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CASE BASED REVIEW

From ulcerative colitis to amyloid storm: a case-based review

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Abstract

The most important complication of familial Mediterranean fever (FMF) are AA amyloidosis and amyloid nephropathy. Amyloid nephropathy typically follows a slow, indolent course. However, "amyloid storm" is a recently described acute condition, characterized by sudden onset of severe proteinuria and rapidly progressive renal failure with markedly acute-phase reactants, progressing over days to weeks into end-stage renal disease (ESRD). Amyloid storm has been rarely reported in the literature. Most cases have occurred in patients with known FMF-related AA amyloid nephropathy. However, it has also been reported as the initial presentation of AA amyloidosis. No standardized treatment guidelines exist, but management has included supportive care, colchicine, and interleukin (IL)-1 blockade. This report presents the first case of amyloid storm associated with ulcerative colitis in the literature. The patient exhibited impaired oral intake and fatigue, alongside acute kidney injury, spot urine proteinuria of approximately 100 g/day, and elevated C-reactive protein levels (CRP 118 mg/L). The diagnosis of AA amyloidosis was made after a kidney biopsy. In addition to starting haemodialysis, the patient was treated with anakinra and colchicine given the clinical presentation indicative of an amyloid storm. Subsequently, the acute-phase reactants decreased and the patient's general health improved, but haemodialysis was still required. After the patient discontinued anakinra treatment, he was re-hospitalized with complaints of fatigue and weakness but died on day 71 after the amyloid storm diagnosis. In our literature review, we identified a total of 36 patients with amyloid storm, 29 of whom were from two separate original studies. According to our review, the frequency of FMF-associated amyloid storm is 5.5%. The majority of patients were male, and while FMF was the most common cause of AA amyloidosis, ankylosing spondylitis, non-FMF autoinflammatory diseases, and ulcerative colitis were among the other causes. Our analysis revealed that infections were the most common triggering factor of an amyloid storm. ESRD rates were found to be up to 67%, and mortality rates ranged from 33 to 67%. This case report aims to highlight the relatively new and little-known concept of amyloid storm, which, despite its rarity, discusses its clinical features, laboratory findings, treatment options, and outcomes based on our case as well as a few cases in the literature.

Keywords Amyloidosis · Familial Mediterranean fever · Ulcerative colitis · Interleukin-1 receptor antagonists

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Introduction

The primary manifestation of AA amyloidosis is renal involvement, characterized by a latent period of tissue amyloid deposition. This is followed by a gradual increase in proteinuria, progressive renal dysfunction, and the eventual development of end-stage renal disease (ESRD) over the next 10 to 20 years [1–3]. The rate of progression may vary depending on the degree of inflammation, as well as genetic and environmental factors [4, 5]. Under certain circumstances, the typically indolent course of amyloid nephropathy may accelerate markedly. In recent years, a severe, acute, and dramatic entity called "amyloid storm" has been described. This syndrome presents with abrupt onset of heavy proteinuria (often > 10 g/day), rapidly progressing renal failure, and markedly elevated acute-phase reactants, often leading to ESRD within days to weeks [6]. Although most cases have involved patients with familial Mediterranean fever (FMF)-related AA amyloidosis (FMF-AA), amyloid storm has also been reported in association with ankylosing spondylitis, non-FMF autoinflammatory conditions, and even idiopathic cases without known inflammatory disease [7]. AA amyloidosis is a rare but serious complication of inflammatory bowel disease (IBD). Renal involvement is the most lethal clinical manifestation in AA amyloidosis associated with IBD, as it often leads to ESRD [8]. The incidence of AA amyloidosis in Crohn's disease ranges from 0.3 to 10.9%, while in ulcerative colitis it is between 0 and 0.7%, with significant geographic variation [9–12].

Here, we present the first case in the literature of amyloid storm associated with ulcerative colitis, in a patient who developed acute kidney injury following a pulmonary infection, with no previously known renal issues. The aim of this report is to draw attention to the rarely observed and still not well-understood concept of amyloid storm.

