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The kidney, heart, and skin manifestations; a triad of systemic burden in hemodialysis patients – a narrative review

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ABSTRACT

End-stage renal disease (ESRD) affects a growing number of patients worldwide, and hemodialysis (HD) remains the most common form of kidney replacement therapy despite its incomplete correction of uremic physiology. Patients on maintenance HD carry a disproportionate burden of cardiovascular diseases (CVDs), which remains the leading cause of death in this population and is amplified by chronic inflammation, anemia, and accumulation of protein-bound uremic toxins such as indoxyl sulfate. Left ventricular hypertrophy, arrhythmia, and sudden cardiac death are common downstream consequences of this cardio-renal interplay. Simultaneously, the skin serves as an under-recognized but revealing organ in uremic manifestations, necrosis, pruritus, pigmentary change, and occasionally calciphylaxis. Uremic pruritus alone affects a substantial proportion of HD patients internationally and is linked to poor sleep, depression, and reduced quality of life. Emerging evidence suggests that cardiovascular and dermatological manifestations are not isolated phenomena but rather converge through shared pathways of systemic inflammation, oxidative stress, and inadequate dialysis clearance. This review synthesizes current literature describing the cardiovascular, dermatological, and interconnected systemic burden experienced by HD patients and argues for integrated, multidisciplinary models of care that jointly address nephrological, cardiological, and dermatological needs.

Review

Implication for health policy/practice/research/medical education:
This narrative review shows that hemodialysis (HD) patients face a systemic triad in which cardiovascular diseases (CVDs), common skin disorders (particularly xerosis and uremic pruritus), and shared uremic pathophysiology interact to worsen outcomes and quality of life. The CVDs, driven by left ventricular hypertrophy, anemia, uremic toxins, and arrhythmias, is the leading cause of death, while dermatological manifestations are highly prevalent yet frequently under-recognized markers of the same underlying inflammatory and toxin-mediated milieu. The review argues that these cardiac and cutaneous complications are mechanistically intertwined rather than separate problems and therefore calls for multidisciplinary, integrated care models and research targeting shared pathways (inflammation, oxidative stress, protein-bound toxins) to improve survival and patient-reported outcomes in HD populations.

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Introduction

Chronic kidney disease (CKD) progressing to end-stage renal disease (ESRD) represents one of the most pressing challenges in global health, with millions of patients now dependent on kidney replacement therapy (1,2). Hemodialysis (HD) remains the dominant modality worldwide, yet substantial disparities persist in access, quality, and outcomes across regions with differing resources (1). Even where HD is delivered according to contemporary standards, it corrects only a fraction of the physiological derangements caused by kidney failure, leaving patients exposed to persistent uremic toxin accumulation, chronic volume fluctuation, and systemic inflammation between sessions (3,4). These residual abnormalities do not remain confined to renal physiology; instead, they radiate outward to affect multiple organ systems simultaneously, producing a recognizable triad of systemic burden (3-5). Cardiovascular disease (CVD) dominates this triad as the principal cause of mortality among HD patients, accounting for a disproportionate share of deaths relative to the general population (4,6). Less visible, but no less important, are the dermatological manifestations of uremia, which are frequently underappreciated in clinical practice despite their high prevalence and considerable impact on daily life (5,7). Xerosis, pruritus, pigmentary changes, and vascular skin complications such as calciphylaxis collectively signal the broader systemic disturbance produced by kidney failure (5,7,8). Importantly, cardiovascular and dermatological manifestations do not evolve independently; both are shaped by shared mechanisms including chronic micro-inflammation, oxidative stress, and the retention of protein-bound uremic toxins that are poorly cleared by conventional dialysis (3,9). Understanding HD-associated disease as an interconnected triad, renal failure driving cardiovascular and cutaneous pathology through common pathophysiological threads, offers a more coherent framework for both clinical management and future research (4,5,9). This narrative review synthesizes current evidence on cardiovascular and dermatological manifestations in HD patients, examines their shared mechanistic underpinnings, and considers the implications for integrated multidisciplinary care (1,4).

