirming the disease.

3. Urine tests (Urine analysis)

- Findings are typical of acute glomerulonephritis.
- Low-grade proteinuria.
- Gross or microscopic hematuria.

4. Anti-GBM antibody testing

- Serologic tests for anti-GBM antibodies are important for confirming the diagnosis and monitoring treatment response**.**
- Radioimmunoassays and ELISAs for anti-GBM antibodies are highly sensitive (>95%) and highly specific (>97%).
- Positive results should be confirmed by western blot, especially if a kidney biopsy is not performed.

5. Chest radiograph

- Chest X-ray typically shows patchy parenchymal consolidations**.**
- Apices and costophrenic angles are usually spared**.**
- Up to 18% of patients may have a normal chest X-ray.

6. Biopsy

- Kidney biopsy is recommended in patients with diffuse alveolar hemorrhage and renal involvement to determine the cause and guide treatment.

- Percutaneous kidney biopsy is the preferred method for diagnosing anti-GBM disease.[1]

Treatment

➢ The treatment for Goodpasture syndrome centers on a triple-therapy approach :

- Plasma exchange to remove antibodies.
- Corticosteroids to reduce inflammation.
- Cyclophosphamide to suppress the immune system.

➢ For patients who cannot tolerate the standard regimen, Alternative Therapies include Rituximab or Mycophenolate Mofetil.[2]

CASE PRESENTATION

A 56-year-old male patient was admitted to vijayanagara institute of medical science(VIMS), Ballari (Karnataka) with chief complaints of altered sensorium since 3am in the morning in the form of reduced responsiveness. There was no history of fever, chest pain, weight loss, or recent infection.

On day 5 the patient was adviced to check for GBM Antibodies test were it was found to be positive(++).

Social History - No habits

Past History - K/C/O Type-II Diabetes mellitus since 7 years on medication T.Glycomet GP 2mg

Family History - Nothing significant

On Examination

- BP- 130/80mm/hg
- PR- 86bpm
- SPO2- 98% decreased at RA
- GRBS- 21mg/dl
- Patient was Drowsy
- Pupil- BERL(Bilaterally equally reactive to light)
- E2V2M6(GCS reduced consciousness)
- S1 S2 heard
- P/A was soft and non-tender.

Laboratory Investigations

PARAMETERS	RESULT (30-07-25)	RESULT (02-08-25)	RESULT (04-08-25)	RESULT (05-08-25)	REFERENCE RANGE
Haemoglobin	8.3	7.3	8.4	8.3	13-18gm/dl
WBC	16870	16810	18000	16110	4000-11000 cells/cumm
Neutrophils	83	88	88	91	40-70%
Lymphocytes	12	08	10	04	20-40%
RBC count	3.51	3.00	3.47	3.38	5.5-6.5 million/cumm
PCV	24.2	20.8	23.8	22.9	45-55%
RBS	40				70-140mg/dl
Albumin	2.1	2.2			3.2-5.4g/dl
Sr.creatinine	3.8	6.2	6.4	0.6	0.7-1.4mg/dl
Blood urea	51	94	129	78	15-45mg/dl
Urine albumin	Trace		Trace		
Urineprotein-Cr ratio	420				10-150mg/g
Mean blood glucose	120				70-99mg/dl
Microscopy			5-6 pus cells/HPF plenty of RBCS/HPF		

➢ **Other laboratory investigations**

- GBM-Antibodies – Positive (++).
- USG Abdomen - B/L grade-I Renal paranchymal disease.

Treatment

SL.NO	NAME OF MEDICATIONS	DOSE	ROUTE	FREQUENCY
01	INJ.25%DEXTROSE		IV	TID FOR 2 DAYS
02	IV FLUIDS	3PINT DNS	IV	FOR 2 DAYS
03	INJ PANTOPRAZOLE	40mg	IV	OD FOR 9 DAYS
04	INJ ONDANSETRON	4mg	IV	TID FOR 9 DAYS
05	INJ CEFTRIAXONE	1gm	IV	BD FOR 5 DAYS
06	TAB LEVIPIL	500mg	PO	BD FOR 9 DAYS
07	TAB TELMISARTAN	20mg	PO	OD FROM 4TH DAY FOR 4 DAYS
08	TAB DAPAGLIFLOZIN	50mg	PO	OD FROM 4TH DAY FOR 2 DAYS
09	SYP SUCRALFATE	5ml	PO	TID FROM 4TH DAY FOR 6 DAYS
10	TAB SOBOSIS	500mg	PO	TID FROM 4TH DAY FOR 6 DAYS
11	TAB IFA	333mg	PO	BD FROM 4TH DAY

				FOR 2 DAYS
12	TAB FOLIC ACID	5mg	PO	OD FROM 4TH DAY FOR 2 DAYS
13	TAB CALCIUM	500mg	PO	OD FROM 4TH DAY FOR 5 DAYS
14	INJ FUROSEMIDE	100mg	IV	BD FROM 4TH DAY FOR 6 DAYS
15	TAB TORSEMIDE	100mg	PO	BD FROM 4TH DAY FOR 4 DAYS
[16	INJ PIPZO	2.25gm	IV	TID FROM 6TH DAY FOR 3 DAYS
17	INJ.METHYLPREDNISOLONE	1gm in 100ml NS	IV	OD FROM 6TH DAY FOR 3 DAYS

DISCUSSION

It is a rare autoimmune disease that mainly affects the kidneys. Diagnosis is often delayed because the condition is uncommon and the early symptoms are nonspecific. Laboratory investigations showed anemia, elevated serum creatinine, and increased blood urea levels, with the presence of traces of blood and protein in the urine, indicating kidney damage caused by inflammation of the glomerular basement membrane. As confirmatory evidence for Goodpasture syndrome, anti-GBM antibodies were positive. To support these findings, ultrasonography of the abdomen indicated bilateral parenchymal kidney disease. The patient's past history of diabetes mellitus had already compromised renal function, contributing to the severity of the disease. The patient was treated with a high dose of intravenous methylprednisolone to suppress the immune response. Other treatments included antibiotics, diuretics, antiepileptics, along with fluid management, which were given as supportive therapy.

CONCLUSION

This case highlights that Goodpasture syndrome is a rare but serious autoimmune condition. Early detection of anti-GBM antibodies is essential for confirming the diagnosis and initiating appropriate treatment. Rational immunosuppressive therapy, along with supportive care, helps reduce disease progression. This case emphasizes the need for early detection, accurate diagnosis, and the use of appropriate pharmacotherapy. Reporting such cases helps increase awareness among healthcare professionals and may be useful for early recognition of the condition in patients.

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