Case

A 66-year-old male with a 10-year history of hypertension and a 19-year history of ulcerative colitis in remission (on mesalazine) presented to primary care three weeks earlier with symptoms of pulmonary infection and was prescribed antibiotics. He subsequently developed worsening fatigue and poor oral intake and was admitted to our emergency department. Laboratory findings revealed blood urea nitrogen (BUN) of 156 mg/dL and serum creatinine of 19.93 mg/dL, prompting admission to nephrology and initiation of haemodialysis. The patient's fluid therapy was initiated and ceftriaxone 2 g/day was started. His chronic medications included mesalazine (2 × 500 mg daily) and candesartan (16 mg daily). On physical examination, he was conscious and appeared slightly drowsy, his blood pressure was 165/86 mm/Hg, his pulse rate was 87/minute, his abdominal examination was normal, and his respiratory examination revealed crepitant rales in bilateral lower zones and bilateral + 1 pretibial edema. He was oligo-anuric on admission to the hospital. On chest radiography, cardiothoracic ratio was normal, costophrenic angles were slightly blunted, and there was no obvious pneumonic infiltration. Three weeks earlier, serum creatinine was 0.98 mg/dL, and complete urine analysis (CUA) 22 months prior revealed no proteinuria. Laboratory tests on admission showed hemoglobin (Hb) 13.2 g/dL, leukocytes 13.5×10^3/µL, platelets 576×10^3/µL, sodium: 139 mEq/L, potassium 7.83 mEq/L, glucose: 91 mg/dl, serum albumin 2.91 g/dL, liver enzymes were normal, creatine kinase is 139 U/L (46–195), C-reactive protein (CRP) 118 mg/L, and erythrocyte sedimentation rate (ESR) 88 mm/h. In CUA, there was + 3 protein, erythrocytes 15/HPF, leukocytes 12/HPF, and urine sediment examination showed 5–6 monomorphic erythrocytes and leukocytes per high-power field. Spot urine protein/Cr ratio was 38.38 g/day. There were no pathological findings in the peripheral blood smear other than toxic granulation in neutrophils. Ultrasound revealed normal kidney size without postrenal obstruction. Complement C3 was 1.86 g/L (0.9–1.8), and complement C4 was 0.450 g/L (0.1–0.4). Hepatitis serology, anti-nuclear antibody (ANA), anti-glomerular basement membrane (GBM) antibody, anti-myeloperoxidase (MPO), and anti-proteinase 3 (PR3) were negative. No plasma cell increase was observed in bone marrow aspiration performed after a suspicious peak was observed in serum protein electrophoresis. Bone marrow biopsy showed AA amyloid deposition in the vessel walls. No microorganisms were detected in the blood and urine cultures. Kidney biopsy revealed severe AA-type amyloid deposition (Fig. 1). There was no history, symptom, or finding suggestive of FMF, other autoinflammatory diseases, chronic pulmonary diseases, or any inflammatory conditions other than ulcerative colitis. There was no family history of FMF or other autoinflammatory diseases. FMF gene mutation testing was negative.Given the acute kidney injury, massive proteinuria, and high acute-phase reactants, amyloid storm was considered. Although our patient had a history of ulcerative colitis, we chose to use an interleukin (IL)-1 inhibitor rather than anti-TNF therapy because the primary concern was pneumonia-induced amyloid storm, and the ulcerative colitis was in remission. Therefore, we used anakinra and colchicine. Within two weeks, there was a clinical improvement, and CRP decreased to 14.5 mg/L. The patient was discharged with a tunneled jugular catheter for haemodialysis three times weekly. The patient was admitted to the nephrology clinic again 25 days after being discharged, as