Search strategy

A focused literature search was conducted using PubMed, Scopus, Web of Science, Cochrane Library, and Google Scholar, restricted to English-language, peer-reviewed articles published up to 2025. Search terms included combinations of "hemodialysis," "end-stage renal disease," "cardiovascular disease," "left ventricular hypertrophy," "uremic toxins," "uremic pruritus," "xerosis," "calciphylaxis," and "quality of life." Priority was given to original research, systematic reviews, and authoritative narrative reviews addressing cardiovascular or dermatological complications of HD. Articles lacking peer review, non-English texts, and studies unrelated to the dialysis population were excluded, and reference lists of key articles were also examined to identify additional relevant sources.

Cardiovascular manifestations in HD

Cardiovascular diseases have long been recognized as the leading cause of death among patients receiving HD, contributing to mortality rates far exceeding those observed in age-matched individuals with normal renal function (6). This excess risk cannot be explained by traditional cardiovascular risk factors alone, prompting investigators to examine the role of uremia-specific processes such as chronic inflammation and oxidative stress in accelerating cardiovascular injury; Dai et al describe how persistent low-grade inflammation in ESRD interacts with malnutrition and vascular calcification to create a self-perpetuating cycle of cardiovascular damage, often termed the malnutrition-inflammation-atherosclerosis complex (3). Zoccali et al, writing on behalf of the European Renal and Cardiovascular Medicine Working Group, further emphasize that chronic kidney disease should be regarded as a CVD equivalent, given the density of shared pathophysiological pathways linking the two conditions (4). Left ventricular hypertrophy is among the most prevalent and consequential cardiac abnormalities observed in this population, arising from a combination of chronic volume overload, hypertension, and anemia-induced compensatory mechanisms; Chao et al demonstrated that the geometric pattern of left ventricular hypertrophy, when combined with coexistent vascular calcification, independently predicts cardiovascular mortality in patients with end-stage kidney disease, this finding underscores that structural cardiac remodeling in HD patients is not a benign adaptation but rather an active marker of accumulating cardiovascular risk (10). Anemia plays a particularly important role in this process, as chronic reduction in oxygen-carrying capacity forces the heart to increase cardiac output, thereby promoting ventricular remodeling over time; Hayashi et al report that anemia present at the initiation of HD is associated with an elevated risk of early cardiovascular events, suggesting that inadequate erythropoiesis correction leaves patients vulnerable from the outset of dialysis (11). Uremic toxins represent another mechanistically important cardiovascular insult unique to the dialysis population; Lim et al provide a detailed account of how protein-bound toxins, particularly indoxyl sulfate, accumulate progressively in patients with declining kidney function, contributing to endothelial dysfunction, vascular inflammation, and accelerated atherosclerosis (9). Wakamatsu et al extend this mechanistic picture by demonstrating that indoxyl sulfate induces macrophage toxicity and foam cell formation within arterial plaques, thereby promoting the kind of unstable atherosclerotic lesions that underlie

acute cardiovascular events (12). Charytan et al further articulate the bidirectional nature of cardio-renal injury in uremia, showing that the bone-mineral axis and anemia serve as mediators linking kidney dysfunction to progressive cardiac damage (13). This bidirectional interplay, where cardiac dysfunction itself impairs renal perfusion and accelerates kidney disease progression, exemplifies the complexity of managing cardiovascular risk in the HD population (4,13). Arrhythmia and sudden cardiac death represent the most feared cardiovascular outcomes in this group (14,15). Makar and Pun highlight that sudden cardiac death among HD patients is driven by a combination of structural myocardial disease, electrolyte instability during and between dialysis sessions, and autonomic dysfunction (14). Previously, Samanta and colleagues add that bradyarrhythmia and ventricular tachyarrhythmia each contribute significantly to the arrhythmic burden in ESRD, and that conventional risk stratification tools developed in the general population may underperform in dialysis patients (15). Effective cardiovascular management in HD therefore requires a strategy that reaches beyond standard cardio-protective pharmacotherapy to address the uremia-specific drivers of heart disease (4).

