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Dengue with Glomerulonephritis-Case Study

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Abstract: Dengue is viral infection, not only threat to SriLanka but also threat to the world. It belongs to family Flaviviridae and it responsible for nearly 100 million cases of dengue fever. It affects almost all the systems in our body including, heart, brain, liver, blood and kidney. Dengue fever, affects kidney in various manner, it ranges from glomerulonephritis to acute kidney injuries. It is understandable that, renal involvement may occur in dengue hemorrhagic fever in the absence of shock, sepsis, haemolysis, or rhabdomyolysis. However, acute glomerulonephritis is a rare complication encountered in our clinical practice. Even though various pathological mechanisms play for the renal involvement, but mesangial proliferation and immune complex deposition are the dominant histologic features of dengue-associated glomerulonephritis. The presence of more than 25% urine dysmorphic red cell is specific but not sensitive for glomerulonephritis. This case study elaborates the possible pathogenesis of the glomerulonephritis due to dengue fever
Keywords: Dengue with glomerulonephritis and dengue with renal involvement.

INTRODUCTION

Dengue fever has appeared as a worldwide problem, it is due to family *Flaviviridae* RNA virus, transmitted by a mosquito bite. According to the World health organization(WHO) report, it is endemic in many countries such as Africa, America, Eastern Mediterranean, South-East Asia and the Western Pacific[1].

Also it stated that Western Pacific and South-East Asia regions are mostly affected by this protracted dengue. It is commonly presented as uncomplicated Dengue Fever (DF), Dengue Hemorrhagic fever (DHF) or Dengue Shock syndrome (DSS) [2]. Interestingly, it is produced by four serotype of dengue virus[3]. During the dengue endemic, wide range of complications were reported including encephalitis[4], hepatitis[5], acute coronary syndrome[6], appendicitis[5], epididymo-orchitis, Parotitis[7] and acute kidney injury[8]. Dengue with renal involvement had shown high incidence of haemoconcentration, thrombocytopenia, and raised liver enzymes [9]. However, available literature stated that wide spectrum of renal involvement ranging from glomerulonephritis to Acute renal failure[10].

However, acute glomerulonephritis is a rare complication encountered in our clinical practice. Several types of glomerulonephritis have been reported during dengue outbreak[11]. Mesangial proliferation and immune complex deposition are the dominant histologic features of dengue-associated glomerulonephritis. Post infectious glomerulonephritis (PIGN) may occur in association with bacterial, viral, fungal, protozoal, and helminthic infections. Viral-induced GN manifests in significantly different histologic forms of glomerular injury, depending on the duration of viral activity.

CASE HISTORY

Previously healthy, 42-year-old female was brought to our hospital with complaints of fever with headache, joint pain and body ache for six days duration. Second days of the fever, she noticed that her face become puffy and swelling of the whole body. She also complained of decreased urine output. There was no history of hematuria, burning micturition, rash, loose stool, vomiting, sore throat, icterus, or bleeding from any site. There was no history of skin sepsis and sore throat. There was no history of hypertension. On examination, she was conscious, febrile (39.4°C), heart rate 98/min, respiratory rate 18/min, blood pressure was 140/110 mmHg, facial puffiness, bilateral pitting ankle edema, decreased breath sound bilaterally in basal areas of lungs and shifting dullness in abdomen was present. There was no icterus or cyanosis. Laboratory tests revealed hemoglobin 11.1 gm/dl, total leukocyte count 8800/cmm, differential leukocyte count-polymorphs 35%, lymphocytes 60%, monocytes 2%, eosinophils 3% and platelet count 1,00,000/cmm. Peripheral smear showed no evidence of haemolysis. Malaria parasite was negative. blood urea 21 mg/L, serum creatinine 1.1 mg/dL, serum Na+ 132 MEq/L, serum K+ 5.2 MEq/L, total bilirubin 0.3 mg/dl, SGOT 39 IU/L, SGPT 28

IU/L, alkaline phosphatase 296 IU/L, serum albumin 3.4 g/dL, and serum cholesterol 108 mg/dL. Urine analysis showed microscopic hematuria and proteinuria, red cell cast and granular cast. Urinary dysmorphic red cells were more than 25%> Urine test for myoglobin was negative. C-reactive protein (CRP) was negative. An abdominal ultrasonography showed normal size kidney with mild ascites. Chest X-ray showed bilateral plural effusion. Echocardiography and renal Doppler were normal. Urine and blood culture were sterile. Serology [immunoglobulin (Ig)M and IgG enzyme-linked immunosorbent essay] against dengue virus was positive. Coagulation profile was normal. Patient was treated conservatively with maintenance fluid, and supportive care. She was gradually recovered with improving urine output and renal functions and discharged 12 days after admission.

DISCUSSION

Various types of glomerulonephritis have been reported during or shortly after dengue infection in humans and mouse models of dengue infection. In our patient's urine report exposed dysmorphic red cells (dRBCs) and granular cast. Dysmorphic red blood cells on urine microscopy have been associated with glomerulonephritis[12]. Furthermore, her clinical picture of generalized body swelling and reduced urine output, clearly highlight the features of acute glomerular nephritis. The mechanism of glomerulonephritis in an adult with dengue hemorrhagic fever remains unclear. Various mechanisms are implicated in the pathogenesis of glomerulonephritis associated with viral infections[13]. The common cause of acute glomerulonephritis is a post-streptococcal infection. However, we excluded in the absence of a recent sore throat and skin sepsis and negative ASOT titer,

Several possible pathogenic events occur in viral diseases associated with glomerular injury, including the following: Formation of circulating immune complexes involving viral antigens and antibodies, formation of circulating immune complexes induced by the release of antigens following virally induced cellular injury, formation of in situ antigen-antibody reactions or cell-mediated injury and autoimmune reactions to glomerular structures induced by the virus[14]. host antiviral antibodies and injury due to various inflammatory mediators released in response to glomerular or tubular cytopathic effects[15]. It has been assumed that dengue virus infection produces an immune response to viral antigens and results in immune-complex deposit in the glomerulus. Immune complex deposits in immune complex mediated glomerulonephritis consist predominantly of IgG, IgM, and C3 and deposit in the mesangial in a coarse granular pattern. It is not clear whether the virus causes direct damage by an invasion of the kidneys. However, recent studies using immune-histochemistry techniques have established the presence of viral antigens in kidney tubules of patients with serologically or virologically confirmed dengue infections [16]. Furthermore, Lizarraga and Nayer proposed that dengue infection is associated with systemic autoimmune disorders, which on rare occasions might involve the kidneys[17]. However, there is a recent report that dengue viral infection can also aggravate other immune complex diseases such as systemic lupus erythematosus[18].

CONCLUSION

Wide range of kidney involvements has been reported following dengue fever. However, dengue causing acute glomerulonephritis is rare phenomenon. Presence of urinary sediment with significant percentage of urinary dysmorphic red cells more suggestive to make diagnosis. Early diagnosis is mandatory, otherwise may end up with acute kidney injuries.

Ethical approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the parents for publication of this case report

Availability of data and material

All data gathered during this study are included in this published article.

Competing interests

The authors declare that they have no competing interests.

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