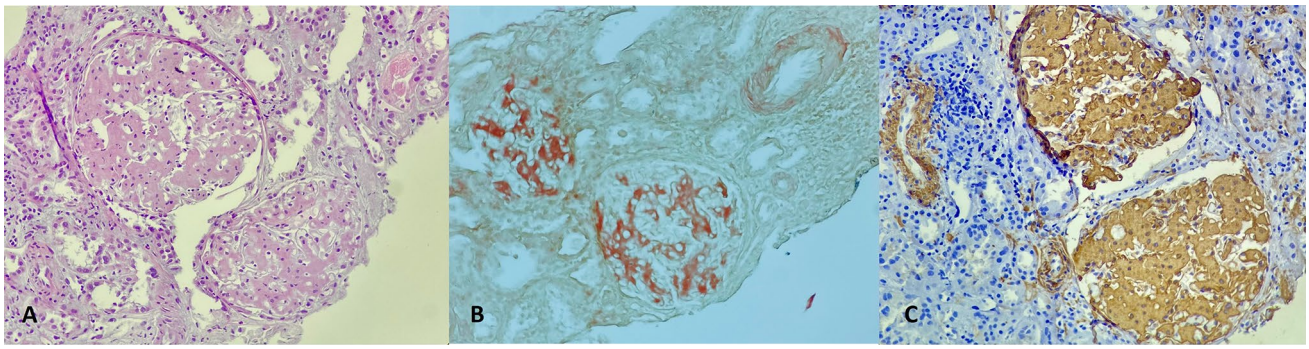


Fig. 1 Extensive pale eosinophilic amorphous material accumulation is noted in the glomerular mesangial areas, interstitium, and vascular walls (H&E, X200) (**A**). The areas consistent with amyloid deposition showed a brick-red color with Congo red staining, and these areas exhibited apple-green birefringence under polarized light microscopy (Histochemical staining, X200) (**B**). The amyloid-positive areas demonstrated brownish positive expression with Amyloid A stain (Immunohistochemical staining, X200) (**C**)

hypotension (80/40 mm/Hg) was detected during an outpatient clinic check-up, and weakness in the arms and legs, loss of appetite, and decreased oral intake were observed. It was discovered that he had interrupted anakinra for the preceding 15 days. After blood, urine, and catheter cultures were obtained, the patient with a tunneled catheter was empirically started on 500 mg ceftazidime once daily and 420 mg daptomycin every 48 h. The etiology of hypotension was investigated. The patient was evaluated by a cardiologist for cardiac amyloidosis, and echocardiography revealed no septal wall thickness or diastolic dysfunction. Due to refractory hypotension possible adrenal insufficiency or adrenal involvement of amyloid was excluded with adrenal function tests, and sepsis was excluded based on clinical and laboratory findings during the second hospitalization, Hb was 12.7 g/dl, leukocytes 9.5 10^3 /ul, platelets 184 10^3 /ul, CRP 42.8 mg/dl, ESR 102 mm/h, liver enzymes were normal, creatine kinase was 45 U/L (46 – 195), there was no lung or urinary tract infection, no growth was observed in urine, blood, or catheter cultures. In the neurological evaluation, muscle strength was 4/5 in the lower and upper extremities, no neurological deficit was detected. There was no response to empirical treatments of 3 × 20 mg methylprednisolone and 0.1 mg fludrocortisone initiated for deepening hypotension. It was thought that the cause of hypotension and muscle weakness might be due to the autonomic nerve system and muscle involvement by amyloidosis, but orthostatic tests and muscle biopsy could not be performed due to the rapid deterioration of the patient's general condition. Inotropic support was required and the patient was transferred to the intensive care unit. The patient died 71 days after diagnosis due to multi-organ failure secondary to amyloid storm. The clinical and laboratory data are presented in Table 1, and the timeline of clinical course is shown in Fig. 2.

Search strategy

Since there are few publications on amyloid storm, we aimed to analyse the findings of other rare cases of amyloid storm in the literature, alongside our own case. This case-based review was prepared in accordance with the CAse-BAsed REview sTandards (CABARET) recommendations of the EQUATOR Network [13]. We searched Pubmed/MEDLINE, Web of Science, Scopus and Directory of Open Access Journals (DOAJ) up to October 2025 using the terms ("amyloid storm") OR ("amyloid" AND "storm") OR ("amyloidosis" AND "storm"). Original articles written in English, case series, case reports, and case-based reviews were selected for the study. Non-English articles, review articles, congress abstracts, and articles not addressing amyloid storm were excluded. As a result of the search strategy, we identified a total of 6 articles, including 2 original studies with 20 and 9 patients with amyloid storm, 2 case series with 5 cases, 1 case-based review, and 1 case report with 1 patient. [6, 7, 14–17].

Discussion

Amyloid storm is a severe and rapidly progressing complication observed in FMF-related AA amyloidosis, marked by sudden worsening of kidney function and an intense inflammatory response. It was initially recognized in the literature in 2021, highlighting its potential to cause life-threatening complications [6]. Since it is a recently recognized entity, only a limited number of studies have been published on this condition [6, 7, 14–17].