Dermatological manifestations in HD patients

The skin is frequently overlooked as a diagnostic window into systemic uremic disease, yet cutaneous manifestations are extraordinarily common among patients receiving HD; Markova and colleagues note that dermatological changes in this population are often dismissed as cosmetic nuisances rather than recognized as clinically meaningful indicators of underlying metabolic derangement (5). Onelmis et al similarly document a high prevalence of cutaneous abnormalities among HD patients, reporting that pigmentary changes, xerosis, and pruritus are observed in the majority of individuals undergoing long-term dialysis treatment (7). Uremic xerosis, characterized by dry, rough, and often scaling skin, is among the most prevalent dermatological findings in this population and has been linked to abnormal sebaceous gland function as well as reduced dermal hydration; Szepietowski et al explain that xerosis in uremic patients likely reflects a combination of factors, including atrophy of sweat glands, altered stratum corneum lipid composition, and vitamin A accumulation secondary to impaired renal clearance, because xerosis compromises the integrity of the skin barrier, it can also predispose patients to secondary infection and may itself act as a trigger for pruritus, compounding patient discomfort (8). Uremic pruritus represents perhaps the most burdensome dermatological complication of HD, with international data indicating that it affects a substantial proportion of patients across diverse healthcare settings; Rayner and colleagues, reporting findings from the Dialysis Outcomes and Practice Patterns Study, demonstrate that pruritus remains common and frequently underdiagnosed, with considerable variation in awareness and treatment practices between countries, this variation suggests that pruritus is not merely an inevitable consequence of uremia but is also shaped by differences in clinical recognition, dialysis quality, and access to targeted therapy (16). The pathophysiology of uremic pruritus is multifactorial and incompletely understood, but current evidence implicates dysregulation of the endogenous opioid system, peripheral and central sensitization, immune system activation, and the accumulation of incompletely cleared uremic solutes (17,18). Combs and colleagues point to the role of divalent ion imbalance, particularly elevated phosphate and calcium levels, as contributors to pruritic symptom generation in patients with chronic kidney disease, connecting dermatological symptoms to the broader mineral metabolism dysregulation of uremia (17). Manenti et al provide a comprehensive account of the clinical features of uremic pruritus, noting that it characteristically worsens at night and during HD sessions, and that its intensity correlates poorly with objective laboratory markers in many patients (18). The quality-of-life consequences of uremic pruritus are profound and extend well beyond physical discomfort (18,19). The study by Manenti et al also describes how chronic pruritus in dialysis patients frequently causes sleep disruption, mood disturbance, and a sense of social isolation, all of which contribute to a substantially diminished sense of well-being (18). Al-Nashri and Almutary report that depression and anxiety exert independent negative effects on the quality of life of HD patients, and that the symptom burden, including dermatological symptoms, acts as a mediator in this relationship (19). Treatment of uremic pruritus remains challenging, with no single agent achieving consistent efficacy across the population; Simons et al, in a systematic review of randomized controlled trials, conclude that gabapentin shows reasonable evidence for prurito-genic relief in HD patients, though study heterogeneity and methodological limitations temper the strength of this conclusion (20). Cirstea and colleagues provide a more recent appraisal of treatment options, noting that emerging therapies targeting the opioid receptor system, including kappa-opioid receptor agonists such as difelikefalin, have shown meaningful efficacy in clinical trials, offering hope for patients who do not respond to older agents (21). Beyond xerosis and pruritus, HD patients are susceptible to a range of other cutaneous manifestations (5,7). Markova et al describe pigmentary changes, particularly a yellowish-grey discoloration of the skin related to carotenoid and nitrogenous waste retention, as well as half-and-half nails, which offer a clinical sign of chronic renal failure at the bedside (5). Likewise, calciphylaxis, a severe and life-threatening complication characterized by painful ischemic skin lesions resulting from vascular calcification and thrombosis, represents the most serious cutaneous manifestation in this population, requiring

urgent recognition and interdisciplinary management (5,7).

