orphanaesthesia

Anesthesia recommendations for

Goodpasture syndrome

Disease name: Goodpasture syndrome

ICD 10: M31.0

Synonyms: Goodpasture's syndrome (GS), anti-glomerular basement membrane disease (Anti-GBM disease), crescentic glomerulonephritis type 1, GPS

Disease summary: Goodpasture syndrome is a rare, organ-specific autoimmune disease (Gell and Coombs classification type II). It is mediated by anti-glomerular basement membrane (anti-GBM) antibodies [1]. The disease was first described by Dr Ernest Goodpasture in 1919 [2], and the glomerular basement membrane was subsequently identified as the target antigen in the 1950s. More than a decade later, researchers demonstrated the association between antibodies derived from affected kidneys and nephritis [1].

The disorder is characterized by autoantibodies directed against the NC1 domain of the α3 chain of type IV collagen (α3(IV)NC1) in the glomerular and alveolar basement membranes, leading to activation of the complement cascade and subsequent tissue injury [1, 3]. In addition to α3(IV)NC1, other components of the glomerular basement membrane, such as peroxidasin and laminin-521, have recently been identified as target antigens [4]. The exclusive presence of this α3 subunit in basement membranes of the lungs and kidneys accounts for the selective involvement of these two organs in GPS [1]. This distinct immunopathology underlies the entity historically referred to as GPS. The term GPS was historically used to describe pulmonary-renal involvement in the presence of anti-GBM antibodies [5]. However, the preferred term today is anti-GBM disease, as atypical forms have since been described [5]. Typical cases involve IgG autoantibodies against the α3(IV)NC1 domain, usually resulting in a positive ELISA and rapidly progressive glomerulonephritis with or without pulmonary hemorrhage [5]. Atypical variants may be seronegative in standard ELISAs and involve other immunoglobulin subclasses such as IgA or IgG4; these forms often show milder or incomplete clinical manifestations – such as isolated renal or pulmonary disease – while retaining similar histological features to classic anti-GBM disease [5].

The etiology and triggering factors for anti-GBM production remain unknown. Because patients with specific human leukocyte antigen (HLA) types are more susceptible, an HLA-associated genetic predisposition appears likely [1, 6]. However, because this strongly associated allele is relatively common, additional behavioural or environmental factors are believed to influence immune response and disease expression. These may include respiratory infections (e.g., influenza A2), exposition to hydrocarbon fumes, organic solvents, metallic dust, tobacco smoke, certain drugs (e.g., rifampicin, allopurinol, cocaine), as well as physical damage to basement membrane (e.g., lithotripsy or membranous glomerulonephritis) or lymphocyte-depletion therapy (such as alemtuzumab), although conclusive evidence is lacking [1, 3, 6, 7].

The incidence is estimated at approximately 0.5 to 2 cases per million population per year in European Caucasoid and Asian groups [1, 8, 9]. Unusually for an autoimmune disease, GPS

affects more males than females among Caucasians and is even more prevalent in the Maori people of New Zealand [8]. It accounts for acute renal failure in approximately 10-20 % of cases of rapidly progressive or crescentic glomerulonephritis. The age distribution shows two peaks – one between 20 and 30 years (often with more pronounced hemorrhagic features) and another between 60 and 70 years. In the younger group, men are more frequently affected, whereas in the older age group, women predominate [1, 6]. Furthermore, older patients more often present with isolated renal involvement [10].

GPS typically presents as acute renal failure caused by a rapidly progressive glomerulonephritis, often accompanied by pulmonary hemorrhage that can be life-threatening without prompt diagnosis and treatment [1, 3]. Symptoms may develop gradually or progress rapidly within a few days [11]. Systemic and nonspecific initial symptoms such as fatigue, weakness, lethargy, nausea, vomiting, diarrhea, pruritus, loss of appetite, and weight loss are common. Patients may also experience malaise, chills, fever, headache, arthralgias, pallor, general discomfort, or, in rare cases, seizures [1, 6, 12-14].

About 60-80 % of patients present with both renal and pulmonary involvement, whereas 20-40 % exhibit renal disease alone, and fewer than 10 % have isolated pulmonary involvement [1, 6, 8]. Pulmonary symptoms include hemoptysis, dry cough, shortness of breath, inspiratory crackles over lung bases, chest pain, cyanosis, dyspnea, tachypnea, which may progress to respiratory failure [1]. In children, pulmonary findings rarely occur before puberty [15].

