Research Article Volume 01, Issue No. 02. 2025

The Medical Journal of Clinical Pathology and Microbiology



Mitochondrial dysfunction in kidney injury

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Article History :

Received date : 2024/07/17
Revised date : 2024/08/09
Accepted date : 2024/09/11
Published date : 2025/01/12

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ABSTRACT

Background: The body of literature reviewed collectively underscores the critical role of mitochondrial dysfunction in kidney injury, particularly in the contexts of acute kidney injury (AKI) and chronic kidney disease (CKD). The foundational work by (A. Popkov et al., 2019) establishes that mitochondria are not only targets but also sources of uremic toxins, contributing to oxidative stress and subsequent renal cell death. This dual role is pivotal in understanding the mechanisms that lead to kidney damage. **Literature Review:** Subsequent studies, such as those by (Karina Aranda-Rivera et al., 2021) and (Li et al., 2021), further elaborate on the relationship between mitochondrial redox signaling and oxidative stress, emphasizing how excessive production of reactive oxygen species (ROS) is a hallmark of both AKI and CKD. The high energy demands of renal cells, particularly during acute insults, highlight the importance of mitochondrial integrity in maintaining renal function, as discussed by (Inagi, 2022). The exploration of metabolic derangements associated with mitochondrial dysfunction, as presented by (Inagi, 2022), reveals how impaired mitochondrial biogenesis and an imbalance in mitochondrial dynamics can exacerbate kidney injury. The findings suggest that mitochondrial health is crucial for cellular metabolism and energy production, which is often compromised in kidney diseases. **Conclusion:** In conclusion, the literature illustrates a multifaceted understanding of mitochondrial dysfunction in kidney injury, emphasizing its implications for both acute and chronic conditions. The ongoing research highlights the potential for mitochondrial-targeted therapies as promising avenues for improving outcomes in renal diseases, thereby paving the way for innovative treatment strategies that address the underlying metabolic derangements associated with these conditions.

Keyword: Mitochondrial dysfunction, kidney injury

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INTRODUCTION

The literature surrounding mitochondrial dysfunction in kidney injury has gained significant attention over recent years, highlighting the pivotal role of mitochondria in the pathophysiology of both acute kidney injury (AKI) and chronic kidney disease (CKD). (A. Popkov et al., 2019) laid the groundwork by elucidating how mitochondria serve as both a target and a source of uremic toxins, emphasizing their involvement in the initial damaging stimuli that lead to renal cell death through the production of reactive oxygen and nitrogen species. This study underscores the dual role of mitochondrial dysfunction as a contributor to oxidative stress and as a mediator of subsequent kidney damage, setting the stage for understanding the mechanisms that underpin CKD development.

Building upon these insights, (Karina Aranda-Rivera et al., 2021) expanded the discussion to explore the relationship between mitochondrial redox signaling and oxidative stress in kidney diseases. They pointed out that excessive reactive oxygen species production leads to oxidative stress, which is a hallmark of both AKI and CKD. Their review emphasizes the importance of understanding redox-sensitive signaling pathways that regulate mitochondrial function, thereby providing a critical perspective on the metabolic dysregulation that occurs in renal pathologies.

(Li et al., 2021) further contributed to this discourse by focusing specifically on intrinsic AKI mechanisms and the role of mitochondrial dysfunction in tubular injury. Their analysis highlights the high energy demands of the kidney and how compromised mitochondrial oxidative phosphorylation can lead to energy deficits, exacerbating cellular injury in AKI. This work reinforces the notion that mitochondrial health is integral to renal function, particularly in the context of acute insults.

(Inagi, 2022) introduced the concept of organelle stress and metabolic derangement, detailing how mitochondrial dysfunction manifests as altered metabolism and

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energy production in kidney diseases. The review discusses the implications of impaired mitochondrial biogenesis and the imbalance between mitochondrial fusion and fission, which are critical for maintaining cellular health. This perspective is crucial for understanding how metabolic dysregulation contributes to the progression of kidney diseases.

