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Dengue fever-associated glomerulonephritis; an updated narrative mini-review

Abdul Amir H. Kadhum[1*], Aliaa Saadoon Abdul-Razaq Al-Faraji[2], Sudad Jasim Mohammed[2], Adil Turki Al-Musawi[2], Maytham Ahmed AbdulAemah[3], Qais R. Lahhob[4]

[1]Department of Chemistry, College of Medicine, University of Al-Ameed, Karbala, Iraq
[2]Market Research and Consumer Protection Center, University of Baghdad, Iraq
[3]College of Pharmacy, Al-Mustaqbal University, Babylon, Hillah, 51001, Iraq
[4]Collage of Pharmacy, National University of Science and Technology, Dhi Qar, 64001, Iraq

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ABSTRACT

Introduction: Dengue fever-associated acute kidney injury (AKI) has multifactorial nature. These glomerular changes are likely mediated by the host immune response to dengue virus infection, particularly the deposition of immune complexes and the release of inflammatory cytokines like IL-17. The glomerular injury can lead to proteinuria, hematuria, and even acute glomerulonephritis. Dengue fever-associated glomerulonephritis is diagnosed through a combination of clinical evaluation, laboratory tests, renal biopsy, imaging studies, and serological tests. Laboratory tests revealed proteinuria, hematuria, and granular casts. Additionally, elevated levels of inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate are often detected. Glomerular abnormalities in kidney biopsy consisted of mesangial proliferation, endothelial swelling, thrombotic microangiopathy, inflammatory cell infiltration and immune complex deposition. Treatment of dengue-associated glomerulonephritis is primarily supportive, with management of fluid and electrolyte balance, blood pressure control, and dialysis if necessary. In some cases, immunosuppressive therapy may be required.

Mini-Review

Implication for health policy/practice/research/medical education:
Dengue fever-associated glomerulonephritis often presents with a combination of symptoms such as fever, headache, retro-orbital pain, myalgia, and arthralgia. Dengue fever-associated glomerulonephritis is caused by a combination of direct viral injury, immune complex deposition, and hemodynamic factors. Dengue virus can productively infect up to 80% of primary human endothelial cells, including glomerular endothelial cells, resulting in the rapid release of infectious virions. Dengue virus infection leads to endothelial cell dysfunction and weakening of the endothelial barrier integrity in the glomeruli. Renal biopsy in this disease is typically characterized by mesangial proliferation and immune complex deposition.

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Introduction

Acute kidney injury (AKI) is a serious complication of dengue virus infection, with an incidence ranging from 0.83–14.40% and a mortality rate of 11.30%–60.00% (1). Acute kidney injury is accompanying by increased length of hospital stay and fatal cases of dengue (2). Renal manifestations of dengue also include proteinuria, glomerulonephritis, hemolytic uremic syndrome, acute tubular necrosis and nephrotic syndrome (3). Laboratory tests revealed proteinuria, hematuria, and granular casts. Additionally, elevated levels of inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate are often detected (4). Independent risk factors for developing AKI in dengue cases are male gender, obesity, advanced age, hemorrhagic fever, diabetes mellitus, rhabdomyolysis, multiple organ dysfunction, concomitant bacterial infection, delay in hospital consultation and use of nephrotoxic agents (5). Dengue fever-associated glomerulonephritis is more common in tropical and subtropical regions where dengue is prevalent, whereas

***Corresponding author**: Abdul Amir H. Kadhum, Email: Amir1719@gmail.com

other types of glomerulonephritis may be more common in different regions or populations (2,3,5). Dengue fever-associated glomerulonephritis often presents with a combination of symptoms such as fever, headache, retro-orbital pain, myalgia, and arthralgia (2,3,5). The prognosis of dengue-associated glomerulonephritis is generally good, with most patients recovering fully; however, patients with pre-existing chronic kidney disease are at higher risk (3). In some cases, this condition can progress to end-stage renal disease, particularly if left untreated or if there are underlying comorbidities (2,3,5). Previous studies shown the risk of developing chronic renal failure is significantly increased in patients who survive AKI associated with dengue infection; since, AKI caused by dengue can lead to long-term kidney damage and scarring, which can progress to chronic kidney disease (CKD) over time (5,7). This increased risk is due to the multifactorial nature of AKI in dengue, which involves direct viral damage to the kidneys, hemodynamic instability, rhabdomyolysis, and other factors (5-7). In addition, patients who develop severe dengue and AKI are at higher risk of developing CKD due to the severity of the illness and the prolonged duration of hospitalization, which can lead to increased exposure to nephrotoxic medications and other risk factors for CKD (7,8).

Search strategy

For this review, we conducted a search across several databases, including PubMed, Web of Science, EBSCO, Scopus, Google Scholar, the Directory of Open Access Journals (DOAJ), and Embase. We utilized various keywords such as "Dengue fever," "glomerulonephritis," "acute kidney injury," "immune complex," "chronic kidney disease," "hemolytic uremic syndrome," "tubular injury," "cytokine," and "glomerular filtration rate."

