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Divergent Clinical Phenotypes, Organ Involvement, and Survival in AL Amyloidosis and Light Chain Deposition Disease: Insights From a Large Single-Center Cohort

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Abstract

This retrospective single-center cohort included 480 immunoglobulin light-chain amyloidosis (AL) and 61 light chain deposition disease (LCDD) patients from Peking University First Hospital to compare clinical profiles and outcomes. AL frequently presented with multisystem (≥ 3 organs, 31.0%) and cardiac involvement (60.8%). LCDD was kidney-dominant (eGFR < 50 mL/min/1.73m², 85.2%) with higher clonal burden (dFLC > 180mg/L, 44/60 [73.3%] with available data), a trend toward higher CR rate (65.8% vs 48.8% AL, *P* = 0.058), and 100% ≥ PR to daratumumab in a small subgroup (n = 11). PI-based therapy was the main backbone (66.3% vs 66.0%). LCDD had superior OS (*P* < 0.001), while AL showed better renal survival in high-risk strata (albumin < 30g/L, eGFR < 50, 24h-UTP > 5g; all *P* < 0.05). These findings support disease-specific management: deep hematologic remission for AL and renal protection plus clone-directed therapy for LCDD.

Introduction

Light chain amyloidosis (AL) and light chain deposition disease (LCDD) are both classified as monoclonal light chain (LC)-associated plasma cell clonal disorders, yet they exhibit distinct clinical phenotypes and organ involvement patterns: AL is characterized by systemic involvement, with cardiac complications driving mortality risk, whereas LCDD is predominantly kidney-focused, where progressive renal function decline dictates outcomes. These two disorders also differ in light chain isotype preferences (λ-light chain predominance in AL and κ-light chain predominance in LCDD)[1–7]. This intraspectral heterogeneity gives rise to significant discrepancies in diagnosis and management, making early differentiation and strategy-driven care a key clinical challenge[1, 2, 4].

Over the past two decades, evidence regarding risk stratification and treatment for AL has accumulated rapidly: The Mayo 2004/2012 staging systems enhanced risk prediction and treatment decision-making for AL by integrating cardiac biomarkers and difference between involved and uninvolved free light chains (dFLC)[8–10]. Proteasome inhibitors (PIs) and anti-CD38 monoclonal antibodies have improved hematologic and organ responses, with the phase Ⅲ ANDROMEDA trial establishing the first-line role of daratumumab combined with cyclophosphamide, bortezomib, and dexamethasone (CyBorD)[11–13]; autologous stem cell transplantation (ASCT) confers long-term benefits in patients with minimal organ burden[13]; For LCDD, cohort studies consistently demonstrate that achievement of deep hematologic response directly correlates with renal benefit, and first-line PI-based regimens improve renal survival[4–6]. However, systematic cross-disease comparisons using a consistent methodology remain scarce, particularly a lack of generalizable comparative evidence in the Chinese real-world setting[1, 4]. Thus, in a large single-center real-world cohort of Chinese patients (480 with AL; 61 with LCDD), this study systematically compared the two disorders in terms of clinical phenotypes, organ involvement, treatment approaches, and treatment responses using a standardized methodological framework. It also evaluated actionable prognostic and follow-up biomarkers (e.g., N-terminal pro-B-type natriuretic peptide [NT-proBNP], cardiac troponin I [cTnI], estimated glomerular filtration rate [eGFR], serum creatinine, dFLC),

aiming to provide evidence-based support for disease-specific management and precision stratification of these two disorders in the Chinese population.

Materials and Methods

Patients

Patients with light-chain amyloidosis (AL) or light-chain deposition disease (LCDD) confirmed by histopathological examination at Peking University First Hospital (PKUFH) between January 2011 and June 2025—with the actual follow-up cutoff date being June 2025, by which all patient outcomes and follow-up data had been completely collected—were included in this study. The diagnosis of systemic AL amyloidosis was based on internationally accepted criteria, requiring Congo red–positive deposits with characteristic apple-green birefringence under polarized light, immunohistochemical or mass-spectrometry confirmation of monoclonal κ or λ light-chain composition, and evidence of an underlying monoclonal gammopathy[14]. LCDD was diagnosed by renal immunofluorescence demonstrating linear deposition of monoclonal κ or λ light chains along the glomerular or tubular basement membranes, Congo red negativity, and granular electron-dense deposits on electron microscopy[15]. Demographic, clinical, laboratory, pathological, treatment, and follow-up data were extracted from the electronic medical record system at PKUFH. The study protocol was approved by the Biomedical Research Ethics Committee of Peking University First Hospital (approval No. 2021-yan-323) and conducted in accordance with *the Declaration of Helsinki*.