(Ashkar et al., 2022) shifted the focus towards dietary interventions, specifically the role of polyphenols in mitigating mitochondrial dysfunction associated with kidney diseases. They highlighted the protective effects of polyphenols in maintaining mitochondrial function and reducing oxidative stress, suggesting that dietary strategies could serve as adjunct therapies in renal pathology management.

(Ho & Shirakawa, 2022) emphasized the therapeutic potential of targeting mitochondrial function to address oxidative stress-related renal injury. Their review synthesized evidence linking mitochondrial dysfunction to progressive kidney disease, advocating for interventions aimed at enhancing mitochondrial antioxidant defenses as a promising therapeutic strategy.

(Tanriover et al., 2023) underscored the interconnectedness of AKI and CKD, reiterating that mitochondrial dysfunction is a critical factor in the pathogenesis of these conditions. Their exploration of mitochondrial biogenesis and energy metabolism highlights the necessity for further research into therapeutic agents targeting mitochondrial processes to improve kidney health.

Finally, (Mao et al., 2024) provided a bibliometric analysis that reinforces the emerging consensus on the importance of mitochondria in kidney disease. Their findings suggest that targeting mitochondrial dysfunction could be a viable therapeutic approach, particularly in the context of oxidative stress and its role in renal pathology progression.

This body of literature collectively illustrates the multifaceted role of mitochondrial dysfunction in kidney injury, emphasizing its implications for both acute and chronic conditions. The ongoing research highlights the potential for

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mitochondrial-targeted therapies to improve outcomes in renal diseases, paving the way for innovative treatment strategies that address the underlying metabolic derangements associated with these conditions.

LITERATURE REVIEW

The article "Mitochondria as a Source and a Target for Uremic Toxins" by (A. Popkov et al., 2019) provides a comprehensive examination of the pivotal role that mitochondria play in acute kidney injury (AKI) and its progression to chronic kidney disease (CKD). The authors elucidate the multifaceted relationship between mitochondrial dysfunction and various pathophysiological conditions that precipitate kidney injury, including ischemic, infectious, and toxic insults.

One of the key insights presented in the article is the dual role of mitochondria as both a target and a source of injury in AKI. The authors detail how initial damaging stimuli can compromise mitochondrial structure and function, leading to the generation of pathological reactive oxygen and nitrogen species (ROS and RNS). This oxidative stress not only exacerbates cellular damage but also contributes to inflammatory processes and cell death, which are critical in the progression of kidney injury. The authors effectively highlight the cascade of events following mitochondrial membrane damage, including a drop in transmembrane potential and the opening of the mitochondrial permeability transition pore, which culminates in the release of pro-apoptotic factors.

Furthermore, the article emphasizes the connection between mitochondrial dysfunction and the emergence of CKD. The authors argue that oxidative stress-related mechanisms are central to the pathogenesis of CKD, linking it to various conditions such as diabetes, hypertension, and glomerulonephritis. This connection underscores the importance of addressing mitochondrial health in the management of kidney diseases. The authors propose that therapeutic strategies targeting mitochondria, such as the use of mitochondria-targeted antioxidants, may offer protective benefits against AKI and potentially reduce the risk of CKD development.

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Moreover, the article discusses the broader implications of mitochondrial dysfunction in relation to comorbid conditions, particularly cardiovascular diseases, which are prevalent in CKD patients. The authors assert that the interplay between oxidative stress, mitochondrial damage, and cardiovascular morbidity highlights the systemic nature of these pathologies and the need for integrated treatment approaches.

The article titled "Mitochondrial Redox Signaling and Oxidative Stress in Kidney Diseases" by (Karina Aranda-Rivera et al., 2021) provides a comprehensive examination of the intricate relationship between mitochondrial dysfunction, oxidative stress, and kidney diseases, specifically focusing on acute kidney injury (AKI) and chronic kidney disease (CKD). The authors highlight the significant economic burden posed by kidney diseases globally, categorizing them into AKI and CKD, with an emphasis on the rapid loss of renal function characteristic of AKI.