Alteration of glomerular filtration rate in dengue fever-associated glomerulonephritis

The virus can cause glomerular injury, leading to a decrease in the area of the Bowman's space and compression of glomerular capillaries. This disorder can reduce filtration rates and facilitate the entrapment of immune complexes (1,2,9). The virus also can cause an expansion of the mesangial matrix, which can lead to a decrease in the area of the Bowman's space and compression of glomerular capillaries, further reducing filtration rates (9). Meanwhile, the virus can trigger the deposition of immune complexes in the glomeruli, which can cause inflammation and damage to the glomerular capillaries, leading to a decrease in filtration rates (5,9,10). Moreover, the glomerular inflammation can cause hemodynamic alterations, such as increased vascular permeability and reduced blood flow to the kidneys, which can lead to a decrease in filtration rates (9,10). Accordingly, the virus can cause tubular injury, leading to a decrease in the reabsorption of solutes and water, which can further reduce filtration rates. These mechanisms can result in a decrease in the, which can lead to AKI and potentially CKD (3,5).

Mechanistic impact of dengue-associated AKI

Role of cytokines

Dengue infection triggers a cytokine storm, which is described by the release of pro-inflammatory cytokines like tumor necrosis factor alpha (TNF-α), interleukin 6 (IL-6), IL-8, and IL-1β. This cytokine storm contributes to the development of AKI by causing inflammation and tissue damage in the kidneys (11). Interleukin 6 and IL-8 are two key cytokines involved in the pathogenesis of AKI in dengue patients. Elevated levels of these cytokines have been associated with the development of AKI and are potential biomarkers for predicting AKI in dengue patients (11,12). Interleukin 20 is another cytokine that plays a role in the development of AKI in dengue patients. It is a pro-inflammatory mediator that regulates cytokine expression and contributes to the progression of renal diseases (11-13). Transforming growth factor beta (TGF-β) is also a cytokine that is involved in the development of fibrosis and inflammation in the kidneys. Elevated levels of TGF-β have been associated with the development of AKI in dengue patients (14,15). Additionally, cytokines such as TNF-α, IL-6, and IL-8 mediate inflammation in the kidneys, leading to tissue damage and AKI. This inflammation is characterized by the infiltration of inflammatory cells such as neutrophils, lymphocytes, and macrophages into the kidneys (3,5). Furthermore, cytokines such as TNF-α and IL-6 can cause vascular injury by activating the production of vasoconstrictors and increasing the permeability of blood vessels; since, vascular injury can contribute to the development of AKI in dengue patients (16, 17). Likewise, IL-17 have been associated with immune-mediated glomerular diseases and plays a role in the development of glomerular injury in dengue (5,18).

Role of plasma leakage syndrome

Plasma leakage syndrome, a hallmark of severe dengue infection, significantly contributes to the development of AKI in several ways. Plasma leakage leads to a rapid loss of intravascular fluid, resulting in hypovolemia, hypotension, and tachycardia (19,20). This hemodynamic instability can cause a reduction in renal perfusion, leading to AKI. The loss of plasma volume can cause a decrease in blood pressure, which can further exacerbate AKI by reducing renal perfusion and increasing the risk of acute tubular necrosis (5). Plasma leakage can also lead to electrolyte imbalances, such as hyponatremia, which can contribute to the development of AKI. Hyponatremia in dengue patients is often caused by plasma leakage and can result in intravascular volume depletion, especially in severe dengue cases (5,21). Plasma leakage can also lead to rhabdomyolysis and hemolysis, which can cause AKI

through the release of myoglobin and hemoglobin into the bloodstream, respectively. These substances can cause tubular obstruction and damage, contributing to AKI (5,21,22).

Other mechanisms

Rhabdomyolysis is also due to muscle injury by virus invasion, leads to AKI through multiple mechanisms, including fluid sequestration into damaged muscles, intravascular volume depletion, and the release of myoglobin, which can cause tubular obstruction and damage as mentioned above (22,23). In some cases, hemolytic uremic syndrome can be caused by the dengue virus (24). In addition, direct cytopathic effect of the viral protein on glomerular and tubular cells, potentially leads to acute tubular necrosis and glomerulopathy (3).

Endothelial barrier dysfunction in dengue fever-associated glomerulonephritis

Dengue virus can productively infect endothelial cells directly, leading to the release of infectious virions and potentially altering endothelial barrier function (25). Furthermore, type I interferon (IFN) plays a central role in modulating endothelial barrier function (26,27). Dengue virus suppresses TNF-α–mediated hyperpermeability too (27). In addition, immune complex deposition in the glomeruli, particularly IgG, IgM, and C3 deposits, can contribute to endothelial barrier dysfunction (28,29). Moreover, proinflammatory cytokines, such as TNF-α, can decrease endothelial barrier function and increase vascular permeability. Elevated levels of these cytokines are present in patients with dengue hemorrhagic fever (30). Notably, dengue virus infection can promote endothelial-to-mesenchymal-like changes, which can lead to the loss of endothelial barrier function and increased permeability (31). Finally, Dengue virus specifically attaches to heparan sulfate-containing proteoglycan receptors on endothelial cells, which can facilitate infection and potentially alter endothelial barrier function (25).

