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ANTI-GLOMERULAR BASEMENT MEMBRANE DISEASE: CURRENT INSIGHTS INTO ETIOPATHOGENESIS, CLINICAL MANIFESTATIONS, DIAGNOSIS, AND THERAPEUTIC STRATEGIES

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The purpose of the study was to perform a bibliometric analysis of the global catalog of scientific literature indexed in leading scientometric databases, in order to identify the chronological dynamics of anti-glomerular basement membrane disease detection and to analyze data on its etiopathogenesis, clinical manifestations, diagnostics, and treatment strategies. The literature search was conducted using a specialized query that combined terms related to hemorrhagic pulmonary-renal syndrome in the context of anti-glomerular basement membrane disease (Goodpasture syndrome). The year of publication, document type, industry affiliation, as well as geographical and institutional distribution were analyzed using a quantitative-structural-analytical method. The results of the analysis demonstrated that original clinical studies and systematic reviews were the most common, and that the overall structure reflects the predominance of empirical research in the study of anti-glomerular basement membrane disease, highlighting the high level of clinical relevance, validity, and reliability of the information. The lack of information about this life-threatening pathology, the ambiguity of its epidemiology, the rarity of documented cases of anti-glomerular basement membrane disease, insufficient understanding of autoimmune reactions, variability of its clinical manifestations, and the growing interest in this issue due to increasing morbidity, among patients-especially after coronavirus disease-determine the relevance of the study.

Key words: anti-glomerular basement membrane disease, Goodpasture syndrome, hemorrhagic pulmonary-renal syndrome, antibodies to glomerular basement membrane, antibodies to alveolar basement membrane.

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ХВОРОБА, АСОЦІЙОВАНА З АНТИТІЛАМИ ДО ГЛОМЕРУЛЯРНОЇ БАЗАЛЬНОЇ МЕМБРАНИ: СУЧАСНІ УЯВЛЕННЯ ПРО ЕТІОПАТОҐЕНЕЗ, КЛІНІЧНІ ПРОЯВИ, ДІАГНОСТИКУ І ТЕРАПЕВТИЧНІ СТРАТЕГІЇ

Метою дослідження було проведення бібліометричного аналізу світового каталогу наукової літератури, індексованої у провідних наукометричних базах даних із метою виявлення хронологічної динаміки виявлення хвороби, асоційованої з антитілами до гломерулярної базальної мембрани, аналізу даних щодо її етіопатогенезу, клінічних проявів, сучасних принципів діагностики та лікувальної тактики. Пошук літературних джерел здійснювався шляхом спеціалізованого пошукового запиту, що поєднував терміни, пов'язані з геморагічним легенево-нирковим синдромом на тлі хвороби, асоційованої з антитілами до гломерулярної базальної мембрани (синдрому Гудпасчера). За допомогою кількісно-структурно-аналітичного методу було проаналізовано рік публікації, тип документа, галузева приналежність, географічний та інституційний розподіл. Результати аналізу показали, що найпоширенішими були оригінальні клінічні дослідження та систематичні огляди, що структура відображає переважання емпіричних досліджень при вивченні хвороби, асоційованої з антитілами до гломерулярної базальної мембрани, підкреслює високий рівень клінічної значущості, актуальності та достовірність інформації. Недостатність інформації про цю життєзагрожуючу патологію, неоднозначність її епідеміології, рідкісність задокументованих випадків хвороби, асоційованої з антитілами до гломерулярної базальної мембрани, недостатнє розуміння аутоімунних реакцій, варіабельність її клінічних проявів, зростання інтересу до цієї проблеми, внаслідок збільшення захворюваності, особливо – після перенесеної коронавірусної хвороби, визначають актуальність дослідження.

Ключові слова: хвороба, асоційована з антитілами до гломерулярної базальної мембрани, синдром Гудпасчера, геморагічний легенево-нирковий синдром, антитіла до базальної мембрани клубочка, антитіла до базальної мембрани альвеол.

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Anti-glomerular basement membrane disease (anti-GBM disease) is a disease associated with antibodies to the glomerular basement membrane (GBM) (formerly Goodpasture syndrome). This disease is a rare but life-threatening, rapidly progressive, organ-specific systemic vasculitis with a predominant involvement of the kidneys and lungs small vessels [1, 2, 5, 6, 20].

In medical practice, cases of anti-GBM disease do occur, but a significant proportion remain unrecognized during the patient's lifetime and end fatally. The presence of nonspecific symptoms at disease onset, variability of clinical manifestations after its progression, and the peculiarities of the course of anti-GBM disease often complicate timely diagnosis [1–3, 5, 7–8, 11–12, 19, 22]. It is generally accepted that early diagnosis of anti-GBM disease and timely initiation of immunosuppressive therapy combined with plasmapheresis can prevent complications and reduce mortality [1, 2, 19, 22, 23, 27].

The lack of information about this life-threatening pathology, the rarity of documented cases of anti-GBM disease, and the increasing incidence of the condition-especially in the post-COVID period-necessitate the preparation of this article.

The purpose of the study was to conduct a bibliometric analysis of the global catalog of

scientific literature indexed in leading scientometric databases, in order to identify the chronological dynamics of anti-GBM disease detection and to analyze data on its etiopathogenesis, clinical manifestations, modern principles of diagnosis, and treatment strategies.

Materials and methods. This literature review included studies reporting on the epidemiology, etiopathogenesis, clinical manifestations, diagnostic principles, and treatment of anti-GBM disease. The literature search was conducted in the PubMed, Google Scholar, Web of Science, and SCOPUS databases from January 1, 2021, to February 3, 2026. The last search query was performed on February 3, 2026. As a result, 33 publications in English were selected, confirming the adequacy of the information for conducting a systematic review and achieving the purpose of the analysis.