Renal involvement may result in hematuria, foamy urine, peripheral swelling, high blood pressure, edema, uremia, oliguria, anuria, and flank pain [1, 6, 7]. Autoimmune inner ear disease (AIED) may also occur, presenting with vertigo, tinnitus (ringing, hissing or roaring) and sudden hearing loss in one ear, progressing rapidly to the other within weeks or months [1]. More than 90 % of the patients with GPS have circulating serum anti-GBM antibodies [16].

A definitive diagnosis is established by percutaneous kidney biopsy showing the characteristic linear IgG deposition along the glomerular basement membrane (preferred over lung biopsy) and confirmed by immunofluorescence and enzyme-linked immunosorbent assay (ELISA) testing for pathognomonic circulating anti-GBM antibodies [1]. Chemiluminescence immunoassay (ChLIA) offers a highly sensitive alternative for anti-GBM antibody detection [5]. Atypical variants, including IgA- or IgG4-dominant forms may present with milder or incomplete clinical courses, occasionally resulting in false-negative ELISA findings and diagnostic uncertainty [5].

Differential diagnosis include Granulomatosis with polyangiitis (formerly Wegener's granulomatosis), systemic lupus erythematosus, microscopic polyangiitis, other forms of systemic vasculitides (e.g., Churg-Strauss syndrome, essential mixed cryoglobulinaemia, Henoch-Schönlein purpura, microscopic polyarteritis, drug-induced vasculitis), pulmonary embolism, and other inflammatory or infectious disorders such as Pneumocystis carinii pneumonia or rheumatoid arthritis [1, 6, 8].

Because of the rarity of the disorder, systematic data and controlled therapeutic trials are lacking. Nevertheless, rapid recognition and treatment are crucial in GPS. The three key therapeutic principles are: (1) rapid removal of circulating antibodies, primarily by plasmapheresis; immunoadsorption represents an alternative to plasma exchange with comparable efficacy (2) inhibition of further antibody production through immunosuppressive therapy (high-dose corticosteroids and cyclophosphamide are standard, though other agents as azathioprine or rituximab may be used); and (3) elimination of potential triggering agents that may have initiated antibody formation [1, 17]. Imlifidase, an IgG-cleaving enzyme, is currently being investigated as a new therapeutic approach, among others, for GPS [17, 18]. Atypical forms (IgA-dominant, seronegative, or isolated pulmonary) require individualized treatment [17].

Renal replacement therapy (RRT) or kidney transplantation may be required to restore renal function. Most centers recommend at least six months of sustained negative anti-GBM antibody testing before undertaking transplantation [19]. Several case reports describe the use of extracorporeal membrane oxygenation (ECMO) for refractory hypoxemic respiratory failure in patients with severe pulmonary hemorrhage due to GPS [7, 11, 15, 20, 21].

GPS carries a poor prognosis, which largely depends on the timing of diagnosis and initiation of treatment for this rapidly progressive disease. If left untreated, mortality ranges from 77-96% [7]. With the triple therapeutic regimen comprising corticosteroids, immunosuppressive agents, and plasmapheresis, one-year survival rates of 70-90% and five-year survival up to 80 % have been reported [1, 6]. Among patients with RRT for end-stage renal disease in New Zealand and Australia, the median survival was nearly six years [9]. In addition to age, a history of pulmonary hemorrhage is associated with an increased risk of mortality while on RRT [9, 21]. Patients who are dialysis-dependant at presentation rarely achieve full recovery of renal function. However, recent population-based data suggest a 5.8 % one-year recovery rate of kidney function among dialysis-dependent patients with anti-GBM disease [22]. Those requiring only temporary RRT may regain satisfactory renal function, and fewer than 30 % of surviving patients remain dependant on long-term dialysis [1, 6, 21, 23].

Double positivity for serum antineutrophil cytoplasmic antibodies (ANCAs) and anti-GBM is another indicator of poorer renal prognosis and higher mortality [6]. Pulmonary involvement, by contrast, often resolves completely with prompt and adequate treatment [8, 20]. Low serum C3 complement levels have also been associated with a poorer overall and renal prognosis in GPS [24]. Long-term outcomes in children with GPS may be more favorable than in adults [15].