Information regarding how often amyloid storm occurs in FMF-associated AA amyloidosis is limited. Previous studies have not clearly defined its incidence among patients with amyloidosis, making it difficult to estimate the true frequency of this severe complication [6]. A cohort involving

Table 1 Clinical and laboratory findings of our patient before admission and during the amyloid storm

Findings	Before Admission	At Admission (February 2025)
Creatinine (mg/dL)	0.98 (0.7–1.2) (January 2025)	19.93 (0.7–1.2)
Proteinuria (g/day)	Urine/24-h protein negative (April 2023)	Spot urine protein/Cr = 38.382
CRP (mg/L)	9.72 (0–5) (14.01.25)	118 (0–5)
Serum Albumin (g/dL)	–	2.91 (3.2–4.5)
Procalcitonin (ng/mL)	–	1.09 (0–0.046)
Trigger for Amyloid Storm	–	Pneumonia
Kidney Biopsy Findings	–	Severe amyloid accumulation in all glomeruli, mesangial area, and vascular walls (AA amyloidosis)
Pre-existing Rheumatic Disease	None	–
Pre-existing Inflammatory Disease	Ulcerative colitis (19 years)	–
MEFV Mutation	–	Negative
Final Diagnosis	–	AA amyloidosis secondary to ulcerative colitis + Amyloid storm
Treatment Initiated	–	Colchicine 0.5 mg/day, Anakinra 100 mg/day, Hemodialysis, Antibiotics
Current Status (Survival and Kidney)	–	ESRD, He died on day 71 after diagnosis
Bone Marrow Aspiration	–	No plasma cell increase, AA amyloid
ANA	–	Negative
Anti-MPO	–	Negative
Anti-PR3	–	Negative
Anti-GBM	–	Negative
Complement C3 (g/L)	–	1.86 (0.9–1.8)
Complement C4 (g/L)	–	0.450 (0.1–0.4)

CRP C-reactive protein, *MEFV* Mediterranean Fever, *ESRD* end-stage renal disease, *ANA* Anti-nuclear antibody, *MPO* Myeloperoxidase, *PR3* Proteinase 3, *GBM* glomerular basement membrane

174 patients, the majority with FMF-associated AA amyloidosis, reported an approximate prevalence of 5.5% for amyloid storm [7]. Reported cases have generally involved middle-aged adults. Comparisons between patients with FMF-AA who experienced amyloid storm and those who did not suggest that, although the initial onset and severity of proteinuria were similar, the kidney disease tended to progress more rapidly in those affected by the storm [6].

FMF represents one of the leading etiologies of AA amyloidosis. While in Western countries rheumatoid arthritis remains the predominant underlying disease—followed by ankylosing spondylitis, IBD, and FMF—the situation differs in Türkiye, where FMF constitutes the most frequent cause of AA amyloidosis rather than rheumatoid arthritis [18]. Amyloid storm was initially reported in patients with FMF-AA [6]. Subsequently, amyloid storm was also described in patients without FMF [7]. Torun et al. reported three cases of amyloid storm due to ankylosing spondylitis, idiopathic cases, and cases with nonspecific granulomatous disease [14]. Bronchiectasis continues to be a cause of AA amyloidosis, and a case of amyloid storm secondary to cystic bronchiectasis has recently been reported [15]. Since our patient had been diagnosed with ulcerative colitis for 19 years and had no other inflammatory conditions, ulcerative colitis was suspected to be the cause of AA amyloidosis.

While the majority of cases in the literature represent a worsening of the underlying amyloid nephropathy, amyloid storm can rarely be the first manifestation of amyloidosis [17] (Data on the characteristics of our case and other patients with amyloid storm in the literature are provided in Table 2). While amyloid storm is more likely to be considered in acute renal deterioration in a patient with amyloid nephropathy, it may be difficult to consider amyloid storm in the differential diagnosis of patients with rapidly progressing acute renal injury and elevated acute-phase reactants without known underlying amyloidosis. Renal biopsy is crucial in this situation. When AA amyloidosis is detected by biopsy, amyloid storm should definitely be included in the differential diagnosis.

Several triggering factors for amyloid storm have been identified. Infections were the most common triggers, while surgery and caesarean section, and non-compliance with treatment were also reported as additional precipitating factors [6, 7, 17]. In our patient, infection was identified as the precipitating factor for the amyloid storm as well.