The search was conducted by developing a specialised query with the help of logical operators (AND, OR, NOT) and truncated terms as the variability of the key concepts. Publications pertaining to the affiliation of authors in the Russian Federation were not included in the analysis in order to guarantee the scientific relevance, adherence to the existing international context. The selection based on the set criteria was automated and formed the final sample.

Study Selection and Characteristics. To ensure comprehensive coverage of the research, we used the following search strategy:

Database 1 – PubMed. Search query (verbatim): (“hemorrhagic pulmonary-renal syndrome” OR “anti-GBM disease” OR “Goodpasture syndrome”) AND (“etiology anti-GBM disease” OR “risk factors anti-GBM disease”) AND (“prevalence anti-GBM disease” OR “epidemiology anti-GBM disease”) AND (“consequences anti-GBM disease” OR “mortality anti-GBM disease”).

Database 2 – Scopus. Search query (verbatim): TITLE-ABS-KEY (“anti-GBM disease” OR “Goodpasture syndrome” OR “hemorrhagic pulmonary-renal syndrome” OR “a disorder associated with antibodies to the glomerular basement membrane” OR “antibodies to glomerular basement membrane” OR “antibodies to alveolar basement membrane” OR “anti-glomerular basement membrane disease following COVID-19 infection” OR "alveolar hemorrhage” OR “primary renal-limited glomerulonephritis with pulmonary hemorrhage” OR “cloud-like infiltrates” OR “Anti-glomerular basement membrane vasculitis”)) AND NOT (AFFILCOUNTRY (“Russia” OR “Russian Federation”)).

Database 3 – Web of Science. Search query (verbatim): TS= (“anti-GBM disease” OR “Goodpasture syndrome”) AND TS= (“hemorrhagic pulmonary-renal syndrome” OR “primary renal-limited glomerulonephritis with pulmonary hemorrhage”, AND TS= (“risk factors” OR “determinants”) AND TS= (“prevalence” OR “epidemiology”).

Database 4 – Google Scholar. Search query (verbatim): “anti-GBM disease” OR “Goodpasture syndrome” AND “risk factors” AND “prevalence”. Search results were screened manually, and the first 132 most relevant records were evaluated for eligibility.

Under this condition, we applied the following filters: people, 2021–2026 years of publication, article or review. Additionally, the review included clinical practice guideline for the management of glomerular diseases KIDNEY DISEASE | IMPROVING GLOBAL OUTCOMES (KDIGO) 2021 [15].

The bibliometric analysis entailed evaluation of quantitative and structural indicators. The key parameters were: dynamics of the publishing activity over the years, which allowed to track the changes in the scientific interest; the typology of documents (original research, reviews, conference materials, etc.); the sectoralisation of publications by the Scopus classification.

This review article was based on open scientometric data without the use of any personalized or confidential information, and therefore did not require additional ethical approval.

The study protocol was performed in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. Data summarisation was done using comparative and trend analysis. Studies that did not contain original data, conference abstracts, editorials, preprints, and non-peer-reviewed articles were excluded from the search. In total, 417 articles in English were initially identified. After removing duplicates and excluding publications that did not meet the inclusion criteria, 33 full-text articles documenting cases of anti-GBM disease (Goodpasture syndrome) were selected for detailed review (Table 1).

Results of the study and their discussion. Chronological analysis showed that, the first clinical case of hemorrhagic pulmonary-renal syndrome with concomitant anemia in an 18-year-old patient was described by the American physician and pathologist Ernest William Goodpasture in 1919, scientific interest in this disease only emerged in 1958, after M. Stanton and J. Tange described 7 similar cases. They named the identified symptom complex Goodpasture syndrome (GS) in his honor [4, 5, 22, 24]. In the late 1970s and early 1980s, the formation and circulation of specific autoantibodies against the EA and EB epitopes of the non-collagenous (NC1) domain of the alpha-3 (α3) chain of type IV collagen [α3(IV)NC1] in the GBM were definitively demonstrated that interest in this problem has increased somewhat. Since then, the term “Goodpasture’s syndrome” has been used to describe patients with combined pulmonary and renal involvement [16, 19, 22, 24, 30]. In the article Ponticelli C. et al. (2023) it was reported that some nephrologists have referred to hemorrhagic pulmonary-renal syndrome associated with circulating antibodies to the GBM in such patients as “Goodpasture’s disease” [19]. However, until the beginning of 2013, the number of publications remained minimal, with no more than a few articles appearing per year.

Table 1

Simplified PRISMA Flow

Stage	Description	Number of Records/Studies
1. Identified	Total number of records identified through database searching and other sources	417
2. Duplicates Removed	Number of records removed before screening (e.g., duplicates)	78
3.Screened (Title/Abstract)	Number of records screened after duplicates were removed	339
4. Assessed for Eligibility (Full-text)	Number of full-text articles assessed for eligibility against the inclusion/exclusion criteria	184
5. Included in Review	Total number of primary studies finally included in the systematic review	33

In 2012 at the International Chapel Hill Consensus Conference on the Nomenclature of Systemic Vasculitides, the term "Goodpasture's disease" was not considered, and the combined lung and kidney involvement, which was previously called Goodpasture's syndrome, was designated as anti-GBM disease [23, 27].

Since 2018, publication activity has gradually increased. Its highest figures were obtained starting in 2020 (196 articles (57.8 %), which indicates increased scientific attention in the post-COVID period.

Structural analysis of scientific publications in the studied databases showed that, among all the full-text sources selected, 54.3 % were original articles. This distribution reflects the predominance of empirical research in the study of anti-GBM disease. Moreover, the fact that most studies were published in specialized journals indicates a high level of clinical relevance, validity, and reliability of the information.