A	**AIRWAY / ANESTHETIC TECHNIQUE**	no special airway malformations, but GA may be challenging (due to respiratory status) – availability of tracheal suction catheter / bronchoscopy (pulmonal bleeding) – anesthesia only / in cases of stable disease if applicable – be aware of severe / refractory hypoxemic failure due to pulmonal hemorrhage with necessity of VV-ECMO – consider neuraxial / peripheral RA as a feasible alternative if applicable
B	**BLOOD PRODUCTS (COAGULATION)**	be prepared for recurring transfusions in case of persistent intrapulmonary bleeding (sufficient storage of blood products) – be aware of low platelets and fibrinogen in patients undergoing plasmapheresis
C	**CIRCULATION**	anticipate hemodynamic deviation due to pre-existing hypertension when undergoing anesthesia – consider IBP (blood gas analysis) and (non-)invasive hemodynamic (to avoid fluid overload)
D	**DRUGS**	no risk for MH – be aware of secondary insufficiency of the adrenal glands (long-term corticosteroid-therapy) and infectious complications (immunosuppressive therapy) – consider drug dose adaption in case of renal impairment
E	**EQUIPMENT**	use ultrasound for vessel cannulation / peripheral RA (edema, swollen limbs) – perioperative availability of dialysis may be necessary

Typical surgery and procedures

Percutaneous kidney biopsy and alternatively transbronchial or (rarely) open lung biopsy for diagnosis [1].

Bronchoscopy / (rarely) thoracoscopy for diagnostic issues [1].

Renal transplantation [1, 25].

Arteriovenous fistula for dialysis.

Type of anesthesia

A general recommendation regarding an ideal anesthetic approach cannot be given, as there is a lack of data for anesthesia in patients with GPS. As usual and whenever possible, anesthesia should only be considered in cases of a stable disease.

Anesthesiologists / intensive care physicians are often involved for securing the airway in instable GPS patients with pulmonary hemorrhage.

General anesthesia may be challenging due to the patient's respiratory status in GPS. Neuraxial (epidural) anesthesia has been used in a patient with GPS undergoing bilateral nephrectomy, and was reported to be uneventful [26].

Peripheral regional anesthesia techniques are generally feasible in most patients. However, due to possible generalized edema, particulaly in the extremities, a landmark-guided approach may be difficult. Therefore, ultrasound-guided regional anesthesia is recommended.

Necessary additional pre-operative testing (beside standard care)

Chest radiograph: may show normal findings (18%) as well as infiltrates and patchy parenchymal consolidations, which are usually bilateral, symmetric perihilar and bibasilar [1, 8, 11].

Chest radiograph / ultrasound: pleural effusions are unusual but can occur [1, 8].

Computed tomography: may reveal localized / nodular / diffuse / extensive / bilateral infiltrates with a pattern of alveolar damage [11].

Pulmonary function test: may help to better characterize or identify further progress of pulmonal impairment before scheduled surgery.

Blood testing: may show anemia of variable degree (due to pulmonary bleeding or often secondary to iron deficiency as well as renal failure), high level of waste products as well as elevated blood urea nitrogen (BUN) and serum creatinine levels.

Urine analysis: proteinuria and / or hematuria may be observed.

Particular preparation for airway management

As far as is known, GPS itself is not associated with specific anatomic airway abnormalities. Nevertheless, a standardized approach to airway assessment and identification of airway potential challenges is recommended. Comprehensive preparation for airway management should be guided by the evaluation results.

In cases of pulmonary bleeding, urgent endotracheal intubation may be required. Depending on the severity of hemoptysis and pulmonary bleeding in the individual patient, video-assisted or fiberoptic intubation techniques may be considered. Direct laryngoscopy should also be available, as it may represent the more reliable option for some anesthesiologists, particularly in active bleeding situations.

Because pulmonary bleeding in GPS is typically bilateral, lung separation techniques are typically not useful for isolating a “good” from a “bad” lung. In cases of refractory bleeding with severe hypoxemia, urgent transfer to an ECMO center may be the only live-saving measure, as venovenous ECMO (VV-ECMO) can serve bridge-to-recovery in conjunction with appropriate medical therapy for GPS.