A major concern in amyloid storm is the rapid progression to ESRD. Previous reports indicate that a substantial proportion of affected patients may experience swift deterioration of kidney function shortly after the onset of the storm, highlighting the severity of this complication. [6]. A study reported ESRD in 67% of patients with amyloid storm. The study found that the amyloid storm group had a higher incidence of involvement of three or more organs and more frequent bone marrow amyloid deposition [7]. Amyloid deposition was also detected in the bone marrow in our patient.

The main objective in treating AA amyloidosis is to address the underlying disease processes by reducing the liver's production of serum amyloid A (SAA), which is induced by cytokines. Prompt control of inflammation is especially critical, since delays in managing inflammatory activity may fail to prevent amyloid fibril deposition and can contribute to further disease progression [2]. Treatment

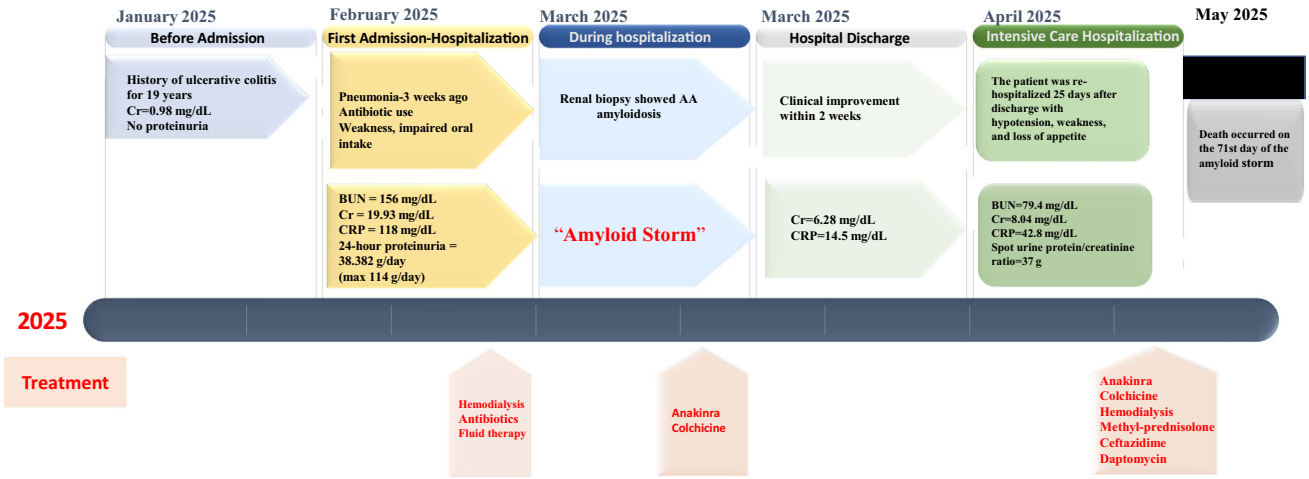


Fig. 2 Clinical course timeline of our patient with amyloid storm associated with ulcerative colitis

should be individualized, with infections managed using appropriate antimicrobials and rheumatologic or genetically mediated autoinflammatory conditions addressed through targeted anti-inflammatory therapies. Colchicine has become a cornerstone drug for the prevention of amyloidosis in FMF patients. Studies evaluating anti-TNF therapy in AA amyloidosis have primarily included patients with rheumatoid arthritis and spondylarthritis. IL-1 blockade with anakinra and canakinumab is used in the treatment of autoinflammatory pathologies such as FMF, and their effects on disease control have been demonstrated [19]. Supportive treatments (dialysis, fluid therapy, antibiotics, etc. in eligible patients) and anti-inflammatory agents have been used as treatment options in amyloid storm. There are no established treatment guidelines for the treatment of amyloid storm. The role of IL-1 blockers in the prevention and treatment of amyloid storm is still unknown [6, 7, 14]. Although there are only a few reports on the efficacy of IL-1 blockade in IBD, we found no publications regarding the use of IL-1 inhibitors in ulcerative colitis-associated AA amyloidosis [20]. In our patient, although TNF inhibitors could theoretically have been considered first-line therapy, we decided that anakinra represented a more rational and individualized choice, taking into account that the ulcerative colitis was in remission and the primary concern was pneumonia-induced amyloid storm. As soon as AA amyloidosis was diagnosed by kidney biopsy in our patient, a renal dose of colchicine and an IL-1 receptor antagonist were started immediately, considering the possibility of amyloid storm. Two weeks after the treatment, improvement in the patient's general condition and improvement in inflammatory markers were observed. However, it is plausible that interrupting anakinra treatment after discharge due to treatment non-compliance accelerated the course of the disease and caused deterioration in his general condition (Treatment agents used for amyloid storm are given in Table 2). Although we cannot prove the cause of our patient's hypotension and muscle weakness, we believe amyloidosis may be related to autonomic and muscle involvement. The presence of amyloid involvement in our patient's bone marrow biopsy suggests widespread disease. Although autonomic neuropathy and muscle involvement are more frequently reported in primary (AL) amyloidosis, they have also been described in patients with AA amyloidosis. It has been reported that creatine kinase levels in these patients are often not elevated, and their prognosis is poor [21–23]. The muscle weakness observed in our patient could also have resulted from prolonged immobilization or decreased functional capacity.