Interplay between cardiovascular and skin manifestations

Although CVD and dermatological complications are typically discussed as separate domains within nephrology, a growing body of evidence suggests that they arise from overlapping pathophysiological processes (3,9). Chronic systemic inflammation, a hallmark feature of ESRD, contributes simultaneously to vascular injury and cutaneous nerve sensitization, linking cardiovascular risk and pruritic symptoms through a common inflammatory substrate; Dai et al describe how persistent inflammation in uremia fuels a cascade of endothelial dysfunction and tissue injury that is not confined to the vasculature alone but extends to other organ systems, including the skin (3). Protein-bound uremic toxins, particularly indoxyl sulfate, further exemplify this shared pathophysiology, as their accumulation has been implicated in both vascular calcification and heightened oxidative stress that may plausibly extend to cutaneous tissue (9,12). Lim et al note that the same toxins responsible for driving atherosclerotic changes in uremic patients also contribute to a broader state of systemic oxidative injury, reinforcing the concept that skin and cardiovascular pathology are two visible expressions of a single underlying uremic milieu (9). Clinically, uremic pruritus has also emerged as more than a dermatological nuisance, functioning instead as a potential marker of inadequate dialysis clearance and, by extension, of unresolved cardiovascular risk; Rayner and colleagues observe that variation in pruritus prevalence across dialysis centers correlates with differences in dialysis practice, suggesting that inadequate solute clearance may simultaneously worsen both cutaneous symptoms and the burden of circulating uremic toxins relevant to CVD (16). This observation positions pruritus as a potentially useful bedside indicator that could prompt clinicians to reassess dialysis adequacy and, in turn, address a shared driver of both skin and cardiovascular pathology (16,20). Skin signs in dialysis patients also carry direct cardiovascular implications in certain scenarios (5). Calciphylaxis, beyond its devastating dermatological course, reflects a state of advanced vascular calcification that is mechanistically continuous with the calcific arterial disease that drives cardiovascular mortality in end-stage kidney disease (5,7). Markova et al approved that the recognition of calciphylaxis lesions in clinical practice should trigger immediate evaluation of the patient's cardiovascular risk profile, given that the same mineral metabolism derangements underlie both the cutaneous and vascular disease (5). The quality-of-life impact of carrying both cardiovascular and dermatological burdens simultaneously is considerable and underappreciated in the existing literature; Al-Nashri and Almutary document that psychological distress in HD patients is shaped by the accumulation of multiple concurrent symptoms, with patients experiencing combined physical and emotional burden reporting the worst quality-of-life outcomes (19). Recently, Manenti et al reinforce this picture by showing that pruritus-related sleep loss and mood disturbance are not simply symptomatic inconveniences but active contributors to the overall clinical deterioration of HD patients (18). Recognizing this convergent burden points clearly toward the need for clinical models that assess and address cardiovascular and dermatological health simultaneously, rather than treating them as independent consultations (4,5). Notably, Zoccali and colleagues call for a more integrated approach to the care of patients with kidney disease and cardiovascular complications, noting that fragmented specialty care is poorly equipped to manage the complex, multi-system physiology of ESRD (4). Bringing dermatological awareness into this integrated framework is a logical extension of that argument, given the mechanistic and clinical evidence linking skin manifestations to the broader uremic burden (5,16).

Clinical implications and future directions

The overlapping burden of cardiovascular and dermatological disease in HD patients carries direct implications for how care should be organized and delivered (1,4). Traditional models of nephrology care, which often address cardiac and cutaneous complaints through separate referral pathways, may be poorly suited to a patient population in which these manifestations are mechanistically intertwined (4,5). A more effective model would involve multidisciplinary teams in which nephrologists, cardiologists, and dermatologists collaborate closely, sharing clinical information to identify patients at risk of compounding cardiovascular and cutaneous complications (4). Bello and colleagues, in their epidemiological review of HD outcomes, highlight substantial global variation in the quality and comprehensiveness of dialysis care, suggesting that structural barriers to multidisciplinary collaboration remain common in many healthcare settings (1). Thurlow et al similarly document persistent disparities in access to comprehensive kidney replacement therapy across regions, reinforcing the notion that improvements in integrated care must be pursued alongside broader efforts to expand equitable access to dialysis services (2). Dialysis adequacy optimization represents a particularly promising shared target for both cardiovascular and dermatological improvement, given its documented relevance to uremic toxin clearance, pruritus severity, and cardiovascular risk reduction (16,20,21). Simonsen and colleagues note that while pharmacological treatments for pruritus show variable efficacy, ensuring adequate dialysis delivery remains a foundational, low-cost intervention with potential benefits extending beyond symptom control alone (20). Future research should prioritize the identification of biomarkers capable of capturing the shared inflammatory and toxin-mediated pathways

