Rahman S[1,*], Wazib A[1], Bahar T[2]

[1]Department of Medicine, Enam Medical College and Hospital, Savar, Bangladesh

[2]National Institute of Cancer Research and Hospital, Dhaka, Bangladesh

Received 28 July 2020
Revised 08 Dec 2020
Accepted 20 Dec 2020
Published Online 31 Dec 2020

*Corresponding author:
Shaila Rahman.
E-mail: shailars43@gmail.com

Atypical Manifestations of Dengue Fever: A Narrative Review

***Abstract*— Dengue fever (DF) is an arboviral infection caused by one of four serotypes of the dengue virus. The worldwide burden of DF is high with a low fatality rate. It is endemic in tropical and sub-tropical regions. It is transmitted by mosquito bites. After a certain incubation period, it produces typical symptoms of fever, body ache, and headache with low blood pressure and thrombocytopenia. However, there are several atypical manifestations of DF such as acalculous cholecystitis, hepatitis, fulminant hepatic failure, acute pancreatitis, febrile diarrhea, encephalopathy, seizure, meningitis, encephalitis, acute motor weakness, myocarditis, acute respiratory distress syndrome (ARDS), acute kidney injury (AKI) and myositis, which are often overlooked leading to delayed diagnosis and high morbidity and mortality. This narrative review has compiled different atypical manifestations of DF. This article would be beneficial for clinicians to diagnose DF with atypical presentations early and save many lives by timely interventions.**

***Keywords* — AKI, ARDS, atypical manifestations, Dengue fever, Myocarditis**

1 INTRODUCTION

Dengue fever (DF) is the most common arboviral disease transmitted globally. The World Health Organization (WHO) ranked dengue as one of the top ten threats to global health in 2019 {1}. Asia represents about 70% of the global burden of disease. In certain countries, an increase in dengue cases is observed. The countries are Bangladesh, Nepal, Sri Lanka, Thailand, parts of India, Pakistan, and Sudan. The dengue virus is an RNA virus and is a member of the flavivirus group. DF is caused by four antigenically distinct dengue virus serotypes (DENV1, DENV 2, DENV 3, and DENV 4). Infection with one serotype confers lifelong immunity against that particular serotype and a brief period of partial immunity for two years against others. The infection is transmitted by infected female Aedes aegypti mosquitoes, and occasionally by Aedes albopictus [2,8].

DF has an incubation period of 4-7 days while viral replication takes place in target dendritic cells. Infection of the dendritic cells (DC), macrophages, hepatocytes, and endothelial cells, results in the production of cytokines that alter the immune response to the initial and subsequent virus infections [3-5].

The chances of severe dengue increases, if a person gets a second dengue infection with preexisting actively or passively (maternally) acquired immunity to a heterologous dengue virus serotype. Antibody-dependent enhancement (ADE) of viral replication is key to its pathogenesis [6,7].

Initial dengue viral infection may be asymptomatic in 50-90% of cases or may have undifferentiated febrile illness lasting for 5-7 days [8]. In 2009 WHO has classified DF as dengue with/ without warning signs, and severe dengue. Dengue shock syndrome (DSS), severe haemorrhage, and severe organ involvement are included in severe dengue. In 2012 WHO incorporated the new term expanded dengue syndrome (EDS) to describe the involvement of various organs in DF [2]. Patients of DF may come with the features of flu-like illnesses such as the rapid onset of high fever, myalgia (break-bone fever), headache, sore throat, and retroorbital pain, vomiting, and a centrifugal maculopapular rash, leukopenia, and lymphopenia [2,8].

The diagnostic tests for dengue are non-structural-1 protein (NS1) antigen, by virus-specific immunoglobulin M (Anti-Dengue IgM) and reverse transcriptase-polymerase chain reaction (RT-PCR) [2,8].

Besides the classical presentations of DF, there have been many atypical manifestations that may lead to a diagnostic dilemma and are often overlooked.

This narrative review has focused mostly on various atypical manifestations, pathogenesis, diagnostic approach, and treatment of such cases. Different search engines like PubMed

central and Google scholar, and google was searched from 1991 to 2019 for original and review articles, case reports and letter to the editor using the keywords DF, DHF, severe dengue, and atypical manifestations. Inclusion criteria included articles on epidemiological and clinical characteristics and pathogenesis of dengue and the atypical manifestations. Full downloadable pdf articles in English are chosen. Articles other than the English language and unpublished local data are excluded [Figure 1].