Dengue fever-associated glomerulonephritis

Patients with dengue fever-associated glomerulonephritis typically present with symptoms such as fever, headache, retro-orbital pain, myalgia, and arthralgia, along with signs of kidney dysfunction such as proteinuria, hematuria, and edema (3). The pathophysiology of this disease is mainly due to immune complex deposition and mesangial proliferation (32,33). Dengue infection has been associated with various types of glomerulonephritis, including membranoproliferative glomerulonephritis (MPGN), mesangial proliferative glomerulonephritis, and rapidly progressive glomerulonephritis (33, 34). Diagnosis is typically made by kidney biopsy, which shows characteristic histological features such as mesangial proliferation and immune complex deposition (3).

Renal biopsy in dengue-associated glomerulonephritis

Dengue-induced glomerular injury is a complex condition involving various mechanisms, including direct viral injury, immune complex deposition, and hemodynamic factors (5). Renal biopsies have shown diffuse proliferative glomerulonephritis. An increase in glomerular volume is also observed in dengue-induced glomerular damage, which is associated with diffuse glomerular hypercellularity and immune complex deposition (5,8,35). Mesangial proliferation with mesangial IgA-dominant immune complex deposits has been observed in some cases (3,5,35,36). Additionally, endothelial swelling across with endocapillary hypercellularity is another histopathological feature of dengue-induced glomerular damage, indicating inflammation in the glomerular capillaries (37,38). In contrast, glomerular atrophy, characterized by a decrease in glomerular count and area, has been observed in dengue-infected kidneys. Dengue-induced glomerular atrophy is a complication that can occur in severe cases of dengue infection (5,35,39). Previous studies provide insights into the mechanisms and outcomes. The initial damage to the glomeruli can occur due to the expansion of the mesangial matrix, leading to a decrease in the area of the Bowman's space and compression of the glomerular capillaries. This condition can result in decreased filtration rates and facilitate the entrapment of immune complexes (6,40). Hemorrhage and edema are common histopathological changes observed in dengue-induced kidney injury, indicating vascular leakage and damage to the glomerular capillaries (3,41). Necrotic areas in the glomeruli and tubules are also seen in dengue-infected kidneys, which can lead to loss of glomerular function and tubular damage (3,41). In addition, IgG, IgM, and C3 deposits have been found in the glomeruli, basement membrane thickening, and hyperplasia of renal mesangial cells in areas with immune complex deposits (3,42). Thrombotic microangiopathy has been reported in some cases, characterized by glomerular and arteriolar microthrombi (24,43). Crescentic glomerulonephritis has been diagnosed in some cases, often associated with anti-glomerular basement membrane and perinuclear anti-neutrophil cytoplasmic antibodies (44,45). Likewise, a lymphomononuclear infiltrate, neutrophils, and nuclear fragmentation have been observed in the glomeruli of fatal cases of this disease (46). in addition, monocyte-like cells have been observed infiltrating the glomeruli in fatal dengue hemorrhagic fever cases (37,46).

Reversibility of glomerular changes

Reversibility of glomerular changes has been observed in some cases, with repeated biopsies showing resolution of glomerular injury after clinical recovery from dengue and AKI (1,3). However, dengue hemorrhagic fever, is associated with more severe glomerular damage, including mesangial proliferation and immune complex deposition, along with persistent glomerular changes, particularly

proteinuria (3,44-48). Proteinuria is more frequent and severe, often reaching nephrotic range, which is seen in up to 74% of patients with dengue hemorrhagic fever (44-50).

Treatment

Careful fluid management is crucial to prevent volume overload and pulmonary edema, particularly in patients with pre-existing CKD or heart failure (51). Renal replacement therapy in the form of sustained low-efficiency dialysis or continuous renal replacement therapy may be required during the critical phase of dengue to manage fluid overload and electrolyte imbalances (52). Supportive care, including management of bleeding, shock, and organ dysfunction, is the mainstay of treatment for severe dengue (53). In rare cases of severe glomerular injury, such as crescentic glomerulonephritis, immunosuppressive therapy may be considered, but its efficacy in dengue-induced glomerular damage is not well-established (3,44). Close monitoring of renal function, fluid balance, and electrolytes is essential in managing dengue patients with glomerular injury (5).

Conclusion

The glomerular changes in dengue-associated glomerulonephritis are indicative of a complex interplay between the host immune response and the dengue virus, leading to the development of thrombotic microangiopathy and glomerular damage.

Authors' contribution

Conceptualization: Abdul Amir H. Kadhum.
Data curation: All authors.
Investigation: Qais R. Lahhob.
Resources: Maytham Ahmed AbdulAemah.
Supervision: All authors.
Validation: Aliaa Saadoon Abdul- Razaq Al-Faraji.
Visualization: All authors.
Writing—original draft: All authors.
Writing—review & editing: 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 work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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