Clinical definitions and response assessment

Clinical staging and response assessment were performed according to internationally accepted criteria. Risk stratification for AL patients was based on the Mayo 2004 and Mayo 2012 staging systems, incorporating cardiac troponin I (cTnI), N-terminal pro–B-type natriuretic peptide (NT-proBNP), and the difference between involved and uninvolved free light chains (dFLC). In cases where NT-proBNP was unavailable and only BNP was measured, BNP thresholds of 81 pg/mL (for Mayo 2004) and 400 pg/mL (for Mayo 2012) were applied. Organ involvement and hematologic or organ responses were defined by established AL international criteria[10, 16, 17]. Given the absence of universally accepted criteria for organ involvement and treatment response in LCDD, the same AL-based criteria were applied to LCDD in this study to ensure methodological consistency and comparability between the two diseases. Overall survival (OS) was defined as the duration from diagnosis to death or last follow-up. Renal survival was assessed separately for light-chain amyloidosis (AL) and light-chain deposition disease (LCDD), which was defined as the duration from diagnosis to the initiation of dialysis. Renal survival of both AL and LCDD patients was subjected to stratified subgroup analysis based on baseline albumin (ALB < 30 g/L), estimated glomerular filtration rate (eGFR < 50 mL/min/1.73 m²), and 24-hour urinary total protein (24h-UTP > 5 g/24h).

Statistical analysis

All statistical analyses were performed using SPSS Statistics version 27.0.1. Continuous variables were expressed as medians with interquartile ranges (IQRs) and compared using the Mann–Whitney U test. Categorical variables were compared using the χ^2 test or Fisher's exact test. Correlations between variables were assessed using Spearman's rank correlation coefficients and visualized through correlation heatmaps. Overall survival and renal survival were estimated using the Kaplan–Meier method, and differences between groups were evaluated with the Log-rank test. Missing data were analyzed on an available-case basis without imputation. All P values were two-sided, and statistical significance was defined as $P < 0.05$.

Results

Patients

From 2011 to 2025, a total of 809 individuals with biopsy-confirmed amyloidosis and 75 with monoclonal immunoglobulin deposition disease (MIDD) were screened in this study. Among the amyloidosis cases, systemic involvement was predominant, and after excluding patients with non-AL or indeterminate amyloid subtypes and those with concomitant multiple myeloma or other B-cell lymphoproliferative disorders, 480 patients with primary systemic AL amyloidosis were included (Fig. 1A). Within the MIDD cohort, light-chain deposition constituted the vast majority; following the exclusion of patients with heavy-chain or combined light- and heavy-chain deposition (HCDD/LHCDD) and those with coexisting lymphoproliferative disorders, 61 patients fulfilled the diagnostic criteria for isolated light chain deposition disease (LCDD) (Fig. 1B). These two rigorously defined cohorts formed the final study population for subsequent analyses (Fig. 1).

Initial Clinical Manifestations

Marked differences in initial clinical manifestations were observed between AL amyloidosis and LCDD patients (Fig. 2). The most common initial clinical manifestations of AL amyloidosis were proteinuria and edema, accounting for approximately 32.1% and 45% respectively. In contrast, in LCDD, the proportions of these two manifestations are approximately 26.2% and 21.3% respectively. Elevated serum creatinine is the most common initial clinical manifestation in patients with LCDD (49.2%), whereas it is uncommon in patients with AL amyloidosis (4.6%). Additionally, symptoms such as respiratory and gastrointestinal manifestations were more prevalent in patients with AL amyloidosis.

Baseline Clinical and Laboratory Characteristics of AL and LCDD

Clinical characteristic between patients with AL amyloidosis and LCDD, were summarized in Table 1 and Fig. 3. AL amyloidosis is predominantly characterized by lambda light chain deposition (81.4%). In contrast, LCDD is absolutely dominated by kappa light chain deposition, with 80.3% of LCDD cases

presenting this subtype. AL amyloidosis showed high rates of nephrotic-range proteinuria (70.7%) and hypoalbuminemia (69.3%). In contrast, LCDD demonstrated higher clonal burden (dFLC > 180 mg/L: 73.3%; BMPC > 10%: 56.4%), and significantly more advanced renal dysfunction (eGFR < 50 mL/min/1.73 m²: 85.2%). These trends were reinforced by box-plot comparisons (Fig. 3), where LCDD exhibited lower 24h UTP and eGFR, higher dFLC and BMPC, than AL amyloidosis (all *P* < 0.001).

Table 1
Baseline Clinical and Laboratory Characteristics of Patients with AL and LCDD

	AL amyloidosis n = 480, cases (%)	**LCDD** n = 61, cases (%)	***P* value**
Age ≥ 65 years	159 (33.1)	18 (29.5)	0.571
Male sex	307 (64.0)	38 (62.3)	0.799
lambda light chain	390/479 (81.4)	12/61 (19.7)	< 0.001
dFLC > 180 mg/L	109/358 (30.4)	44/60 (73.3)	< 0.001
BMPC ratio > 10%	78/465 (16.8)	31/55 (56.4)	< 0.001
24-h UTP > 3.5g/24h	323/457 (70.7)	19/61 (32.2)	< 0.001
eGFR < 50 mL/min/1.73m²	134/479 (28.0)	52/61 (85.2)	< 0.001
ALB < 30 g/L	332/479 (69.3)	6 (9.8)	< 0.001
NT-proBNP > 332 ng/L	322/457 (70.5)	40/58 (69.0)	0.815
NT-proBNP > 1800 ng/L	157/457 (34.4)	17/58 (29.3)	0.444
LVEF < 45%	20/439 (4.6)	1/48 (2.1)	0.670
IVST > 12 mm	137/402 (34.1)	6/51 (11.8)	< 0.001

AL, light chain amyloidosis; ALB, albumin; BMPC, bone marrow plasma cell; dFLC, difference between involved and uninvolved free light chains; eGFR, estimated glomerular filtration rate; IVST, interventricular septal thickness; LCDD, light chain deposition disease; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; UTP, urinary total protein

Organ Involvement

Organ-involvement profiles in AL amyloidosis and LCDD were showed in Table 2. Compared to LCDD, AL amyloidosis tends to present with a pattern of multi-organ involvement. The percentage of patients with ≥ 2 organs involved in these two groups are 73.8% and 36.1%, respectively (*P* < 0.001). Renal and cardiac involvement are the most involved organs, with rates of 94.4% and 60.8% in AL amyloidosis, respectively, while in LCDD, these rates are 100% and 32.8%. In contrast, liver, nervous system, and gastrointestinal tract involvement are extremely rare in LCDD.