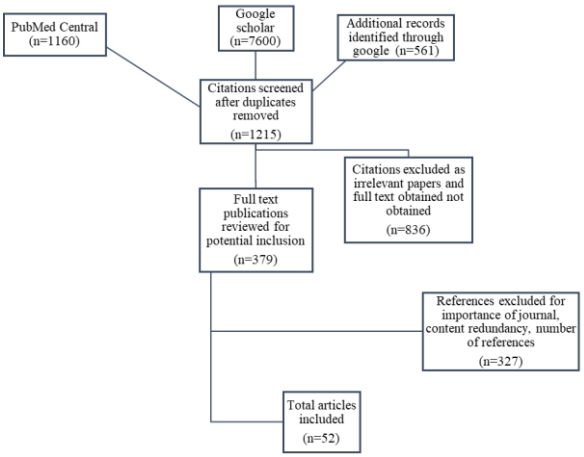

Figure 1: Flow chart summarizing the literature selection process.

2 ATYPICAL MANIFESTATIONS OF DF

The endothelium is the target for the immunopathological process in dengue. The central features are vascular permeability and coagulation disorders. These mechanisms can explain different systemic involvement [9].

3 HEPATOBILIARY AND GASTROINTESTINAL MANIFESTATIONS

Patients of DF may present with hepatitis, fulminant hepatic failure, acalculous cholecystitis, acute pancreatitis, febrile diarrhoea, and acute parotitis [10]. Hepatitis presents as right hypochondriac pain, tender hepatomegaly, and raised aminotransferases peaking after the first week of illness and normalizing in three weeks [9,10]. Jaundice is not evident in most of the cases and patients with hepatitis have a longer recovery time. The direct cytopathic effect of the dengue virus on liver cells has been proven in previous studies [11]. Fulminant hepatic failure is usually secondary to shock and DIC in severe dengue fever. The incidence of acute acalculous cholecystitis is about 7.63% [13]. Acalculous cholecystitis presents with right upper quadrant abdominal pain with radiation to the right shoulder and positive Murphy's sign. Ultrasound of the abdomen reveals gall bladder mucosal edema and pericholecystic fluid without any gallstones secondary to vascular leakage of plasma [12-15]. The exact cause of it is unknown, although it could be the increased vascular permeability [12], cholestasis, increased bile viscosity and infection which are responsible [15]. DF related acalculous cholecystitis resolves spontaneously with supportive care in the majority of the cases. However, there are case reports of gangrene and perforation of the gall bladder with peritonitis requiring surgical intervention [10,12,13]. Acute bilateral parotitis is reported by the isolation of DENV in saliva [16].

Acute pancreatitis is a rare complication of DF. The exact mechanism is unknown; however, it could be the direct cytopathic effect of the virus or an autoimmune response by molecular mimicry. It causes pancreatic outflow obstruction by the resulting edema [17]. An ultrasound abdomen shows an enlarged pancreas. Serum amylase and lipase levels are elevated [17,18]. There are few case reports of febrile diarrhea in dengue patients [9].

4 NEUROLOGICAL MANIFESTATIONS

The neurological manifestations in DF are variable. The mechanisms of neurological complications and the contribution of viral and host factors are yet to be understood. These can be related to the neurotrophic effect of the virus, systemic effects of the infection and can be immune-mediated. There have been different features like meningitis, encephalitis, encephalopathy, and myelitis because of the neurotrophic nature of the virus [19-23]. These are less common in DHF. The presence of DENV in cerebrospinal fluid (CSF) suggests the direct invasion of the central nervous system (CNS) by the virus [24]. Meningitis presents as neck rigidity and positive Kernig's sign. Myelitis is thought to be the result of gray matter involvement like poliomyelitis [23]. Encephalopathy, stroke, and hypokalaemic periodic paralysis have also been reported caused by the systemic complications of dengue infection [19,25,26]. Encephalopathy presents with a reduced level of consciousness, amnesia, restlessness, and is caused by acute liver failure, acute kidney injury, hyponatraemia, cerebral hypoxia, and cerebral oedema. Both haemorrhagic and ischaemic stroke can occur in

dengue infection mostly related to DHF. Haemorrhagic stroke is due to thrombocytopenia, platelet dysfunction and vasculopathy [27], whereas ischaemic stroke is caused by disseminated intravascular coagulation (DIC), vasculopathy and increased plasminogen activator inhibitor type I (PAI-I) which is a procoagulant [27,28]. The probable mechanism of hypokalemia could be either due to redistribution of potassium in cells or transient renal tubular abnormalities leading to increased urinary potassium wasting and it responded well to potassium replacement [26]. Other features like Guillain-Barre syndrome (GBS) [22], acute disseminated encephalomyelitis (ADEM) [29], acute transverse myelitis {30}, optic neuritis [31], phrenic neuropathy [32], oculomotor neuropathy [33], maculopathy [34] and fatigue syndrome {35} has been described as post-infection sequelae. GBS occurs due to antigenic mimicry to DENV. ADEM can be diagnosed by MRI of the brain with haemorrhage within areas of demyelination [29]. Most of the patients improved with supportive care even though few deaths have been reported.