A critical evaluation of the material reveals that the authors effectively delineate the pathophysiological mechanisms underlying AKI, noting that it can arise from various factors such as chemotherapeutic agents, renal ischemia/reperfusion events, and environmental toxins. The high morbidity and mortality associated with AKI, coupled with its potential progression to CKD, underscores the urgency of understanding these mechanisms. The article aptly points out that renal fibrosis, a key feature of CKD, stems from inadequate repair processes following AKI, leading to irreversible damage and loss of renal function.

The role of mitochondria in kidney health is thoroughly discussed, with the authors emphasizing their involvement in crucial cellular functions, including energy metabolism and biosynthesis. Notably, the production of reactive oxygen species (ROS) during these processes is highlighted as a double-edged sword; while ROS are essential for various signaling pathways, their overproduction can lead to oxidative stress (OS). This oxidative stress is implicated in mitochondrial damage,

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cellular dysfunction, and ultimately cell death, marking it as a central player in the progression from AKI to CKD.

Despite the substantial insights provided, the authors acknowledge a gap in the understanding of redox signaling pathways in renal mitochondrial impairment. This area represents a critical avenue for future research, as elucidating these pathways could offer novel therapeutic targets for mitigating mitochondrial dysfunction and its consequences in kidney diseases.

The article "Targeting Mitochondria and Metabolism in Acute Kidney Injury" by (Li et al., 2021) provides a comprehensive overview of the role of mitochondrial dysfunction in the context of acute kidney injury (AKI). The authors effectively delineate the mechanisms underlying intrinsic AKI, emphasizing the critical factors of renal ischemia, nephrotoxins, and sepsis as primary etiological contributors.

One of the key insights of the article is the recognition of mitochondrial dysfunction as a common pathogenic mechanism across these diverse causes of AKI. The authors argue that this dysfunction is particularly significant given the kidney's high metabolic demands, which are predominantly met through mitochondrial oxidative phosphorylation (OXPHOS). The proximal tubule and the thick ascending limb are highlighted as regions of the kidney that are not only rich in mitochondria but also exhibit heightened vulnerability to injury during AKI. This correlation underscores the importance of mitochondrial health in maintaining renal function, particularly in metabolically active segments.

The review further discusses the alterations in energy metabolism that occur during AKI, suggesting that these changes may contribute to the progression of the condition. The authors note that while energy metabolism in the kidney has been studied under normal physiological conditions, there has been a relative scarcity of research focused on its pathophysiological alterations during AKI. This gap in the literature is addressed through a synthesis of current evidence linking mitochondrial dysfunction with metabolic reprogramming, thereby providing a clearer understanding of the pathophysiological landscape of AKI.

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In their critical evaluation, (Li et al., 2021) highlight the potential for targeting mitochondrial pathways as a therapeutic strategy in AKI management. They suggest that interventions aimed at preserving mitochondrial function or restoring metabolic balance could mitigate the severity of kidney injury and promote recovery. This perspective is particularly relevant given the urgent need for effective treatments in AKI, which remains a significant clinical challenge.

In the article "Organelle Stress and Metabolic Derangement in Kidney Disease," (Inagi, 2022) presents a comprehensive examination of mitochondrial dysfunction and its implications in kidney injury, particularly focusing on acute kidney injury (AKI) and chronic kidney disease (CKD). The article emphasizes the significant role of mitochondria in renal cells, particularly proximal tubular cells, which are characterized by their high mitochondrial content. This focus underscores the critical nature of mitochondrial health in maintaining kidney function and highlights how mitochondrial metabolism is dynamically altered in various kidney diseases.