According to most authors anti-GBM disease is still considered rare [4, 6, 18, 32]; however, incidence and prevalence data vary. Most authors report an incidence of 0.5–1.0 cases per million adults per year [4, 6]. Garg P et al. (2021) report on while others indicate 0.5–1.8 cases per million [10], and Reggiani F et al. (2023) – report up to 10 cases per million population annually [22]. At the same time, there is a separate report of 2 cases of this disease per 350 thousand population per year [1]. According to foreign scientific literature, a significant increase in the incidence of anti-GBM disease in the world has been documented during the coronavirus disease (COVID-19) pandemic. Under these circumstances, cases of its development have been documented both following infection with coronavirus (SARS-CoV-2) [18, 20, 29, 32] and after vaccination against COVID-19 [2].

Anti-GBM disease can occur at any age, but a bimodal age distribution with two peaks of disease manifestation is most commonly observed. The first peak typically occurs between 20 and 40 years of age, while the second peak is observed between 60 and 70 years [2, 22]. According to Prema KSJ et al., the mean age of 42 patients with COVID-19-induced anti-GBM disease was 46.2 years (range 16–82 years), including 15 men, 26 women, and 1 transgender patient [22]. The disease is very rare in children, adolescents, pregnant women, and the elderly. Only isolated reports have described anti-GBM disease in an 11-month-old infant, a 4-year-old girl, 17- and 18-year-old adolescents, an 81-year-old man [2, 16, 19, 22, 24], and pregnant women [4].

According to several authors, gender differences have been reported in the development of anti-GBM disease [2, 16, 19, 24, 30]. However, discrepancies exist among these studies. Most authors indicate that men are predominantly affected [2, 7, 22, 26, 27], whereas others report that during the second peak of the disease, men and women are affected with equal frequency [1, 16, 19, 24, 30]. In contrast, F. Reggiani et al. (2023) observed that in the sixth decade of life, anti-GBM disease occurs mainly in women [22].

The results of the study on the etiopathogenesis of anti-GBM disease showed that the exact cause of anti-GBM disease remains unknown, but its development is thought to require the presence of an exogenous or endogenous trigger that initiates the activation of antigen-presenting cells (such as dendritic cells and macrophages), thereby launching an immunopathological response. Genetic predisposition, particularly the presence of specific alleles of the major histocompatibility complex (human leukocyte antigen, HLA) of the DR class-such as HLA-DRW1, HLA-DRW2, HLA-DR15, and HLA-DR4-is considered the main risk factor, as it facilitates the generation of autoreactive T and B lymphocytes. These HLA alleles have been identified in nearly 80 % of affected patients [5]. The presence of specific HLA antigens determines not only the predisposition to autoimmune diseases but also influences the development of particular variants of their clinical course and the corresponding response to treatment [22]. Genetic predisposition is further supported by reported familial cases of anti-GBM disease [9].

According to several authors, various exogenous factors-particularly infectious agents such as viruses, bacteria, and fungal infections caused by Aspergillus species [2, 6, 22, 31] – may act as predictors of anti-GBM disease. In this context, they play a critical role as potential triggers initiating the pathogenetic cascade of immunological reactions. Most authors identify influenza A viruses (H1N1 or H3N2 strains), hepatitis A virus, and SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) as the principal triggering factors [2, 10, 18, 20, 21, 29, 31]. In most reported cases, the disease initially

manifested following influenza A infection. However, the coronavirus disease 2019 (COVID-19) pandemic introduced new considerations, as SARS-CoV-2 was recognized as a causative agent not only of pneumonia but also of autoimmune disorders [2, 10, 14, 30, 31]. Furthermore, according to the literature, SARS-CoV-2 infection may serve as a triggering factor for both the onset of anti-GBM disease and its relapses [2, 20, 31]. Research findings indicate that various immune reactions and underlying syndromes may persist for extended periods in the post-COVID phase. According to Makienko N. and colleagues (2022), this persistence is associated not only with the presence of patient comorbidities but also with the activation of endogenous viruses and imbalances in the intestinal microbiota [18]. It should also be noted that recent reports of anti-GBM disease following the second dose of the COVID-19 vaccine [2] have raised considerable concern. According to the medical literature, the temporal relationship between vaccination and the development of glomerular kidney diseases, including both de novo and relapsed anti-GBM disease, has been investigated. Studies have shown that these diseases typically manifest clinically several days to weeks after acute SARS-CoV-2 infection or COVID-19 vaccination [2, 20]. Although we do not know which vaccine was administered or whether this patient had a genetic predisposition, the issue remains of considerable concern.

Recent studies in Europe have suggested that the rising incidence of anti-GBM disease may be potentially related to environmental changes or factors [2]. One perspective highlights the possible etiological role of hypothermia, endotoxemia, and harmful habits such as prolonged smoking and drug abuse (including smoking cannabinoids or inhaling substances such as cocaine and amphetamines), which exert a direct toxic effect on the vascular wall. The effects of occupational hazards, particularly volatile hydrocarbon derivatives (such as toluene and methylbenzene), metal dust, organic solvents, varnishes, paints, gasoline, and increased oxygen content in the air or other inhaled gas mixtures [3, 6, 21], have also been reported. Inhaled toxins are known to promote localized inflammation by exposing previously hidden epitopes [16]. This is further supported by retrospective studies showing that, at the time of diagnosis of anti-GBM disease with alveolar hemorrhage, 68 % of patients were male, 89 % of whom were active smokers, and 36 % had been exposed to other inhaled agents [26].

Reports in the medical literature have also described the toxic effects of certain medications. According to most authors, anti-GBM disease may be induced by the use of D-penicillamine, carbimazole, or the monoclonal antibody alemtuzumab [1, 3, 16, 17, 21, 31].