Table 2
Organ Involvement in Patients with AL and LCDD

Organs involvement	AL n = 480, cases (%)	LCDD n = 61, cases (%)	*P* value
≥ 3 organs involvement	149 (31.0)	1 (1.6)	< 0.001
≥ 2 organs involvement	354 (73.8)	22 (36.1)	< 0.001
Kidney	453 (94.4)	61 (100)	0.112
Heart	292 (60.8)	20 (32.8)	< 0.001
Liver	62 (12.9)	2 (3.3)	0.028
Nervous system	49 (10.2)	0 (0)	0.009
Gastrointestinal tract	53 (11.0)	0 (0)	0.006
Respiratory system	5 (1.0)	1 (1.6)	0.514
Skin	8 (1.7)	0 (0)	0.606
Soft tissue	6 (1.3)	0 (0)	1.000

Treatment Regimens and Hematologic Response

Notable disparities in treatment strategies and hematologic responses were observed between AL amyloidosis and LCDD patients (Table 3, Supplementary Tables 1–2). In terms of treatment allocation, 54.8% of AL patients received chemotherapy alone, and 17.7% underwent autologous stem cell transplantation (ASCT); in contrast, 75.4% of LCDD patients received chemotherapy alone, with only 1.6% undergoing ASCT. Both cohorts predominantly relied on proteasome inhibitor–based chemotherapy (66.3% in AL; 66.0% in LCDD).

Table 3
Hematologic Response for Patients with AL and LCDD

Hematologic Response	AL, cases (%)	LCDD, cases (%)	*P* value
CR	83/170 (48.8)	25/38 (65.8)	0.058
VGPR	36/170 (21.2)	4/38 (10.5)	0.132
PR	22/170 (13.0)	7/38 (18.4)	0.378
CR, Complete Response; PR, Partial Response; VGPR, Very Good Partial Response			

Hematologic responses (Table 3) showed a more favorable profile in LCDD patients. The overall hematologic response (≥ PR) rate of the LCDD patients was significantly superior to that of the AL patients, reaching 94.7% and 83%, respectively ($P = 0.036$). Notably, the hematologic complete response

(CR) rate also showed a distinct advantage (65.8% vs 48.8%).Subgroup analyses (Supplementary Table 2) confirmed robust responses to proteasome inhibitor–based (≥ VGPR: 63.5% in AL; 77.8% in LCDD) and daratumumab-based regimens (≥ VGPR: 82.9% in AL; 72.7% in LCDD) in both disorders.

Survival Outcomes in AL and LCDD

In the AL cohort, the median follow-up was 36 months, during which 152 patients (31.7%) died. The 1-, 3-, and 5-year OS rates were 83.8%, 72.4%, and 66.8%, respectively. Organ failure—predominantly renal or cardiac dysfunction—accounted for 49.3% (75/152) of these deaths. In contrast, the LCDD cohort had a median follow-up of 22 months, with only 2 deaths occurring; both were attributed to progressive renal failure. For overall survival (OS), LCDD patients exhibited significantly better outcomes than AL patients ($P < 0.001$); notably, neither cohort reached a median overall survival (i.e., < 50% of patients died during follow-up) over the study period.

As shown, the median renal survival for LCDD was 69 months, whereas AL patients did not reach a median renal survival (i.e., < 50% of AL patients experienced renal events during follow-up) and had a more favorable renal course with fewer renal events overall ($P < 0.001$, Fig. 4B). Stratified analyses further characterized disease-specific renal risk profiles: In patients with serum albumin (ALB) < 30 g/L, estimated glomerular filtration rate (eGFR) < 50 mL/min/1.73m² and 24-hour urinary total protein (24h-UTP) > 5 g/24h, AL was associated with superior renal survival compared to LCDD (all $P < 0.05$, Fig. 4C-E).

Discussion

Light chain amyloidosis (AL) and light chain deposition disease (LCDD) are classified within the spectrum of monoclonal immunoglobulin light chain associated disorders[17, 18]. However, clinical management remains challenging due to overlapping clinical phenotypes and inadequate early differential diagnosis. This study was based on a retrospective cohort from Peking University First Hospital, a national-level referral center for renal diseases. It is important to note the presence of referral selection bias— the proportion of patients with renal involvement may be higher than that in the general community population. We integrated multi-modal data, including molecular characteristics, clinical phenotypes, treatment exposure, and outcomes, to systematically characterize the key heterogeneities between AL and LCDD. Furthermore, we linked phenotypic findings with translatable mechanistic inferences and practical clinical insights, aiming to provide an evidence base for disease-specific, differentiated management strategies.