Particular preparation for transfusion or administration of blood products

No specific recommendations are available, and no typical bleeding disorders have been reported in patients with GPS. Nevertheless, complications may occur during plasmapheresis when platelet levels decrease and fibrinogen is removed.

Patients with GPS frequently present with significant anemia due to ongoing intrapulmonary bleeding, often requiring repeated transfusions [1, 7]. Therefore, depending on the planned surgical procedure and the extent of hemoptysis or pulmonal bleeding, an adequate supply of blood products should be ensured.

Particular preparation for anticoagulation

Especially in cases involving pulmonal hemorrhage, dialysis, or plasmapheresis, the method and intensity of anticoagulation should be carefully determined based on an individualized risk-benefit assessment and the type of surgery or procedure.

Particular precautions for positioning, transportation and mobilization

There are no specific recommendations for patients with GPS.

Interactions of chronic disease and anesthesia medications

Long-term corticosteroid therapy should be taken into account because of the potential of secondary adrenal insufficiency. Accordingly, the administration of additional “stress dose corticosteroids dosing” may help reduce the risk of perioperative adrenal crisis [27].

Immunosuppressive therapy may also be associated with an increased risk of infectious complications.

Anesthetic procedure

Preoperative evaluation: see details above.

Premedication: may be administered after carefully weighing the potential benefits and risks in each individual patient.

Patient positioning: no specific recommendations are available.

IV line: placement may be difficult due to swelling / edema of the limbs [1, 20].

Invasive blood pressure measurement: facilitates frequent arterial blood gas analysis, particularly in the presence of pulmonary and renal (e.g., altered potassium levels) impairment.

(Mechanical) ventilation: pulmonary restriction and reduced diffusing capacity are characteristic findings in patients with GPS. Ventilation should therefore be performed in a lung-protective manner, using appropiately low tidal volumes to prevent baro-/volutrauma. Frequent tracheal suctioning may be required because of pulmonal bleeding [26].

Anesthesia: both total intravenous and balanced anesthesia using volatile agents can be performed safely. The use of fentanyl, remifentanil and propofol has been reported as uneventful for induction and maintenance [26, 28]. An increased oxygen concentration in anesthetic gas mixture may be necessary in patients with pulmonary impairment, especially in the presence of ventilation-perfusion mismatch [26]. There are no absolute or relative known contraindications to specific anesthesic drugs attributable to GPS; however, drug dosages should be adjusted according to the degree of renal impairment. There is no known risk for malignant hyperthermia.

Regional anesthesia: may be performed as described above.

Particular or additional monitoring

(Advanced) Hemodynamic monitoring (invasive or non-invasive): may be reasonable to avoid fluid overload in patients with GPS [1].

Possible complications

Pulmonal bleeding / hemorrhage and resulting respiratory failure / hypoxemia as well as anemia.

Necessity of (long-term) dialysis due to renal failure.

Hemodynamic deviation due to pre-existing hypertension when undergoing anesthesia.

Infections in case of fulminant immunosuppression.

Post-operative care

Post-operative care should be based upon the patient's pre-existing conditions as well as the surgical or interventional procedure.

Particular attention should be given to monitoring respiratory and renal function as well as blood pressure. An appropriately extended stay in PACU, IMC or ICU before transfer to the general ward (or discharge at home) is considered acceptable.

Disease-related acute problems and effect on anesthesia and recovery

Emergency-like situations: diffuse alveolar or pulmonary hemorrhage may occur, requiring mechanical ventilation, repeated re-intubations, and in case of refractory hypoxemia due to pulmonary bleeding, the possible initiation of VV-ECMO [1, 8, 12, 20].

Ambulatory anesthesia

Specific recommendations for or against ambulatory anesthesia cannot be given as no published literature exists regarding this topic. In stable GPS disease, ambulatory procedures are possible if the patient is classified as ASA physical status I-III and has no relevant contraindications to outpatient anesthesia.