The autoimmune theory of the GS pathogenesis began to be considered in the 1950s, when C.A. Krakower and S.A. Greenspon first identified the antigenic structures of the alveolar basement membrane (ABM) and renal glomeruli [6, 26, 13, 24]. The formation of circulating and fixed autoantibodies against the noncollagenous α3-chain domains of type IV collagen in GBM, which cross-react with lung basement membranes, and their deposition on the basement membranes of the alveoli and renal glomeruli in the presence of the C3 complement component, represents a major pathogenic mechanism of anti-GBM disease. Although basement membranes are present in many other structures of the body, the clinical manifestations of anti-GBM disease predominantly involve the lungs and kidneys [19, 22]. Anti-GBM disease is believed to arise from a disruption of the quaternary structure of the α345NC1 hexamer, leading to pathogenic conformational changes in the α3NC1 and α5NC1 subunits. These alterations modify the antigenic properties of alveolar and glomerular membrane proteins, break immunological tolerance, and ultimately trigger an autoimmune response. The predominant involvement of alveolar and glomerular membranes in the immunopathological process can be explained, on the one hand, by the high abundance of type IV collagen α3 chains in these structures, and on the other hand, by their maximal structural accessibility for the deposition of circulating autoantibodies [6, 16, 19]. Kuang H. and colleagues (2023), through epitope mapping of α3(IV)NC1, identified several nephritogenic epitopes and cryptic residues that, upon binding to autoantibodies, recognize hidden epitopes in α345(IV) collagen and thereby trigger the development of anti-GBM disease, contributing to the heterogeneity of its clinical presentation [16, 33]. Notably, the pathogenic effect of cryptic epitopes arises from the disruption of sulfilimine crosslinks within the NC1 domain, which are formed through peroxidase-dependent generation of hydrobromic acid. It should be noted that peroxidase, a heme peroxidase, shares significant structural overlap with myeloperoxidase (MPO). Consequently, antineutrophil cytoplasmic antibodies (ANCA) directed against MPO may be present both prior to and at the time of diagnosis of anti-GBM disease [12, 25]. Furthermore, Kuang H. et al. (2023) reported that both epitope spreading and molecular mimicry may contribute to the pathogenesis of anti-GBM disease. Epitope spreading refers to the development of autoimmunity against novel autoepitopes, thereby driving disease progression, whereas molecular mimicry involves the substitution of critical residues with microbial peptides, potentially initiating autoimmunity. Understanding these autoimmune reactions may open new avenues for the development of potential therapeutic strategies to treat this disease. However, the specificity and molecular architecture of tissue-bound autoantibody epitopes remain unknown [16].

As demonstrated in several scientific studies, circulating immune complex antigen-GBM autoantibody usually belong to the polyclonal IgG isotype, with a predominance of the IgG1 and IgG3 subclasses [3, 22]. More rarely, they may be of the

IgA isotype, associated with either kappa or lambda light chains [2]. Rare clinical cases of anti-GBM disease have also been reported in which the autoantibodies belonged to the IgG4 or IgA immunoglobulin subclasses, were monoclonal immunoglobulins [3], or were directed against alternative α-chains of type IV collagen. In addition, cases of double seropositivity have been described, where, during the course of anti-GBM disease, antibodies to basement membrane components coexist with ANCAs directed against MPO or proteinase-3 [12, 26]. These markers are characteristic of ANCA-associated systemic vasculitides, including granulomatosis with polyangiitis (Wegener's syndrome), microscopic polyangiitis, and eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome) [19]. It is noteworthy that the frequency of ANCA detection directed against MPO exceeds the detection of ANCA against proteinase-3 (anti-PR3). F. Reggiani reported that ANCA positivity often precedes the appearance of antibodies against BMC, which may indicate their possible role in the detection of critical epitopes [22].

It has been shown that antibodies binding to α3(IV)NC1 can also recognize α5(IV) to a greater extent, and α4(IV) to a lesser extent. Moreover, in approximately one-third of patients with anti-GBM disease-particularly those presenting with pulmonary hemorrhage-autoantibodies directed against peroxidase and laminin-521 have been detected in addition to anti-GBM antibodies. This contributes to the emergence of critical epitopes and further damage to basement membranes, since laminin is the most abundant component of all basement membranes and represents the principal isoform of glomerular and alveolar basement membranes. However, the role of antibodies against peroxidase and laminin-521 has not yet been fully studied [6, 22, 26].

The role of C3 component of complement activation in renal injury in anti-GBM disease has been demonstrated and carries clinical significance [33].

In addition to the mechanism described above, another major pathway of immunological glomerular injury involves the deposition of immune complexes within the glomeruli, as observed in lupus nephritis or membranous nephropathy. The coexistence of these two mechanisms of glomerular injury is rare and exhibits distinctive features in terms of clinical manifestations, renal pathology, and immunological findings [17, 19].