Inagi elaborates on the mechanisms underlying mitochondrial dysfunction, particularly the balance between mitochondrial fusion and fission. The article posits that an imbalance in these processes can lead to cellular damage, with specific emphasis on Drp1, a protein implicated in mitochondrial fission. The deletion of Drp1 was shown to significantly reduce kidney fibrosis resulting from ischemia-reperfusion injury, suggesting that Drp1 plays a pathogenic role in tubular damage during AKI. This insight is particularly valuable as it provides a potential therapeutic target for ameliorating kidney injury.

Furthermore, the article discusses the suppression of PGC1α expression in AKI, which correlates with impaired mitochondrial function. This suppression contributes to a cascade of events leading to the pathophysiology of CKD, where a vicious cycle ensues, characterized by increased mitochondrial reactive oxygen species (ROS), decreased ATP production, and alterations in mitochondrial

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biogenesis. The interplay of these factors creates a detrimental environment that exacerbates kidney damage.

Inagi also addresses diabetic kidney disease (DKD), elucidating how hyperglycemia triggers mitochondrial fragmentation through the activation of ROCK1 in podocytes and endothelial cells. This finding is significant as it links metabolic stress to mitochondrial dysfunction, furthering our understanding of how metabolic derangement can propagate kidney injury. The abnormal elevation of TCA cycle metabolites in DKD mice, and the subsequent normalization of these metabolites upon treatment with SGLT2 inhibitors, highlights the potential for metabolic interventions in mitigating kidney damage.

The article concludes with a strong assertion regarding the importance of mitochondrial quality over quantity in maintaining mitochondrial metabolism. This perspective shifts the focus from merely increasing mitochondrial numbers to ensuring their functional integrity, which could lead to novel therapeutic strategies aimed at preserving mitochondrial function in kidney diseases.

The article titled "The Effect of Polyphenols on Kidney Disease: Targeting Mitochondria" by (Ashkar et al., 2022) provides a comprehensive examination of the critical role mitochondria play in kidney physiology and the implications of mitochondrial dysfunction in various kidney diseases. The authors emphasize that the kidneys are among the most energy-demanding organs in the body, receiving a substantial portion of cardiac output and consuming a significant amount of oxygen. This high energy requirement underscores the importance of mitochondrial health in maintaining renal function and homeostasis.

The authors delve into the connection between mitochondrial dysfunction and both acute and chronic kidney diseases, highlighting how conditions such as renal ischemia and toxicity are often accompanied by impaired mitochondrial biogenesis, oxidative phosphorylation (OXPHOS), and mitophagy. This dysfunction not only disrupts normal kidney function but also contributes to a cascade of pathological

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processes, including inflammation, apoptosis, and tissue injury, which are critical factors in the progression of kidney diseases.

A noteworthy contribution of this article is its exploration of polyphenols as potential therapeutic agents targeting mitochondrial dysfunction in renal diseases. Despite the growing body of research supporting the beneficial effects of bioactive compounds, the authors point out a significant gap in the literature regarding the specific impacts of polyphenols on mitochondrial health within the context of kidney injury. This highlights an important area for future research, as understanding the mechanisms through which polyphenols exert their effects could lead to novel therapeutic strategies for mitigating kidney injury and improving patient outcomes.

The article "Oxidative Stress and Mitochondrial Dysfunction in Chronic Kidney Disease" by (Ho & Shirakawa, 2022) provides a comprehensive overview of the critical role of mitochondrial function in renal health and disease, particularly focusing on chronic kidney disease (CKD). The authors effectively argue that the kidneys, being one of the most metabolically active organs, require a high mitochondrial density and oxygen consumption to perform essential functions such as waste removal, nutrient reabsorption, and blood pressure regulation. This foundational concept underscores the significance of mitochondrial integrity in maintaining renal function.