linking cardiovascular and cutaneous disease, which could enable earlier risk stratification and more targeted therapeutic intervention; at present, indoxyl sulfate has emerged as a candidate biomarker that bridges both domains, but its clinical utility as a therapeutic target remains to be established in large, prospective trials (9,12). Lim et al suggest that pharmacological strategies aimed at reducing protein-bound toxin accumulation, such as gut microbiome modulation and improved dialysis membranes, may offer a dual benefit in reducing both cardiovascular and dermatological complications, though rigorous clinical evidence remains scarce (9). Similarly, emerging treatments for uremic pruritus that act through the opioid receptor system may have immune-modulatory properties that extend beyond itch control, potentially influencing the inflammatory milieu relevant to CVD; this hypothesis warrants investigation in future trials (21). The importance of systematically measuring quality of life as an outcome in HD research is also underscored by the findings reviewed here; Al-Nashri and Almutary demonstrate that psychological well-being is a significant mediator of overall health outcomes in this population, yet mental health assessment remains inconsistently integrated into routine dialysis care (19). Incorporating validated symptom burden and quality-of-life tools, alongside cardiovascular and biochemical markers, into routine clinical monitoring would provide a more complete picture of patient status and allow earlier identification of those at risk of deterioration across multiple organ domains (1,4).

Conclusion

Hemodialysis patients face a triad of systemic burden in which CVD, dermatological complications, and the shared pathophysiology linking them combine to affect morbidity, mortality, and quality of life profoundly. Cardiovascular disease remains the foremost cause of death in this population, driven by left ventricular hypertrophy, anemia, uremic toxin accumulation, and arrhythmic vulnerability. Dermatological manifestations, particularly xerosis and pruritus, are highly prevalent yet frequently under-recognized, despite their considerable impact on sleep, mood, and overall well-being. The two domains are not independent; rather, they converge through common mechanisms of inflammation, oxidative stress, and inadequate uremic toxin clearance, meaning that interventions targeting one system may plausibly benefit the other. Recognizing this interconnected burden calls for a shift away from soloed specialty care toward genuinely multidisciplinary models that integrate nephrology, cardiology, and dermatology. Continued research into shared biomarkers and targeted interventions will be essential to refining this integrated approach and ultimately improving outcomes for the growing global population of patients dependent on HD.

Authors' contribution

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Writing–original draft: All authors.
Writing–review and editing: All authors.
Supervision: All authors.

Conflicts of interest

The authors declare that they have no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the authors used generative artificial intelligence (Perplexity) and AI-assisted writing tools (Grammarly) to assist with drafting, language editing, and refinement of grammar and style. All content generated by these tools was subsequently critically reviewed, revised, and validated by the authors, who take full responsibility for the accuracy, integrity, and originality of the work.

Ethical issues

Ethical issues (including plagiarism, data fabrication, and double publication) have been fully addressed by the authors.

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References

1. Bello AK, Okpechi IG, Osman MA, Cho Y, Htay H, Jha V, et al. Epidemiology of haemodialysis outcomes. Nat Rev Nephrol. 2022;18:378–95. doi: 10.1038/s41581-022-00542-7.
2. Thurlow JS, Joshi M, Yan G, Norris KC, Agodoa LY, Yuan CM, et al. Global Epidemiology of End-Stage Kidney Disease and Disparities in Kidney Replacement Therapy. Am J Nephrol. 2021;52:98–107. doi: 10.1159/000514550.
3. Dai L, Golembiewska E, Lindholm B, Stenvinkel P. End-Stage Renal Disease, Inflammation and Cardiovascular Outcomes. Contrib Nephrol. 2017;191:32–43. doi: 10.1159/000479254.
4. Zoccali C, Mallamaci F, Adamczak M, de Oliveira RB, Massy ZA, Sarafidis P, et al. Cardiovascular complications in chronic kidney disease: a review from the European Renal and Cardiovascular Medicine Working Group of the European Renal Association. Cardiovasc Res. 2023;119:2017–32. doi: 10.1093/cvr/cvad083.
5. Markova A, Lester J, Wang J, Robinson-Bostom L. Diagnosis of common dermopathies in dialysis patients: a review and update. Semin Dial. 2012;25:408–18. doi: 10.1111/j.1525-139X.2012.01109.x.
6. Collins AJ. Cardiovascular mortality in end-stage renal disease. Am J Med Sci. 2003;325:163–7. doi: 10.1097/00000441-200304000-00002.
7. Onelmis H, Sener S, Sasmaz S, Ozer A. Cutaneous changes in patients with chronic renal failure on hemodialysis. Cutan Ocul Toxicol. 2012;31:286–91. doi: 10.3109/15569527.2012.657726.
8. Szepietowski JC, Reich A, Schwartz RA. Uraemic xerosis. Nephrol Dial Transplant. 2004;19:2709–12. doi: 10.1093/ndt/gfh480.
9. Lim YJ, Sidor NA, Tonial NC, Che A, Urquhart BL. Uremic Toxins in the Progression of Chronic Kidney Disease and Cardiovascular Disease: Mechanisms and Therapeutic Targets. Toxins (Basel). 2021;13:142. doi: 10.3390/toxins13020142.