The exact mechanism by which SARS-CoV-2 induces anti-GBM disease remains unclear. However, it has been suggested that the leading pathogenic pathway involves a direct cytotoxic effect on the endothelium of small vessels in the lungs and kidneys. Proinflammatory cytokines such as IL-2, IL-7, IL-10, G-CSF, IP-10, MCP-1, MIP-1α, and TNF-α contribute to the development of a "cytokine storm" and systemic inflammation [2, 14, 21]. Subsequent activation of the C3 complement component leads to endothelial injury and the unmasking of previously sequestered epitopes on the basement membranes of the alveoli and renal glomeruli, thereby stimulating plasma cells and triggering an autoimmune response [20, 33]. In addition, SARS-CoV-2 can injure podocytes and renal tubular epithelium through activation of the angiotensin-converting enzyme-dependent pathway. This mechanism may result in renal tubular mitochondrial dysfunction, acute tubular necrosis, acute kidney injury (AKI), the formation of protein reabsorption vacuoles within the tubules, and collapsing glomerulopathy. In addition, ACE-2 expression may contribute to the pathogenicity of SARS-CoV-2. Risk factors for the development of AKI in this case may include hypotension, shock, renal hypoperfusion, hypoxia, cardiorenal syndrome, rhabdomyolysis, endotheliosis, macrophage activation syndrome, hypercoagulability [9, 10, 22, 30, 31] and drugs used to treat COVID-19 patients, as they may enhance the nephrotoxic effect of the virus [2, 5, 11].

The clinical presentation of anti-GBM disease can be highly variable, which often contributes to delays in diagnosis. The onset of classic manifestations rarely occurs abruptly; rather, it is typically preceded by a prolonged period of nonspecific symptoms. These most commonly include dyspeptic complaints (abdominal pain, nausea, heartburn, dryness and bitterness in the mouth), asthenic syndrome, arthralgia, myalgia, fever, ankle joint swelling, and unexplained weight loss [16, 22]. The classic clinical features of anti-GBM disease generally emerge under the influence of trigger factors, most often against a background of genetic predisposition [6, 16, 22].

The typical manifestations of anti-GBM disease include hemorrhagic pulmonary-renal syndrome, most often presenting as rapidly progressive glomerulonephritis (RPGN), AKI, and/or recurrent pneumonitis with or without pulmonary hemorrhage [6, 16, 22, 26, 31]. The principal symptoms are recurrent hemoptysis or pulmonary hemorrhage, dyspnea or a sensation of incomplete inspiration, cough, chest pain, hematuria with urine discoloration to red or "meat slops," occasional dull pain in the lumbar region, as well as clinical features associated with arterial hypertension and concomitant anemia. Three clinical variants of the course of anti-GBM disease are most commonly observed: 1) an initial presentation with isolated pulmonary manifestations (in 40–60 % of cases accompanied by alveolar hemorrhage), followed later by renal involvement, or vice versa; 2) a slowly progressive simultaneous injury to both lungs and kidneys; and 3) a malignant course characterized by rapidly progressive glomerulonephritis with renal failure and hemorrhagic pneumonitis [6]. At the same time, patients may present with isolated renal involvement, manifesting as acute glomerulonephritis (1–2 %), rapidly progressive glomerulonephritis (10–15 %), or AKI [22], as well as atypical variants of the disease [8]. The latter, typically characterized by isolated renal involvement, tends to follow a milder course

with slower progression compared to the classic form [5, 8, 13, 22]. This observation was confirmed by the study of Bharati J. et al. According to their findings, 20 patients with an atypical course of anti-GBM disease exhibited isolated renal involvement, manifested as proteinuria, hematuria, and slowly progressive renal failure, without any signs of pulmonary damage or hemorrhage [3]. At the same time, other authors have reported isolated cases of anti-GBM disease occurring in association with antiphospholipid syndrome (APS), thrombotic microangiopathy (TMA), microangiopathic hemolytic anemia (MAHA), thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation (DIC), ANCA-associated systemic vasculitis (SV), and systemic lupus erythematosus (SLE). In contrast to the organ specificity of isolated anti-GBM disease, the clinical presentation in these cases is characterized by rapidly progressive hemorrhagic pulmonary-renal syndrome, multiorgan involvement, a severe course, and a poorer prognosis [12, 17, 22, 25]. The coexistence of anti-GBM disease with ANCA-associated vasculitis (such as microscopic polyangiitis (MPA) or granulomatosis with polyangiitis (GPA)) or with other autoimmune disorders indicates the development of an overlap syndrome [28]. In cases where anti-GBM disease is associated with membranous nephropathy (MN) or IgA nephritis, patients may present with hemoptysis, AKI accompanied by nephrotic-range proteinuria, glomerular hematuria, arterial hypertension, and anemia [1, 3, 7, 9, 11, 24, 27, 32]. At the same time, cases have been reported of anti-GBM disease occurring in combination with post-transplantation Alport nephritis, which develops in some patients with Alport syndrome after kidney transplantation as a result of alloantibody formation directed against GBM [11, 25, 32].

The clinical course of anti-GBM disease depends on age, lifestyle factors, exposure to infectious agents, as well as the morphological variant of renal tissue changes and the type of serological positivity. In patients younger than 30 years, pulmonary involvement is most frequently observed [2, 22], whereas in those older than 50 years, isolated renal involvement predominates [9, 20, 22]. This observation is further supported by reports in the literature indicating that, in cases of COVID-19-induced anti-GBM disease and after COVID-19 vaccination, almost all documented patients were elderly women presenting with hematuria and AKI. Only a single case involved a 26-year-old patient who simultaneously developed symptomatic pulmonary hemorrhage and impaired renal function [2]. Pulmonary manifestations, including restrictive-type respiratory failure and hypoxemia, occur predominantly in smokers [2, 6]. SARS-CoV-2 infection provokes a malignant variant of anti-GBM disease, characterized by pulmonary pathology [2, 18, 30], which is rapidly followed by AKI or RPGN [10, 21, 22, 32].

Anti-GBM disease is considered an unusual autoimmune disorder, as it rarely follows a relapsing-remitting course. Relapses are most often observed in patients with concomitant invasive pulmonary aspergillosis, in cases of COVID-19-induced anti-GBM disease, or in overlap syndromes with other autoimmune conditions [32, 33]. Notably, infection with SARS-CoV-2 can provoke a second exacerbation in patients who had already achieved remission, with symptoms appearing either during the acute phase of COVID-19 or several weeks after recovery [2, 10]. At the same time, isolated spontaneous remissions of the disease have also been documented in the medical literature [23].