5 CARDIOVASCULAR MANIFESTATIONS

The atypical cardiovascular manifestations of dengue are not that common. Different types of arrhythmias such as sinus bradycardia, junctional bradycardia, atrioventricular (AV) nodal block, atrial fibrillation, ventricular arrhythmias, myocarditis, and pericardial effusion have been reported [36,37]. Myocarditis ranging from mild to severe ventricular wall dysfunction presents as dyspnoea, chest pain, palpitation as the result of arrhythmias. Like many viral infections, DENV can cause myocardial injury, either by direct invasion or by autoimmune reaction resulting in myocardial inflammation. By direct invasion, it can incite different cytokines and can release inflammatory mediators like TNF-α, interleukins, oxygen-free radicals. DENV antigen may trigger a cell-mediated immune response causing myocardial injury, which recovers with the resolution of infection [37]. The echocardiogram is an effective tool to see myocardial dysfunction. Serum troponin I, CK-MB are also raised transiently. Cardiac complications are usually self-limiting occasionally needing specific drugs, correction of the underlying electrolyte imbalance, and temporary pacemaker until the resolution of arrhythmias [38].

6 RESPIRATORY MANIFESTATIONS

Lung complications are devastating. Acute lung injury (ALI) and Acute respiratory distress syndrome (ARDS) are the most serious ones which may cause fatality and occurs mostly in DSS. These are thought to be due to increased permeability of the alveolar capillaries resulting in alveolar and interstitial oedema which lead to respiratory failure requiring mechanical ventilation [39]. Cautious fluid management is crucial in the very beginning to prevent DSS thus preventing these complications as well. Pulmonary oedema may occur due to disturbance in colloid oncotic pressure aggravated by fluid overload by platelet transfusion and intravenous infusion. It may also be caused by myocarditis. Pneumonia, pleural effusion, and pulmonary haemorrhage are less common and are usually self-limiting. DF can be complicated by superimposed pneumonia with Staphylococcus aureus and melioidosis [40,41]. Identification of the pathogen and treatment with proper antibiotics is required. Massive pleural effusion requires aspiration [42].

7 RENAL MANIFESTATIONS

The renal manifestations of DF are varied. It may range from proteinuria, haematuria, electrolyte imbalance, to acute kidney injury (AKI). AKI is rare, could be caused by acute tubular necrosis (ATN) resulting from shock due to plasma leakage in DSS, and haemolysis or rhabdomyolysis [9,10]. Other causes like immune-mediated and direct viral invasion are the other probable causes. The renal impairment usually recovers with time if treated appropriately; however, AKI is associated with high mortality, prolonged hospital stay, and often requires dialysis [43,44]. Infection-related glomerulonephritis - proliferative and exudative glomerulonephritis (IRGN) is rare in DF and responds to steroids [44].

8 MUSCULOSKELETAL MANIFESTATIONS

Severe myalgia is a common feature in DF. Rhabdomyolysis and myositis are relatively uncommon in DHF [9]. Myotoxic cytokine tumour necrosis factor (TNF) is responsible for rhabdomyolysis. Raised serum Creatine Phosphokinase (CPK) and myoglobinuria are suggestive of rhabdomyolysis. It needs adequate hydration to prevent AKI. Myositis presents as acute motor weakness and is characterized by raised serum CPK and myopathic electromyography. Myalgia in dengue is short-

lived and resolves spontaneously, but post-infection prolonged myalgia may occur. Both myositis and prolong myalgia respond well to corticosteroids [9,10]. One case report from Australia showed non-responsiveness to non-steroidal anti-inflammatory drugs but, prompt response to steroid [45].