The fundamental distinction between AL and LCDD lies in the structural properties of abnormal monoclonal light chains and their deposition patterns in tissues. Monoclonal light chains in AL form β-sheet structures, which is the core mechanism underlying their assembly into amyloid fibrils (Congo red-positive). In contrast, monoclonal light chains in LCDD typically do not form canonical β-sheet structures; their deposition occurs primarily as non-fibrillar granular aggregates(Congo red-negative)[1, 2, 15] .These differences not only influence the pattern of organ involvement but also directly shape the overall

disease phenotypes and clinical diagnostic/treatment paradigms of the two disorders. Clinically, 75%-80% of AL cases produce monoclonal λ-type immunoglobulin light chains, whereas all reports in monoclonal immunoglobulin deposition disease (MIDD) consistently demonstrate a predominance of κ-type monoclonal light chains (accounting for 80–85%)[5, 17]. In our cohort, 81.4% of AL patients exhibited λ-LC predominance, and 80.3% of LCDD patients showed κ-LC predominance— confirming a difference in the primary pathogenic light chain isotypes between the two diseases, which is generally consistent with previous findings[7, 19, 20]. Regarding initial presenting symptoms, AL exhibits a multisystemic tendency—manifestations such as proteinuria, edema, and respiratory symptoms reflect its systemic pathogenic nature. In contrast, LCDD is characterized by prominent initial symptoms of elevated serum creatinine (Scr) and proteinuria, highlighting the dominant role of renal injury in the early disease stage. This difference aligns closely with the pattern of organ involvement: 73.8% of AL patients had involvement of ≥ 2 organs (60.8% with cardiac involvement), reflecting the multi-organ invasiveness of amyloid deposition. Renal involvement was a defining and nearly universal feature of LCDD in our cohort, whereas cardiac involvement was observed in only 32.8% of cases—underscoring the renal-limited nature of non-fibrillar light chain deposition[21]. This is consistent with the conclusion from previous LCDD studies that "lesions primarily target the renal microenvironment"[1, 4]. Differences in laboratory parameters further confirm the distinction between the two diseases: AL showed more significant abnormalities in multi-organ function indicators (e.g., albumin, cardiac troponin I [cTnI]), while LCDD exhibited severe abnormalities in renal function markers (e.g., Scr, estimated glomerular filtration rate [eGFR])— 85.2% of LCDD patients had eGFR < 50 mL/min/1.73m², compared to only 28.0% of AL patients.

The differences in pathogenesis and clinical phenotypes between AL and LCDD are also reflected in their therapeutic strategies and efficacy assessment— both share the core goal of reducing monoclonal LC burden. Proteasome inhibitors (PIs) serve as the backbone of first-line therapy for both disorders (used in 66.3% of AL patients and 66.0% of LCDD patients), a choice supported by extensive prior evidence demonstrating that PIs effectively inhibit the synthesis of monoclonal LCs[22–24]. As a systemic plasma cell clonal disorder, AL necessitates heterogeneous therapeutic strategies stratified by the extent of organ involvement and biological features. Specifically, 17.7% of patients underwent ASCT, 54.8% received chemotherapy alone, and 27.5% were administered palliative treatment; within the chemotherapy subgroup, regimens based on proteasome inhibitors (66.3%), daratumumab (20.6%), IMiDs (10.3%), and conventional chemotherapy (2.8%) constitute a multifaceted therapeutic system, fully reflecting the stratification and individualization of treatment approaches. Consistent with current clinical guidelines and consensus statements, daratumumab combined with PIs and alkylating agents (e.g., Dara-CyBorD) has emerged as the preferred first-line regimen for most patients. In carefully selected populations with low disease burden or mild organ damage, ASCT remains capable of achieving deep clonal remission and long-term survival benefits[9, 11, 25, 26]. Our observation that "the non-response rate to traditional chemotherapy (predominantly alkylating agents/hormones alone) reached 100%" aligns with the recent shift in the AL treatment landscape: traditional chemotherapy alone is no longer recommended as first-line therapy or is only considered in specific clinical scenarios. This is attributed to

its inability to rapidly and deeply reduce pathological free light chains (FLCs), thereby failing to reverse ongoing damage to key organs (especially the heart and kidneys). In contrast, the phase Ⅲ ANDROMEDA trial demonstrated that Dara-CyBorD significantly improved hematologic and organ response rates in AL patients[8, 11, 25, 27]. Compared with AL, a higher proportion of LCDD patients in our cohort opted for the "simplified treatment regimen (chemotherapy alone)": 75.4% of LCDD patients received chemotherapy alone, compared to 54.8% of AL patients; in the small LCDD subgroup treated with daratumumab, the ≥ partial response (PR) rate reached 100% (n = 11). Prior evidence also suggests that early, deep FLC reduction in LCDD is associated with subsequent improvements in renal function and overall outcomes; multi-center retrospective studies of daratumumab in LCDD have further shown trends of rapid FLC reduction and stable renal function. It is important to emphasize that current daratumumab-related evidence in LCDD is limited to case reports and small case series, necessitating prospective validation[4, 18, 28].