Anti-GBM disease can be suspected primarily on clinical grounds when patients develop pulmonary symptoms such as dry cough, dyspnea, pulmonary hemorrhage, or hemoptysis, and/or isolated renal involvement. Rapid progression of symptoms accompanied by urinary abnormalities-including increasing proteinuria, microhematuria-together with anemia, leukocytosis, eosinophilia, and a marked acceleration of the erythrocyte sedimentation rate (ESR) may strongly suggest anti-GBM disease [23]. During physical examination, attention should be paid to pallor of the skin, cyanosis of the mucous membranes, and elevated blood pressure (BP). Pulmonary findings may include dullness to percussion over large foci of inflammation, scattered dry or moist wheezes, and occasionally crepitations on auscultation. Cardiovascular signs may involve leftward expansion of the cardiac dullness border, muffled heart sounds, and a soft systolic murmur. In cases of severe renal failure, a pericardial friction rub may also be detected. The kidneys are usually not palpable, although percussion tenderness may be present bilaterally. The volume of diuresis depends on the nosological form of renal involvement and the stage of disease progression. In the complete blood count: iron-deficiency hypochromic anemia with hypochromia, anisocytosis, and poikilocytosis of erythrocytes, leukocytosis with a left shift of the leukocyte formula and an accelerated ESR are also typical. Urinalysis reveals urinary abnormalities such as hematuria and proteinuria, and in some cases features of nephrotic syndrome. As chronic kidney disease (CKD) progresses, the relative density of urine decreases and isohyposthenuria develops, which can be detected using the Zimnytsky test. In the biochemical blood analysis: elevated levels of urea, creatinine, haptoglobin, seromucoid, transaminases, and fibrinogen are observed, along with reduced serum iron. Hypercholesterolemia, hyperkalemia, and hypoproteinemia are frequently present. In the proteinogram-increased concentrations of α2- and λ-globulins. Sputum analysis reveals erythrocytes, hemosiderin, and siderophages. Serological studies for determining antibody titers against the glomerular and alveolar basement membranes are performed using indirect immunofluorescence or radioimmunoassay [22]. In some cases, antibodies to the α3NC1 domain of the GBM can only be detected using highly sensitive

biosensor metod. Occasionally, modified techniques reveal autoantibodies of other immunoglobulin subclasses, such as IgG4, or antibodies of monoclonal origin. However, in most cases of atypical anti-GBM disease, no identifiable antibodies are detected despite thorough patient evaluation [3, 5, 14]. The presence of laminin-521 antibodies may indicate a subgroup of patients with a more severe disease phenotype and an unfavorable prognosis [26].

An immunogram makes a significant contribution to diagnostic verification. Findings may include the presence of circulating immune complexes, a reduction in suppressor T lymphocytes (CD3+CD8+ cells), and an increase in activated T lymphocytes (CD25+) and T helper cells (CD4+) during the acute phase, which react with the α3(IV)NC1 domain. Notably, a decrease in T helper cells-key regulators of the immune response-during recovery may account for the rare occurrence of relapses and may explain the gradual decline in pathogenic autoantibodies over time, due to their reduced participation in immune reactions [3, 13, 16, 19, 27].

To investigate the possible coexistence of anti-GBM disease with APS, TMA, MAHA, TTP, DIC, ANCA-associated vasculitis, SLE, or IgA nephritis, a comprehensive evaluation is required. This includes HLA genotyping, assessment of functional and genetic aspects of the complement system, determination of ADAMTS13 activity (in TTP), coagulation studies, and screening for specific autoantibodies characteristic of the aforementioned autoimmune disorders. Reports indicate that double seropositivity occurs in approximately 30 % of such patients. One possible link between these conditions may be a shared autoantibody profile [11, 12, 17, 25].

The diagnosis becomes evident when chest radiography reveals diffuse, bilateral, symmetrical basal focal-infiltrative polymorphic changes of a confluent nature with indistinct margins, as well as “dust-like” or cloud-like infiltrates predominantly localized in the middle and lower lung regions. These findings, in the absence of positive clinical or laboratory dynamics following nonspecific antibiotic therapy, strongly support the diagnosis.

During chest computed tomography (CT), bilateral infiltrative-focal opacities with indistinct margins, a “ground-glass” appearance, and radiological signs of alveolar hemorrhage can be detected. It is noteworthy that these infiltrates are transient in nature: they may appear during pulmonary hemorrhage as a result of alveolar bleeding and subsequently resolve spontaneously [2, 10, 19, 27, 32].

Spirography typically demonstrates a restrictive pattern of external respiratory dysfunction, to which, as the disease progresses, an obstructive component is added [2, 10, 27, 31]. Electrocardiography usually reveals signs of pronounced myocardial dystrophy of anemic and hypoxic origin, as well as evidence of left ventricular hypertrophy resulting from prolonged arterial hypertension [3, 5, 9].

The gold standard for verifying alveolar hemorrhage is fiberoptic bronchoscopy with bronchoalveolar lavage. The detection of erythrocytes and siderophages in the lavage fluid confirms the presence of pulmonary hemorrhage [2, 30, 32]. The diagnostic standard for anti-GBM disease is renal and/or pulmonary biopsy [6, 19]. The key pathomorphological substrates are hemorrhagic necrotizing alveolitis and necrotizing glomerulonephritis [5, 6, 26].

Indications for renal biopsy in anti-GBM disease include the presence of nephrotic syndrome, proteinuria exceeding 1 g/L, persistent or recurrent glomerular hematuria, renoparenchymal arterial hypertension of unclear origin, acute kidney injury of unknown etiology, and acute kidney injury accompanied by systemic signs or symptoms of hemorrhagic pneumonitis [15, 19].