Obstetrical anesthesia

Patients with GPS are fertile; therefore, obstetrical anesthesiologists may encounter these patients for labor analgesia. During pregnancy, the disease can pose a serious threat to the lives of both mother and child. Moreover, anti-GBM antibodies may cross the placenta, potentially causing pulmonary-renal syndrome in the fetus with subsequent spontaneous abortion or stillbirth [5, 29]. In addition to the risk of prematurity, miscarriage may occur due to the teratogenic effects of essential therapies. Gestational diabetes may also develop as a consequence of corticosteroid treatment. Therefore, the optimal management of pregnant women with GPS requires intensive care in a center with appropiate expertise and close multidisciplinary collaboration among the attending specialists [14, 30, 31].

Hypertension and proteinuria are the most common findings in pregnant patients with GPS. Although differentiation is challenging during pregnancy, it is important to determine whether these findings result from preeclampsia or renal involvement related to GPS [31]. For definitive diagnosis, serologic testing and renal biopsy should be considered; the latter one does not appear to be associated with an increased risk during pregnancy [14]. In addition to blood pressure monitoring, management should include a serial assessment of renal function, hematologic parameters, and pulmonary function tests to evaluate for changes or hypoxemia [13]. Pregnancy-induced hypertension may further exacerbate renal function [14].

Both vaginal delivery and cesarean section have been reported in parturients with GPS [13, 31, 32]. In general, either neuraxial as well as general anesthesia may be used safely in this patient population. Severe anesthesia-related complications have not been reported. However, given the limited data on obstetric anesthesia in GPS, shared decision-making between the anesthesiologist and the patient is essential when selecting the most appropriate anesthetic technique.

References

1. Greco A, Rizzo MI, De Virgilio A, Gallo A, Fusconi M, Pagliuca G, et al. Goodpasture's syndrome: a clinical update. Autoimmun Rev. 2015;14(3):246–53.
2. Goodpasture EW. Landmark publication from The American Journal of the Medical Sciences: The significance of certain pulmonary lesions in relation to the etiology of influenza. Am J Med Sci. 2009;338(2):148–51.
3. Salant DJ. Goodpasture's disease--new secrets revealed. N Engl J Med. 2010;363(4):388–91.
4. Kuang H, Liu J, Jia XY, Cui Z, Zhao MH. Autoimmunity in Anti-Glomerular Basement Membrane Disease: A Review of Mechanisms and Prospects for Immunotherapy. Am J Kidney Dis. 2023;81(1):90–9.
5. Reggiani F, L'Imperio V, Calatroni M, Pagni F, Sinico RA. Goodpasture syndrome and anti-glomerular basement membrane disease. Clin Exp Rheumatol. 2023;41(4):964–74.
6. Dammacco F, Battaglia S, Gesualdo L, Racanelli V. Goodpasture's disease: a report of ten cases and a review of the literature. Autoimmun Rev. 2013;12(11):1101–8.
7. Herbert DG, Buscher H, Nair P. Prolonged venovenous extracorporeal membrane oxygenation without anticoagulation: a case of Goodpasture syndrome-related pulmonary haemorrhage. Crit Care Resusc. 2014;16(1):69–72.
8. Sinha VK, Hibbert C. Near-lethal acute kidney injury due to Goodpasture's syndrome: A case report. J Intensive Care Soc. 2015;16(4):350–4.
9. Tang W, McDonald SP, Hawley CM, Badve SV, Boudville NC, Brown FG, et al. Anti-glomerular basement membrane antibody disease is an uncommon cause of end-stage renal disease. Kidney Int. 2013;83(3):503–10.
10. El-Zaatari ZM. Anti-Glomerular Basement Membrane Glomerulonephritis. N Engl J Med. 2023;389(20):1901.
11. Balke L, Both M, Arlt A, Rosenberg M, Bewig B. Severe adult respiratory distress syndrome from Goodpasture syndrome. Survival using extracorporeal membrane oxygenation. Am J Respir Crit Care Med. 2015;191(2):228–9.
12. Ting IP, Abdul Halim S, Adnan A, Jaafar H. Status epilepticus as the initial presentation of antibody-negative Goodpasture's syndrome. BMJ Case Rep. 2017;2017.
13. Wells SR, Kuller JA, Thorp JM, Jr. Pregnancy in a patient with Goodpasture syndrome and renal transplantation. Am J Perinatol. 1996;13(2):79–80.
14. Vasiliou DM, Maxwell C, Shah P, Sermer M. Goodpasture syndrome in a pregnant woman. Obstet Gynecol. 2005;106(5 Pt 2):1196–9.
15. Menzi CP, Bucher BS, Bianchetti MG, Ardissino G, Simonetti GD. Management and outcomes of childhood Goodpasture's disease. Pediatr Res. 2018;83(4):813–7.
16. Collard HR, Schwarz MI. Diffuse alveolar hemorrhage. Clin Chest Med. 2004;25(3):583–92, vii.
17. McAdoo SP, Pusey CD. Anti-glomerular basement membrane disease - treatment standard. Nephrol Dial Transplant. 2025.
18. Uhlin F, Szpirt W, Kronbichler A, Bruchfeld A, Soveri I, Rostaing L, et al. Endopeptidase Cleavage of Anti-Glomerular Basement Membrane Antibodies in vivo in Severe Kidney Disease: An Open-Label Phase 2a Study. J Am Soc Nephrol. 2022;33(4):829–38.
19. McAdoo SP, Pusey CD. Antiglomerular Basement Membrane Disease. Semin Respir Crit Care Med. 2018;39(4):494–503.
20. Dalabih A, Pietsch J, Jabs K, Hardison D, Bridges BC. Extracorporeal membrane oxygenation as a platform for recovery: a case report of a child with pulmonary hemorrhage, refractory hypoxemic respiratory failure, and new onset goodpasture syndrome. J Extra Corpor Technol. 2012;44(2):75–7.
21. Levy JB, Turner AN, Rees AJ, Pusey CD. Long-term outcome of anti-glomerular basement membrane antibody disease treated with plasma exchange and immunosuppression. Ann Intern Med. 2001;134(11):1033–42.