9 HAEMATOLOGICAL MANIFESTATIONS

Dengue may present with different atypical haematological features like haemophagocytic lymphohistiocytosis (HLH), disseminated intravascular coagulation (DIC), and pancytopenia. Though HLH is mostly associated with Ebstein-Barr virus infection, cases associated with DENV are also reported [46,47]. This rare hyperinflammatory disorder is thought to be due to the activation of bone marrow macrophages by increased production of cytokines, including interferon γ (IFN-γ), tumor necrosis factor α (TNF-α), and interleukin 6 by virus-infected T lymphocytes [48]. HLH is characterized by prolong fever, hepatosplenomegaly, lymphadenopathy with bicytopenia or pancytopenia, increased ferritin, lactate dehydrogenase (LDH), SGPT, hypertriglyceridaemia, hypofibrinogenaemia, low/ absent natural killer cell (NK-cell) activity, high soluble interleukin-2 receptor level [49]. Corticosteroids, etoposide, cyclosporin are the mainstay of treatment and other supportive care with necessary blood products and empirical antibiotics improve the outcome [46,47].

DIC is another serious manifestation of DHF/DSS. Different mechanisms are being postulated for DIC. In DENV infection the increased expression of tissue factor (TF) and endothelial injury led to the activation of the coagulation cascade and excess fibrinolysis resulting in consumption coagulopathy. TNF-α and IL-6 can also activate the coagulation cascade by the TF pathway. DIC could be the result of increased vascular permeability as well. DIC causes clots in microcirculation leading to multi-organ dysfunction. There is a paradoxical haemorrhage due to consumption coagulopathy and thrombocytopenia [50]. Huang et al showed in a study that thrombocytopenia, prolong activated partial thrombin time (APTT), and raised tissue plasminogen activator (tPA) occurring in the acute phase in dengue indicated activation of coagulation and fibrinolysis. In the convalescent phase platelet count and PAI-1 are raised with a decline in tPA level and normalization of APTT. These changes were more in DHF/DSS than DF indicating the relation of DIC with disease severity [51].

10 LYMPHORETICULAR MANIFESTATIONS

Lymphadenopathy may occur in more than half of the cases of DF, however lymph node infarction is rare. Lymphoma needs to be excluded in persistent lymphadenopathy [9,52].

Splenomegaly is found rarely, mostly in infants. It is often complicated with subcapsular haematoma and splenic rupture. Splenic rupture is suspected when there is sudden severe abdominal pain with features of peritonitis, hypotension, and falling haematocrit, and needs urgent splenectomy [53].

11 CONCLUSION

With the increasing burden of DF, the atypical manifestations are also increasing. Increasing awareness of physicians is necessary for early diagnosis and appropriate management. In endemic areas, especially during the outbreak, any patient with fever should be evaluated for dengue, keeping the atypical manifestations in mind.

REFERENCES

[1] World Health Organization. Ten threats to global health in 2019. [Online]. Available from: https://www.who.int/news-room/feature-stories/ten-threats-to-global-health-in-2019. [Cited on 21 November 2019].

[2] World Health Organization. Dengue and severe dengue. [Online]. Available from: https://www.who.int/news-room/fact-sheets/detail/dengue-and-severe-dengue. [Cited on 3 November 2019].

[3] Dejnirattisai W, Duangchinda T, Lin CL, Vasanawathana S, Jones M, et al. A complex interplay among virus, dendritic cells, T cells, and cytokines in dengue virus infections. J Immunol.2008; 181(9):pp.5865-74. https://pubmed.ncbi.nlm.nih.gov/18941175/

[4] Green S, Rothman A. Immunopathological mechanisms in dengue and dengue hemorrhagic fever. Curr Opin Infect Dis.2006; 19(5):pp.429-36. https://pubmed.ncbi.nlm.nih.gov/16940865/

[5] Restrepo BN, Ramirez RE, Arboleda M, Alvarez G, Ospina M, et al. Serum levels of cytokines in two ethnic groups with dengue virus infection. Am J Trop Med Hyg.2008; 79(5):pp.673-7. https://www.unboundmedicine.com/medline/citation/18981502/

[6] Dejnirattisai W, Jumnainsong A, Onsirisakul N, Fitton P, Vasanawathana S, et al. Cross-reacting antibodies enhance dengue virus infection in humans. Science.2010; 328(5979):pp.745-8. https://pubmed.ncbi.nlm.nih.gov/20448183/

[7] Schmidt AC. Response to dengue fever--the good, the bad, and the ugly?. N Engl J Med.2010;

363(5):pp.484-7. https://pubmed.ncbi.nlm.nih.gov/20818870/

[8] Kyle JL, Harris E. Global spread and persistence of dengue. Annu Rev Microbiol.2008; 62:pp.71-92. https://pubmed.ncbi.nlm.nih.gov/18429680/