For this purpose, immunofluorescence examination of renal or lung tissue biopsies is performed. A bright linear staining along the basement membranes of glomeruli or alveoli indicates deposition of IgG or IgM together with the C3 complement component. Light microscopy reveals morphological features such as proliferative-membranous, mesangial-proliferative, or necrotizing glomerulonephritis, glomerular sclerosis, renal parenchymal fibrosis, and either focal necrotizing or diffuse crescentic glomerulonephritis. These findings correspond to anti-GBM nephritis (type I) or immune complex glomerulonephritis (type II) [5, 15].

In cases of overlap syndrome with ANCA-associated vasculitis, features of anti-GBM/ANCA-associated glomerulonephritis (type IV) are observed, with SLE-against the background of one of the above changes, signs of lupus nephritis (classes I–IV) are detected, in cases combining anti-GBM disease with membranous nephropathy (MN), characteristic signs of MN are present, and similar associations may occur with other glomerular pathologies [15].

Morphological examination of lung tissue reveals capillaritis of the interalveolar septa, pulmonary infiltrates, hemosiderosis, pneumosclerosis, or alveolar hemorrhage [2, 6, 26].

The pathohistological picture of renal tissue in atypical anti-GBM disease is more heterogeneous than in the classical form [5]. According to the literature, light microscopy most often reveals endocapillary proliferative, mesangial-proliferative, membranous-proliferative glomerulonephritis, or focal-segmental glomerulosclerosis with mesangial hypercellularity, occasionally accompanied by microangiopathic changes and focal lesions [5]. However, immunofluorescence examination of renal tissue consistently demonstrates bright linear staining of the basement membrane characteristic of anti-GBM disease in nearly all patients, though involving different immunoglobulin classes (IgG, IgM, and IgA) [3, 19].

Detection of histopathological changes in renal or pulmonary tissue is essential for differential

diagnosis and for the initiation of specific pathogenetic therapy [15, 25].

A careful analysis of clinical manifestations, medical history, and laboratory as well as instrumental investigations is crucial for establishing a differential diagnosis. Anti-GBM disease must first be distinguished from granulomatosis with polyangiitis (GPA, formerly Wegener's granulomatosis) and microscopic polyangiitis, both of which may present with a similar clinical picture. The presence of organ-specific antibodies together with characteristic pathomorphological changes in renal and pulmonary tissue enables differentiation of these diseases. In general, anti-GBM disease lacks cutaneous and articular syndromes, as well as vascular lesions in other organs, which are typical of ANCA-associated systemic vasculitides [17]. It should be noted that anti-GBM IgA antibodies are frequently detected in patients with monoclonal gammopathy and bronchial carcinoma [33], making differentiation from these conditions essential. Moreover, the possibility of double seropositivity must be considered: in anti-GBM disease, antibodies to the basement membrane may coexist with ANCA directed against MPO or proteinase-3, particularly in children [12, 27]. In addition, ANCA positivity may precede the appearance of anti-BM antibodies [19]. In cases of recurrent anti-GBM disease, concomitant disorders should be carefully excluded [22, 27, 29].

Early diagnosis and timely initiation of immunosuppressive therapy combined with plasmapheresis are crucial to prevent unfavorable outcomes [20]. The treatment of anti-GBM disease is challenging and includes induction, maintenance, and symptomatic therapy [15, 19, 22]. For induction therapy of rapidly progressive glomerulonephritis with circulating anti-GBM antibodies, glucocorticosteroids, cytostatic agents, and plasmapheresis are prescribed. In cases complicated by alveolar hemorrhage, infusions of fresh frozen plasma are additionally administered [19, 22].

Glucocorticosteroid therapy is administered as pulse therapy with agents from the glucocorticosteroid group. Typically, methylprednisolone is prescribed at a dosage of 7–15 mg/kg/day (up to 1 g/day) for 3 consecutive days, followed by oral prednisolone at 1 mg/kg/day, not exceeding 60 mg/day (according to some reports, up to 80 mg/day [22]) for 7 days. The dose is then reduced weekly by 15 mg until reaching 20–30 mg, after which a slower taper is applied (by 2.5 mg every 2 weeks) over 8–10 weeks. After 15–16 weeks of such treatment, the prednisolone dose is gradually reduced according to the standard regimen to 10 mg/day [15, 19].

Cytostatic therapy is administered simultaneously with glucocorticosteroid treatment, typically by oral cyclophosphamide at a dosage of 2–3 mg/kg/day (1.5–2 mg/kg/day in elderly patients) for 3 months. If oral administration is not feasible, cyclophosphamide may be given as an intravenous infusion at a dosage of 0.5 g/m² [15, 19, 22].

Therapeutic plasmapheresis is employed to eliminate pathological plasma components such as autoantibodies, inflammatory cytokines, immune complexes, and extracellular vesicles (EVs), all of which contribute to disease progression. It is performed daily for 14 days with plasma exchange at 60 ml/kg (up to a maximum of 4 L per procedure) until the antibody titer against the glomerular basement membrane normalizes, using a 5 % albumin solution [15, 19, 22, 23]. In the presence of hemorrhagic syndrome, plasma therapy is indicated. Maintenance of remission consists of prednisolone administration at 7.5–10 mg/day for 6 months [15, 19, 22].