22. Kwong YD, Liu KD, Hsu RK, Johansen KL, McCulloch CE, Seth D, et al. Recovery of Kidney Function Among Patients With Glomerular Disease Starting Maintenance Dialysis. Am J Kidney Dis. 2021;77(2):303–5.
23. Marques C, Carvelli J, Biard L, Faguer S, Provot F, Matignon M, et al. Prognostic Factors in Anti-glomerular Basement Membrane Disease: A Multicenter Study of 119 Patients. Front Immunol. 2019;10:1665.
24. Zhu M, Wang J, Le W, Xu F, Jin Y, Jiao C, et al. Relationship Between Serum Complement C3 Levels and Outcomes Among Patients With Anti-GBM Disease. Front Immunol. 2022;13:929155.
25. Lahmer T, Kuchle C, Schirmer L, Heemann U, Lutz J, Thurmel K. Kidney transplant after preexisting posterior reversible encephalopathy syndrome induced by Goodpasture's syndrome. Exp Clin Transplant. 2012;10(3):299–301.
26. Saegusa R, Ando M, Nishino T, Yonezawa T. Anesthetic management of bilateral nephrectomy in Goodpasture's syndrome. Masui. 1977;26(3):335–41.
27. Woodcock T, Barker P, Daniel S, Fletcher S, Wass JAH, Tomlinson JW, et al. Guidelines for the management of glucocorticoids during the peri-operative period for patients with adrenal insufficiency: Guidelines from the Association of Anaesthetists, the Royal College of Physicians and the Society for Endocrinology UK. Anaesthesia. 2020;75(5):654–63.
28. Fukuhara A, Okutani R, Oda Y. Anesthesia for living donor renal transplantation in a patient with Goodpasture's syndrome with a history of repeated alveolar hemorrhage. Masui. 2013;62(10):1199–202.
29. Thomson B, Joseph G, Clark WF, Hladunewich M, Patel A, Blake P, et al. Maternal, pregnancy and fetal outcomes in de novo anti-glomerular basement membrane antibody disease in pregnancy: a systematic review. Clin Kidney J. 2014;7(5):450–6.
30. Hatfield T, Steiger R, Wing DA. Goodpasture's disease in pregnancy: case report and review of the literature. Am J Perinatol. 2007;24(10):619–21.
31. Huser M, Wagnerova K, Janku P, Malaskova L, Stourac P. Clinical management of pregnancy in women with Goodpasture syndrome. Gynecol Obstet Invest. 2015;79(2):73–7.
32. Shah A, Bailey E, Hughes S. Goodpasture's syndrome, haemodialysis and pregnancy. Br J Hosp Med (Lond). 2007;68(1):48–9.

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