In terms of survival, our Kaplan–Meier analysis identified significant differences in overall survival (OS) and renal survival between AL and LCDD (Fig. 4). Consistent with prior research, LCDD patients had notably better OS than AL patients ($P < 0.001$)—a pattern aligned with established disease mechanisms: AL's mortality risk is primarily driven by cardiac involvement, where biomarkers like troponin and NT-proBNP are validated for risk stratification across cohorts, and cardiac failure elevates death risk. By contrast, LCDD is renal-dominant, with fewer life-threatening extra-renal complications, explaining its superior OS.

Renal survival, however, showed an opposite trend: AL patients had significantly better renal outcomes than LCDD patients ($P < 0.001$, Fig. 4B). Though AL had hypoalbuminemia and high 24-hour urinary protein (Fig. 3A, D), it exhibited higher eGFR (Fig. 3C); LCDD had higher clonal burden (dFLC, Fig. 3E) and lower eGFR, resulting in poorer renal survival. Notably, AL maintained better renal survival in subgroups defined by albumin < 30 g/L, eGFR < 50 mL/min/1.73m², and 24-hour urinary protein > 5 g/24h (Fig. 4C-E), highlighting the need for precise stratification. The differential treatment responses (poor renal survival in LCDD; AL requiring diverse approaches to control cardiac risk and sustain renal survival) further reflect their distinct pathophysiologies. This is consistent with previous conclusions: the mortality risk of AL is primarily driven by cardiac involvement, and the core role of cardiac biomarkers in risk stratification has been validated. In contrast, LCDD is dominated by renal involvement, with fewer life-threatening extra-renal complications, leading to better overall survival outcomes[8, 9, 29, 30].

The limitations of this study include selection bias inherent to Peking University First Hospital, a tertiary referral center for renal diseases (resulting in overrepresentation of patients with severe renal involvement), incomplete follow-up, and small sample sizes in certain subgroups—all of which limit the generalizability of the findings. The absence of repeated renal biopsies prevented us from assessing treatment responses at the histological level (e.g., regression of amyloid deposits). Although prior studies have demonstrated a strong correlation between histological regression and deep free light chain (FLC) remission, renal tissue progression has been observed even in the setting of hematologic complete remission (CR); thus, when feasible, repeated biopsies may help resolve the clinical paradox of

"hematologic remission without organ response"[31, 32]. In addition, the study spans more than a decade, during which diagnostic methods and therapeutic regimens evolved markedly (e.g., increased use of proteasome inhibitors, introduction of daratumumab-based therapies, and improvements in supportive care). These temporal changes may introduce some treatment-era heterogeneity and should be considered when interpreting the results.

In conclusion, AL and LCDD warrant disease-specific management strategies: AL should prioritize the achievement of deep hematologic remission (≥ very good partial response [VGPR]/complete remission [CR]), while LCDD should be renal outcome-oriented, with priority given to proteasome inhibitor (PI)/anti-CD38-based regimens for pathogenic light chain clearance. Clinical follow-up should focus on the integrated assessment of NT-proBNP/cardiac troponin, estimated glomerular filtration rate (eGFR)/serum creatinine (sCr), difference between involved and uninvolved FLCs (dFLC), and albumin to guide treatment decisions. Future studies should conduct multicenter prospective trials with standardized organ response endpoints and validate the causal relationship between FLC reduction and organ response via repeated biopsies or functional imaging modalities.

Declarations

Ethics approval and consent to participate

The Ethics Committee of Peking University First Hospital approved this study (No. 2021-yan-323). Signed written consent was obtained from all patients.

Data availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Competing interests

The authors declare that they have no competing interests.

Author Contributions

Yiming Zhao and Weiwei Xie contributed equally. Yiming Zhao designed the study, curated data, performed statistical analyses, and drafted the manuscript. Yujun Dong and Fude Zhou conceived and supervised the project and critically revised the manuscript. Weiwei Xie, Bingjie Wang, Bo Tang, Jianbei Zhao, Huihui Liu, Zhizhen Lai, Xiaoying Yang, Wenqiong Wang, Hongtao Ling, Shuanglian Xie, and Shujing Guo enrolled patients, collected/verified clinical data, and contributed to manuscript revision. All authors approved the final version and agree to be accountable for all aspects of the work.