The scientific literature has repeatedly reported positive outcomes with the use of monoclonal antibody therapy (anti-CD20), specifically Rituximab [19, 22]. According to Li L-L and colleagues, preferential use of B-cell-targeted therapy such as Rituximab is particularly indicated for the treatment of anti-GBM disease in young men with hypogammaglobulinemia. They emphasize that early administration of glucocorticoids and monoclonal antibodies in combination with plasmapheresis can restore renal function [17]. Some authors have reported positive effects of combination therapy with glucocorticosteroids, mycophenolate mofetil, and plasmapheresis [19]. At the same time, Uhlin F. et al. demonstrated encouraging results in a pilot study evaluating the efficacy of endopeptidase, which cleaves both circulating and basement-membrane-bound IgG, in patients with anti-GBM disease [30]. Similarly, Reggiani F. et al. reported favorable outcomes with imlifidase in the treatment of anti-GBM disease. However, the effectiveness of these approaches still requires confirmation in randomized controlled trials [22, 27, 30].

The treatment of anti-GBM disease in children is based on very limited evidence and typically involves acute plasmapheresis in combination with intensive immunosuppression, such as cyclophosphamide and corticosteroids. The evidence supporting the use of biologic agents in pediatric anti-GBM disease remains extremely limited [15].

In the absence of response to pathogenetic therapy, renal replacement therapy (RRT) is indicated, either in the form of dialysis or kidney transplantation.

Anti-GBM disease is associated with a poor prognosis. Historically, it was regarded as a fatal condition. Without timely diagnosis and prompt initiation of adequate immunosuppressive therapy, the clinical manifestations of anti-GBM disease rapidly progress to acute pulmonary lesions (APL), end-stage renal failure, and/or massive pulmonary hemorrhage, often resulting in a fatal outcome. In such cases, the mortality rate among patients reaches 75–90 % [19, 22]. The introduction of aggressive immunosuppressive therapy in combination with plasmapheresis and the use of chronic hemodialysis has increased the one-year survival rate to 86.9 % [19] and the five-year survival rate to 80 % [22]. Nevertheless, early treatment of patients with poor

prognostic factors (rising serum creatinine, reduced glomerular filtration rate) using the standard regimen of glucocorticoids, cyclophosphamide, and plasma therapy often fails to restore renal function, leaving patients dependent on lifelong hemo- or peritoneal dialysis [24, 27]. The prognosis also depends on the severity of the disease. In atypical variants of anti-GBM disease with relatively slow progression of pulmonary and renal involvement, the outlook is somewhat more favorable. In malignant forms, however, death may occur within several days or weeks from disease onset, whereas in other cases the average life expectancy ranges from 5–7 months to 1–3 years [2, 22]. In patients with end-stage renal failure requiring dialysis, or in those with a high proportion of crescents in the glomeruli detected on nephrobiopsy, the renal prognosis is poor [23, 25]. Floyd L. et al. emphasized that there are currently no reliable tools for predicting or treating this rare disease. However, the authors demonstrated that stratifying patients according to the percentage of normal glomeruli observed on nephrobiopsy and the need for renal replacement therapy (RRT) at the time of diagnosis improves outcome prediction and may guide therapeutic decision-making [9].

Limitations. When interpreting the results, it should be taken into account that epidemiological data reported in the publications varied across regions, which limited the possibility of quantitative synthesis and meta-analysis.

Conclusions

1. Anti-glomerular basement membrane disease is a rare but prognostically life-threatening organ-specific systemic vasculitis affecting the small vessels of the kidneys and lungs, with an incidence that remains imprecisely defined.

2. Anti-glomerular basement membrane disease should be considered in the differential diagnosis of all patients presenting with rapidly progressive glomerulonephritis and/or alveolar hemorrhagic syndrome, particularly in the age groups 20–30 and 60–70 years. It should also be taken into account following prior SARS-CoV-2 infection or after vaccination against COVID-19.

3. The clinical course of anti-glomerular basement membrane disease is characterized by considerable variability, determined by the sequence of renal and pulmonary involvement in the pathological process and by the extent of organ damage. This variability necessitates comprehensive screening of all patients with suspected pulmonary-renal syndrome.

4. A recurrent course of anti-glomerular basement membrane disease may indicate the presence of comorbidities or suggest a COVID-19-induced pathogenesis.

5. Verification of the typical course of anti-GBM disease is based on an elevated titer of antibodies to the glomerular and/or alveolar basement membrane, data from a nephrobiopsy and/or transbronchial lung biopsy.

6. Double seropositivity for antibodies to the glomerular basement membrane together with antineutrophil cytoplasmic antibodies, and/or with antibodies specific to other autoimmune diseases, indicates the presence of an overlap syndrome. This overlap determines distinctive features of the clinical course and influences the prognosis of the disease.

7. In atypical anti-glomerular basement membrane disease, serological markers may yield false-negative results, while the pathohistological features of renal tissue are more heterogeneous than in the classical form. This underscores the key role of nephrobiopsy in confirming the diagnosis. In cases of glomerulonephritis immunofluorescence examination of renal biopsies for IgG, IgM, IgA, and complement component C3 should be performed.

8. The gold standard for diagnosing alveolar hemorrhage is fiberoptic bronchoscopy with bronchoalveolar lavage, complemented by computed tomography of the chest.

9. Early diagnosis and timely initiation of intensive combined immunosuppressive therapy together with plasmapheresis are crucial for reducing immune inflammation, slowing the progression of renal failure and pulmonary pathology, and ultimately decreasing patient disability while improving survival rates.

10. Low diagnostic vigilance among physicians may lead to underestimation of the disease, delayed verification of the diagnosis, and postponement of pathogenetic therapy, all of which significantly worsen the prognosis in this patient cohort.

Prospects for future research include the development of diagnostic algorithms, refinement of differential diagnosis, and the creation of novel therapeutic approaches that could provide greater efficacy and a safer profile compared with current standard treatments for anti-GBM disease.

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