Amyloid storm is a clinical entity with a high mortality rate. A study reported a one-year mortality rate of 40% [6]. Another study reported a 33% mortality rate in amyloid storm, with some deaths occurring during the storm and others within five months of diagnosis [7]. Similarly, in the study by Torun et al., the mortality rate was found to be 33% [14]. (Definition and general information about amyloid storm are given in Table 3.) Although an early inflammatory and clinical response was observed in our patient, treatment was interrupted by the patient and died approximately 2.5 months after the diagnosis of amyloid storm.

Previous studies have primarily focused on the insidious progression and long-term outcomes of AA amyloidosis in chronic inflammatory and rheumatic conditions such as FMF and rheumatoid arthritis. These reports typically describe a gradual onset of proteinuria, a slow decline in renal function, and long-term management strategies [24, 25]. The main strength of our study lies in presenting a case of fulminant amyloid storm associated with ulcerative colitis — an acute and rapidly progressive manifestation that has not been previously reported. In addition, this review aims to raise awareness of amyloid storm—a relatively

Table 2 Demographic, clinical, and laboratory features of patients with amyloid storm described in the literature [6, 7, 14–17]

	Article type	Number of cases	Age/Gender	Diseases Causing AA Amyloidosis and Duration	Triggers for Amyloid Storm	Presence of Amyloid Nephropathy Before Storm	CRP Level During Storm (mg/L)	Creatinine Level During Storm (mg/dL)	Proteinuria Level During Storm (g/day)	Treatments for Amyloid Storm	Renal Outcome	Death and Timing
Our case	Case	1 patient	66/Male	Ulcerative colitis (19 years)	Pneumonia	None	118	19.93	38.382 (max 100 g/day)	Hemodialysis,colchicine, anakinra	ESRD	Exitus on Day 71
Kukuy et al. [6]	Original article	20 patients	Mean 39.95 ± 16.8 years/ 12 Male	All had FMF-related AA amyloidosis; FMF onset mean age 7.7 ± 8.56 years	Infection (17) Pregnancy/ misc-arriage (2) Colchicine toxicity (1)	All had known amyloid nephropathy with FMF	Mean 84.2 ± 67.53	Increased from baseline 1.4 ± 0.7 to 3.47 ± 1.16	Mean 15.89 ± 13.2	Supportive therapies (fluids, broad-spectrum antibiotics, plasma, albumin, steroids, antihypertensives, reduced colchicine dose, respiratory support, hemodialysis)	ESRD: 60%	Mortality: 1st month-67% 1st year-40%
Bektaş et al. [7]	Original article	9 patients	Median (IQR) 37 (10) / 4 Male	5 FMF-AA amyloidosis 2 AS-AA amyloidosis 1 idiopathic 1 non-FMF autoinflammatory disease	Infection (5) Non-compliance (3) Surgery (1)	Not reported	Median 48 (77)	Not reported	Median 11 (12)	Not reported	ESRD: 67%	Mortality: 33% (1 during storm, others median 5th month)
Torun et al. [14]	Case series	3 patients		–	–	–	–	–	–	–	–	–
	Case 1		33/Male	Idiopathic	Pneumonia	Present	78.2	7.03	12.86	Hemodialysis,colchicine, canakinumab	ESRD	Alive
	Case 2		47/Male	AS	Pneumonia	None	209	12.6	5.56	Hemodialysis, colchicine, adalimumab	ESRD	Alive
	Case 3		63/Female	Unspecified granulomatous disease	Lymphadenitis	None	206.3	7.99	10.7	Hemodialysis, colchicine	ESRD	Exitus
Abdelsattar et al. [16]	Case	1 patient	28/Male	FMF	Urinary tract infection	Likely present (baseline creatinine 1.4 mg/dL)	10	10.5	6.5	Hemodialysis, antibiotics	ESRD	Alive (3rd month)
Domingos et al. [15]	Case	1 patient	77/Male	Bronchiectasis Chronic kidney disease (G3aA2 stage)	Pyelonephritis	Likely present (urine dipstick protein + ++)	27.7	4.21	11	Supportive therapy (diuretics, ACEi for proteinuria reduction)	ESRD	Alive (1st year)
Yıldırım et al. [17]	Case 1		54/Female	Bronchiectasis	Pneumonia	None	197.2	2.17	2.3	Antibiotics, colchicine	ESRD	Alive
	Case 2		45/Male	Osteomyelitis	Infection	None	242	5.2	7.6	Hemodialysis, antibiotics, colchicine	ESRD	Alive