[9] Gulati S, Maheshwari A. Atypical manifestations of dengue. Trop Med Int Health.2007; 12(9):pp.1087–95. https://onlinelibrary.wiley.com/doi/full/10.1111/j.1365-3156.2007.01891.x

[10] Nimmagadda SS, Mahabala C, Boloor A, Raghuram PM, Nayak U A. Atypical Manifestations of Dengue Fever (DF) - Where Do We Stand Today? J Clin Diagn Res.2014; 8(1):pp.71-3. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3939591/

[11] Souza LJ De, Alves JG, Bastos DA, Wallace E. Aminotransferase changes and acute hepatitis in patients with dengue fever: Analysis of 1,585 cases. Braz J Infect Dis.2004; 8:pp.156–63. https://pubmed.ncbi.nlm.nih.gov/15361994/

[12] Koh FH, Misli H, Chong VH. Acute acalculous cholecystitis secondary to dengue fever. Brunei Int Med J.2011; 7(1):pp.45–49.

[13] Wu KL, Changchien CS, Kuo CM, Chuah SK, Lu SN. Dengue fever with acute acalculous cholecystitis. Am J Trop Med Hyg.2003; 68(6):pp.657–60. https://pubmed.ncbi.nlm.nih.gov/12887023/

[14] Bhatty S, Shaikh NA, Fatima M, Sumbhuani AK. Original Article Acute acalculous cholecystitis in dengue fever. J Pak Med Assoc.2009; 59(8):pp.519–21. https://pubmed.ncbi.nlm.nih.gov/19757695/

[15] Shapiro MJ, Luchtefeld WB, Kurzweil S, Kaminski DL, Durham RM, et al. Acute acalculous cholecystitis in the critically ill. Am Surg.1994; 60:pp.335-9. https://pubmed.ncbi.nlm.nih.gov/8161083/

[16] Torres JR, Liprandi F, Goncalvez AP. Atypical parotitis due to dengue virus. Clin Infect Dis.2000; 31(5):pp.28-29. https://pubmed.ncbi.nlm.nih.gov/11073786/

[17] Gonzalez GR, Henao AF. Dengue hemorrhagic fever complicated by pancreatitis. Braz J Infect Dis.2011; 15(5):pp.490–92. https://www.scielo.br/scielo.php?script=sci_arttext&pid=S1413-86702011000500015

[18] Setiawan MW, Samsi TK, Wulur H, Sugianto D, Pool TN. Epigastric pain and sonographic assessment of the pancreas in dengue. J Clin Ultrasound.1997; 26(5):pp.257–9. https://pubmed.ncbi.nlm.nih.gov/9608369/

[19] Gupta VK, Lalchandani A, Agrawal K. Neurological manifestations of dengue infection. Ann Trop Med Public Health.2015; 8:pp.117-21. http://www.atmph.org/text.asp?2015/8/4/117/162370

[20] Chen WJ, Huang KP, Fang AH. Detection of IgM antibodies from cerebrospinal fluid and sera of dengue fever patients. Southeast Asian J Trop Med Public Health.1991; 22:pp.659-63. https://pubmed.ncbi.nlm.nih.gov/1820657/

[21] Lum LC, Lam SK, Choy YS, George R, Harun F. Dengue encephalitis: A true entity? Am J Top Med Hyg.1996; 54:pp.256-9. https://pubmed.ncbi.nlm.nih.gov/8600761/

[22] Jackson ST, Mullings A, Bennett F, Khan C, Gordon-Strachan G, et l. Dengue infection in patients presenting with neurological manifestations in a dengue-endemic population. West Indian Med J.2008; 57:pp.373-6. https://pubmed.ncbi.nlm.nih.gov/19566019/

[23] Kunishige M, Mitsui T, Tan BH, Leong HN, Takasaki T, et al. Preferential gray matter involvement in dengue myelitis. Neurology.2004; 63:pp.1980-1. https://pubmed.ncbi.nlm.nih.gov/15557535/

[24] Soares CN, Cabral-Castro MJ, Peralta JM, Freitas MR, Puccioni-Sohler M. Meningitis determined by oligosymptomatic dengue virus type 3 infection: Report of a case. Int J Infect Dis.2010; 14:e150-2. https://pubmed.ncbi.nlm.nih.gov/19501535/