A key insight presented in the article is the relationship between oxidative stress and mitochondrial dysfunction. The authors elucidate how excessive reactive oxygen species (ROS) production, a byproduct of adenosine triphosphate (ATP) generation, can lead to cellular injury. This is particularly relevant in the context of kidney tissue, which is characterized by high mitochondrial content. The notion that mitochondrial ROS (mtROS) formation can initiate a cascade of ROS-induced ROS release highlights a critical feedback loop that exacerbates oxidative stress and cellular damage. Such dysregulation is linked to various pathologies, including cell death, which is pertinent to the progression of kidney diseases.

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The article further discusses the implications of mitochondrial dysfunction on ATP levels and ROS accumulation, establishing a clear connection to renal dysfunction. The authors emphasize that a decline in ATP production, coupled with an increase in ROS, contributes to the pathophysiology of CKD. This perspective is crucial for understanding the mechanisms underlying renal injury and the potential for therapeutic interventions.

Moreover, (Ho & Shirakawa, 2022) identify nephron loss, inflammation, and endothelial injury as contributing factors to the progression of acute kidney injury and CKD. This multifactorial approach to understanding kidney disease is commendable, as it considers both mitochondrial and non-mitochondrial pathways that may interact to influence renal health.

The authors advocate for a focus on mitochondrial-targeted therapies as a promising avenue for mitigating oxidative stress-related renal injury. By promoting ATP production and enhancing mitochondrial antioxidant defenses, there is potential to restore mitochondrial homeostasis and improve kidney function. This forward-looking perspective is particularly relevant in the context of developing novel treatment strategies for CKD.

The article titled "The Mitochondrion: A Promising Target for Kidney Disease" by (Tanriover et al., 2023) provides a comprehensive overview of the critical role that mitochondrial dysfunction plays in kidney injury, particularly in the context of acute kidney injury (AKI) and its relationship with chronic kidney disease (CKD). The authors effectively highlight the dual nature of these conditions, wherein AKI can exacerbate CKD, and vice versa, emphasizing the need for a deeper understanding of the underlying mechanisms, particularly those involving mitochondrial health.

One of the pivotal insights from the article is the recognition of the kidney's high energy demands, second only to the heart, due to its substantial mitochondrial density and oxygen consumption. This characteristic makes the kidney particularly vulnerable to mitochondrial dysfunction, which is implicated in various forms of

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kidney disease. The authors discuss several key areas of research, including mitochondrial biogenesis, the balance between mitochondrial fusion and fission, oxidative stress, and energy metabolism defects. These areas are crucial for understanding how mitochondrial dysfunction contributes to the pathogenesis of kidney diseases.

The article also addresses the therapeutic potential of targeting mitochondrial processes in kidney pathology. The classification of potential therapeutic agents into biogenesis activators, fission inhibitors, antioxidants, and mitophagy enhancers reflects a nuanced approach to treatment strategies. This classification is particularly noteworthy as it underscores the complexity of mitochondrial dynamics and their implications for kidney health. Furthermore, the mention of glucose-lowering drugs, such as SGLT-2 inhibitors and GLP-1 receptor agonists, as agents that influence mitochondrial processes adds a contemporary perspective on pharmacological interventions that may mitigate mitochondrial dysfunction in kidney injury.

However, while the article provides a robust overview of the mechanisms and potential therapeutic targets, it could benefit from a more detailed exploration of the clinical implications of these findings. For instance, the authors could elaborate on how these mitochondrial-targeted therapies might be integrated into existing treatment protocols for patients suffering from AKI or CKD. Additionally, a discussion on the challenges and limitations of current research in this field would provide a more balanced view of the potential and pitfalls of targeting mitochondrial dysfunction in kidney disease.

The article titled "The relationship between kidney disease and mitochondria: a bibliometric study" by (Mao et al., 2024) provides a comprehensive examination of the intricate relationship between mitochondrial dysfunction and kidney injury. The authors highlight the significance of extracellular vesicles (EVs) in preserving mitochondrial genetic integrity, particularly through the transfer of mitochondrial-related proteins such as TFAM. This process is crucial in preventing mitochondrial

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DNA (mtDNA) leakage and repairing mitochondrial function, which are essential for mitigating the progression of kidney diseases.