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Figures

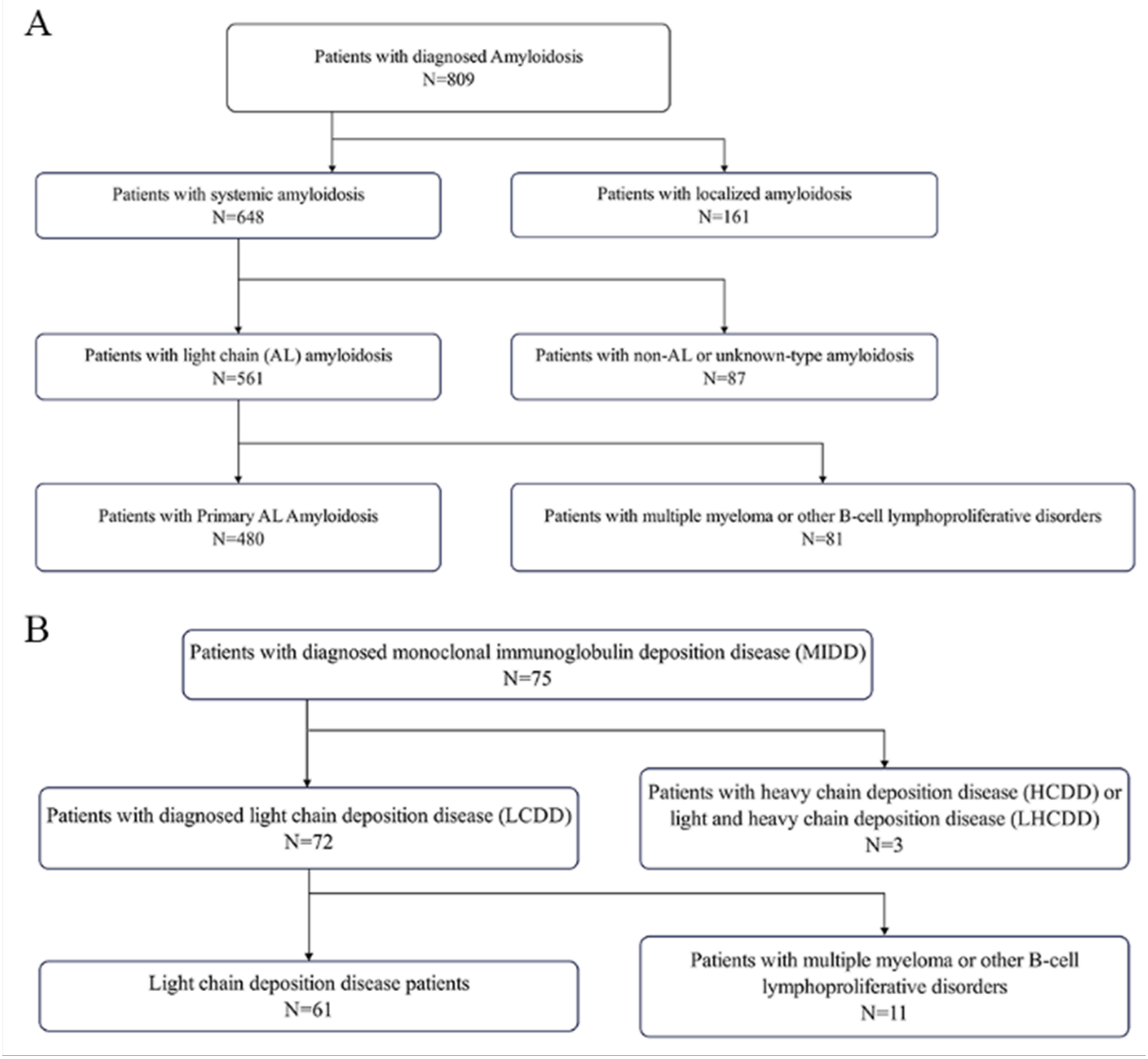


Figure 1

Patient enrollment flowchart for light chain amyloidosis (A) and light chain deposition disease (B).