10. Chao CT, Liao MT, Wu CK. Left Ventricular Hypertrophy Geometry and Vascular Calcification Co-Modify the Risk of Cardiovascular Mortality in Patients with End-Stage Kidney Disease: A Retrospective Cohort Study. J Atheroscler Thromb. 2023;30:1242–54. doi: 10.5551/jat.63870.
11. Hayashi T, Joki N, Tanaka Y, Hase H. Anaemia and early phase cardiovascular events on haemodialysis. Nephrology (Carlton). 2015;20 Suppl 4:1–6. doi: 10.1111/nep.12642.
12. Wakamatsu T, Yamamoto S, Yoshida S, Narita I. Indoxyl Sulfate-Induced Macrophage Toxicity and Therapeutic Strategies in Uremic Atherosclerosis. Toxins (Basel). 2024;16:254. doi: 10.3390/toxins16060254.
13. Charytan DM, Fishbane S, Malyszko J, McCullough PA, Goldsmith D. Cardiorenal Syndrome and the Role of the Bone-Mineral Axis and Anemia. Am J Kidney Dis. 2015;66:196–205. doi: 10.1053/j.ajkd.2014.12.016.
14. Makar MS, Pun PH. Sudden Cardiac Death Among Hemodialysis Patients. Am J Kidney Dis. 2017;69:684–95. doi: 10.1053/j.ajkd.2016.12.006.
15. Samanta R, Chan C, Chauhan VS. Arrhythmias and Sudden Cardiac Death in End Stage Renal Disease: Epidemiology, Risk Factors, and Management. Can J Cardiol. 2019;35:1228–40. doi: 10.1016/j.cjca.2019.05.005.
16. Rayner HC, Larkina M, Wang M, Graham-Brown M, van der Veer SN, Ecder T, et al. International Comparisons of Prevalence, Awareness, and Treatment of Pruritus in People on Hemodialysis. Clin J Am Soc Nephrol. 2017;12:2000–7. doi: 10.2215/cjn.03280317.
17. Combs SA, Teixeira JP, Germain MJ. Pruritus in Kidney Disease. Semin Nephrol. 2015;35:383–91. doi: 10.1016/j.semnephrol.2015.06.009.
18. Manenti L, Tansinda P, Vaglio A. Uraemic pruritus: clinical characteristics, pathophysiology and treatment. Drugs. 2009;69:251–63. doi: 10.2165/00003495-200969030-00002.
19. Al-Nashri F, Almutary H. Impact of anxiety and depression on the quality of life of haemodialysis patients. J Clin Nurs. 2022;31:220–30. doi: 10.1111/jocn.15900.
20. Simonsen E, Komenda P, Lerner B, Askin N, Bohm C, Shaw J, et al. Treatment of Uremic Pruritus: A Systematic Review. Am J Kidney Dis. 2017;70:638–55. doi: 10.1053/j.ajkd.2017.05.018.
21. Cîrstea Ş, Orzan OA, Zilişteanu DS. Pruritus in Uremic Patients: Approaches to Alleviating a Common Symptom in Chronic Kidney Disease. Life (Basel). 2025;15:1001. doi: 10.3390/life15071001.