ACEi angiotensin-converting enzyme ınhibitor, *AS* ankylosing spondylitis, *ESRD* end-stage renal disease, *FMF* Familial Mediterranean Fever

Table 3 Definition and clinical features of amyloid storm [6, 7, 14–17]

Definition of Amyloid Storm	Amyloid storm is defined as at least a twofold increase in serum creatinine and proteinuria levels, along with at least a tenfold increase in CRP levels within less than two weeks in patients with known FMF-related amyloidosis *Although initially described in patients with FMF-related AA amyloidosis/amyloid nephropathy, it should be noted that amyloid storm can also occur in patients with non-FMF inflammatory diseases, with or without known amyloid nephropathy, and even as an initial presentation
Frequency of Amyloid Storm	5.5%
Causes of AA Amyloidosis	FMF, AS, bronchiectasis, granulomatous disease, ulcerative colitis
Triggers for Amyloid Storm	Infections, surgery, gastroenteritis
Frequency of ESRD	60–67%
Mortality Rate	1-year mortality: 40% 5-month mortality: 33%

FMF Familial Mediterranean Fever, *AS* ankylosing spondylitis, *ESRD* end-stage renal disease

new concept particularly for rheumatologists and nephrologists—by revisiting its definition and analysing studies in the literature on ESRD and mortality rates related to amyloid storm. Some rheumatologic diseases constitute medical emergencies requiring urgent intervention. Effective recognition and management of these conditions are essential, as they can be life-threatening and cause significant morbidity and mortality [26]. Amyloid storm will gradually be recognized as one of the rheumatologic emergencies. The principal limitations of this study are that it is a case-based review and that, given the novelty of the concept, there may be a likelihood of cases that have not yet been identified of amyloid storm.

In conclusion, although amyloid storm has been described more frequently in FMF patients with AA amyloidosis, it is a newly defined condition that should be considered in patients with acutely developed heavy proteinuria, rapidly deteriorating renal function, and significant elevations in inflammatory markers, regardless of whether they have a known chronic inflammatory disease. Although commonly considered rare, it may be more prevalent than thought, particularly in patients with amyloid nephropathy. Amyloid storm is a high-mortality complication that nephrologists and rheumatologists, in particular, need to be more aware of. Prospective studies with larger numbers of cases are needed to determine the true prevalence of amyloid storm, to more clearly reveal possible risk factors, to prevent its occurrence and to elucidate the effectiveness of IL-1 blockade in its treatment.

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Data Availability Satetment Data sharing is not applicable to this article as no new datasets were generated or analysed during the current study. All relevant clinical information is included in the article.

Declarations

Conflict of interest The authors declare that there is no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Informed consent Written informed consent for publication was obtained from the patient prior to death.

Previous related congress abstract publication This case was previously presented as an abstract at the 14th Current Kidney Diseases Congress, PS-07, 23–27 April 2025 in Türkiye (https://guncelleme2025.com).

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