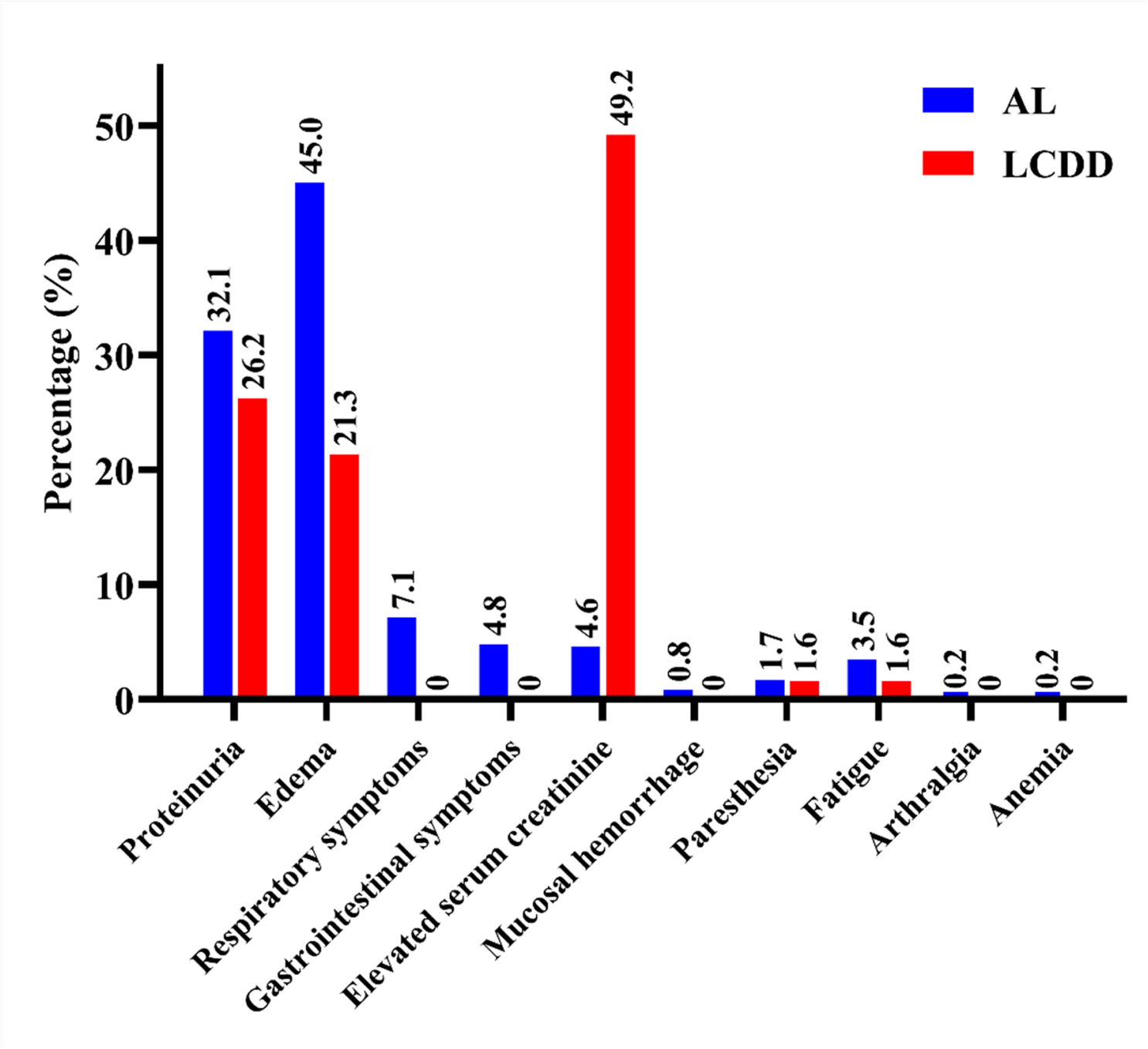


Figure 2

Initial clinical manifestations for patients with light chain amyloidosis (AL) and light chain deposition disease (LCDD).

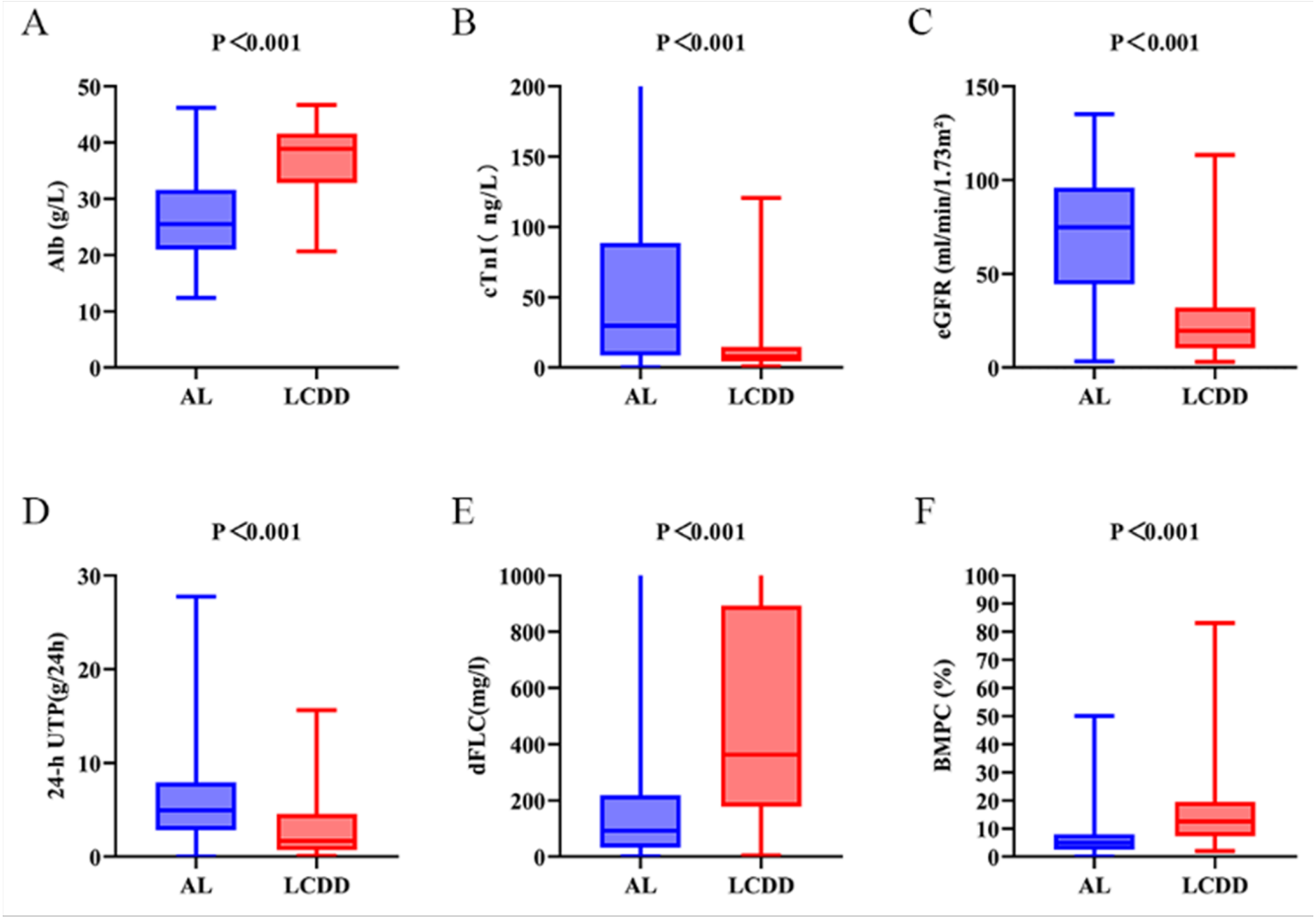


Figure 3

Box plots of laboratory parameters in patients with AL amyloidosis and LCDD. Box plots of albumin (Alb, A), cardiac troponin I (cTnI, B), estimated glomerular filtration rate (eGFR, C), 24-hour urinary total protein (24hUTP, D), difference between involved and uninvolved free light chains (dFLC, E), and bone marrow plasma cell (BMPC, F) in patients with AL amyloidosis and LCDD.