A critical insight from the article is the role of oxidative stress in kidney disease, particularly in the context of diabetes. The authors underscore that increased superoxide production is a primary factor in diabetic tissue damage, activating multiple pathways that contribute to microvascular and cardiovascular complications. This connection between oxidative stress and mitochondrial dysfunction is pivotal, as it delineates a pathway through which diabetes exacerbates kidney injury. The authors provide a solid theoretical framework for understanding how mitochondrial impairments can lead to renal dysfunction, particularly through disrupted tubular reabsorption processes.

Furthermore, the article emphasizes the diverse nature of kidney diseases, categorizing them into acute and chronic forms, as well as primary and secondary types. Regardless of classification, the authors assert that mitochondrial dysfunction is a common theme across these conditions, often culminating in renal failure. This assertion is supported by a growing body of literature that indicates varying degrees of mitochondrial damage in numerous renal pathologies.

The authors advocate for targeting mitochondria as a therapeutic strategy in treating kidney diseases, suggesting that interventions aimed at mitochondrial function could offer promising avenues for clinical application. They point to the increasing focus on mitochondrial studies within the realm of nephrology, underscoring the potential for mitochondrial-targeted therapies to revolutionize the management of kidney diseases.

CONCLUSION

The body of literature reviewed collectively underscores the critical role of mitochondrial dysfunction in kidney injury, particularly in the contexts of acute kidney injury (AKI) and chronic kidney disease (CKD). The foundational work by (A. Popkov et al., 2019) establishes that mitochondria are not only targets but also

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sources of uremic toxins, contributing to oxidative stress and subsequent renal cell death. This dual role is pivotal in understanding the mechanisms that lead to kidney damage.

Subsequent studies, such as those by (Karina Aranda-Rivera et al., 2021) and (Li et al., 2021), further elaborate on the relationship between mitochondrial redox signaling and oxidative stress, emphasizing how excessive production of reactive oxygen species (ROS) is a hallmark of both AKI and CKD. The high energy demands of renal cells, particularly during acute insults, highlight the importance of mitochondrial integrity in maintaining renal function, as discussed by (Inagi, 2022).

The exploration of metabolic derangements associated with mitochondrial dysfunction, as presented by (Inagi, 2022), reveals how impaired mitochondrial biogenesis and an imbalance in mitochondrial dynamics can exacerbate kidney injury. The findings suggest that mitochondrial health is crucial for cellular metabolism and energy production, which is often compromised in kidney diseases.

Moreover, the potential for dietary interventions, particularly the role of polyphenols in mitigating mitochondrial dysfunction, is highlighted by (Ashkar et al., 2022). This perspective introduces an adjunctive therapeutic strategy that could complement existing treatments for renal pathologies.

The synthesis of evidence by (Ho & Shirakawa, 2022) emphasizes the therapeutic potential of targeting mitochondrial function to combat oxidative stress-related renal injury. The interconnectedness of AKI and CKD, as discussed by (Tanriover et al., 2023), further reinforces the need for comprehensive strategies that address mitochondrial dysfunction to improve kidney health.

Finally, the bibliometric analysis by (Mao et al., 2024) supports the emerging consensus on the significance of mitochondria in kidney disease, advocating for targeted therapeutic approaches that could revolutionize treatment paradigms.

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In conclusion, the literature illustrates a multifaceted understanding of mitochondrial dysfunction in kidney injury, emphasizing its implications for both acute and chronic conditions. The ongoing research highlights the potential for mitochondrial-targeted therapies as promising avenues for improving outcomes in renal diseases, thereby paving the way for innovative treatment strategies that address the underlying metabolic derangements associated with these conditions.

DISCLOSURE STATEMENT

- Disclosure Statement : The authors have no conflicts of Interest to declare
- Funding Sources : None
- Acknowledgements : -
- Author Contribution : All authors discussed and contributed the final content for journal submission and publication

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