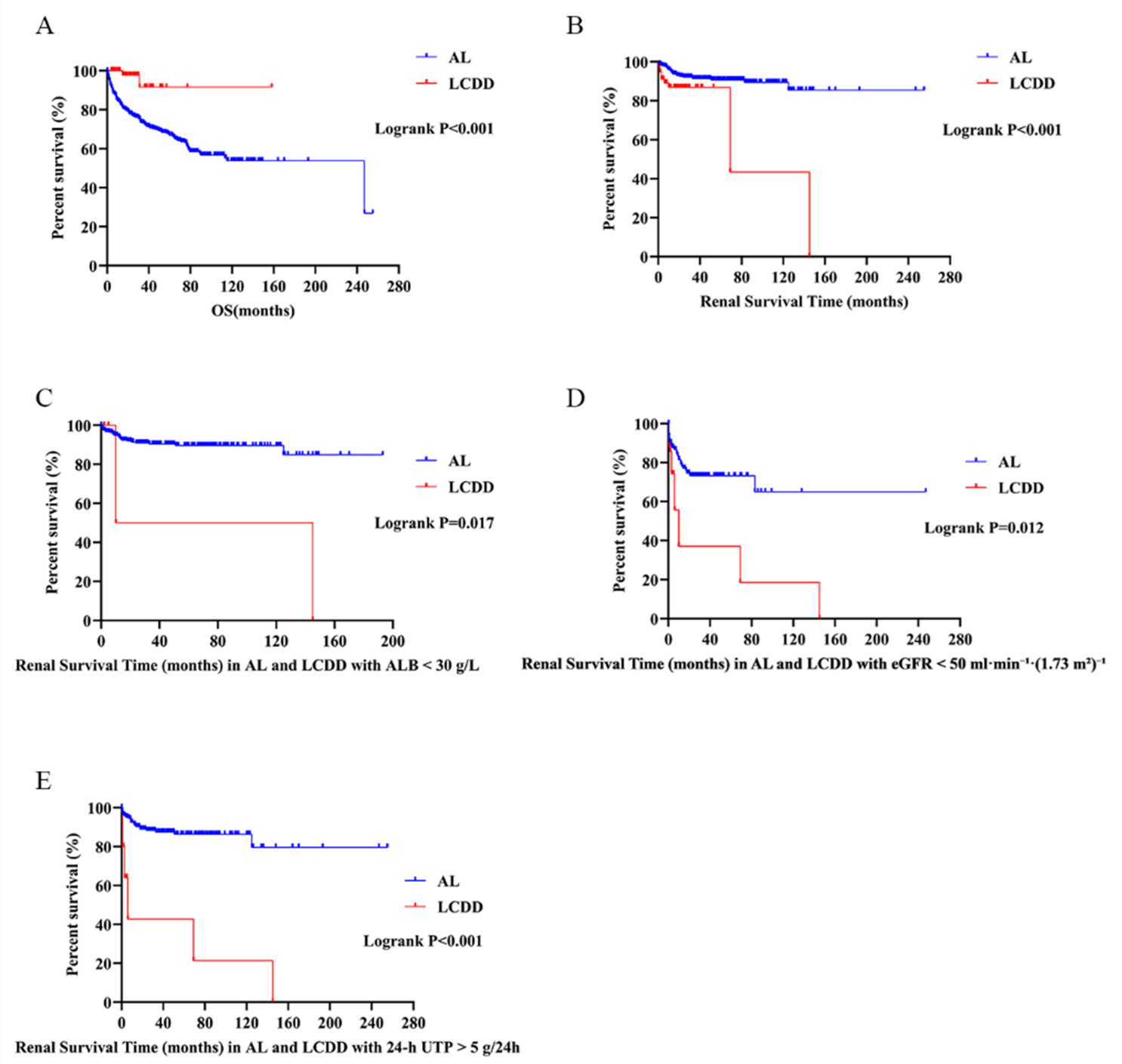


Figure 4

Overall and renal survival in AL amyloidosis (AL) and light chain deposition disease (LCDD). The Kaplan-Meier curves for overall survival (OS) and renal survival (endpoint: initiation of dialysis) demonstrate significant differences between AL and LCDD patients ($P < 0.001$ for both OS and renal survival). Renal survival curves in AL and LCDD patients with albumin (ALB) < 30 g/L and estimated glomerular filtration rate (eGFR) < 50 mL/min/1.73m² also show statistically significant differences ($P = 0.017$ and $P = 0.012$, respectively). Additionally, renal survival was significantly different in patients with 24-hour urinary total protein (UTP) > 5 g/24h ($P < 0.001$). The renal survival curves in the renal survival panels reach zero as

the final renal event occurred at the latest follow-up time, with no patients remaining at risk after this point. Abbreviations: OS, overall survival

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