[25] Mathew S, Pandian JD. Stroke in patients with dengue. J Stroke Cerebrovasc Dis.2010; 19:pp.253-6. https://pubmed.ncbi.nlm.nih.gov/20434057/

[26] Tomar LR, Raizada A, Yadav A, Agarwal S. Dengue fever presenting as hypokalaemic muscle weakness. Trop J Med Res.2016; 19:pp.76-8. http://www.tjmrjournal.org/text.asp?2016/19/1/76/172058

[27] Trung DT, Wills B. Systemic vascular leakage associated with dengue infection - the clinical perspective. Curr Top Microbiol Immunol.2010; 338:pp.57-66. https://pubmed.ncbi.nlm.nih.gov/19802578/

[28] Mairuhu AT, Setiati TE, Koraka P, Hack CE, Leyte A, Faradz SM, et al. Increased PAI-I plasma levels and risk of death from dengue: No association with the 4G/5G promoter polymorphism. Thromb J.2005; 3:pp.17-25. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1308869/

[29] Brito CA, Sobreira S, Cordeiro MT, Lucena-Silva N. Acute disseminated encephalomyelitis in classic dengue. Rev Soc Bras Med Trop.2007; 40:pp.236-8. https://www.scielo.br/scielo.php?script=sci_arttext&pid=S0037-86822007000200019

[30] Seet RC, Lim EC, Wilder-Smith EP. Acute transverse myelitis following dengue virus infection. J Clin Virol.2006; 35:pp.310-2.

[31] de Aragao RE, Barreira IM, Lima LN, Rabelo LP, Pereira FB. Bilateral optic neuritis after dengue viral infection: Case report. Arq Bras Oftalmol.2010; 73:pp.175-8. https://pubmed.ncbi.nlm.nih.gov/20549049/

[32] Ansari MK, Jha S, Nath A. Unilateral diaphragmatic paralysis following dengue infection. Neurol India.2010; 58:pp.596-8.

[33] Donnio A, Beral L, Olindo S, Cabie A, Merle H. Dengue, a new etiology in oculomotor paralysis. Can J Ophthalmol.2010; 45:pp.183-4.

[34] Bacsal KE, Chee SP, Cheng CL, Flores JV. Dengue-associated maculopathy. Arch Ophthalmol.2007; 125:pp.501-10. https://pubmed.ncbi.nlm.nih.gov/17420370/

[35] Seet RC, Quek AM, Lim EC. Post-infectious fatigue syndrome in dengue infections. J Clin Virol.2007; 38:pp.2-6.

[36] Datta G, Mitra P. A Study on Cardiac Manifestations of Dengue Fever. J Assoc Physicians India.2019;

67:pp.14-16. https://pubmed.ncbi.nlm.nih.gov/31559761/

[37] Wiwanikit. Dengue Cardiac Infection, A Brief Review. Acta Cardiol Sin.2008; 24:226. https://www.ijmedicine.com/index.php/ijam/article/view/535

[38] Kaushik JS, Gupta P, Rajpal S, Bhatt S. Spontaneous resolution of sinoatrial exit block and atrioventricular dissociation in a child with dengue fever. Singapore Med J.2010; 51(9):e146-8. https://pubmed.ncbi.nlm.nih.gov/20938598/

[39] Lum LC, Thong MK, Cheah YK, Lam SK. Dengue-associated adult respiratory distress syndrome. Ann Trop Paediatr.1995; 15(4):pp.335-9. https://pubmed.ncbi.nlm.nih.gov/8687212/

[40] Miyata N, Yoshimura Y, Tachikawa N, Amano Y, Sakamoto Y,et al. Cavity Forming Pneumonia Due to Staphylococcus aureus Following Dengue Fever. Am J Trop Med Hyg.2015; 93(5):pp.1055-7. https://pubmed.ncbi.nlm.nih.gov/26304914/

[41] Macedo RN, Rocha FA, Rolim DB, Vilar DC, Araújo FM, et al. Severe coinfection of melioidosis and dengue fever in Northeastern Brazil: first case report. Rev Soc Bras Med Trop.2012; 45(1):pp.132-3. https://doi.org/10.1590/S0037-86822012000100028

[42] Wiwanitkit V. Lund Disease in Dengue. Journal of Resp Research.2015; 1(1):pp.7-9. http://www.ghrnet.org/index.php/jrr/article/view/1372/1791

[43] Vachvanichsanong P, Thisyakorn U, Thisyakorn C. Dengue hemorrhagic fever and the kidney. Arch Virol.2016; 161(4):pp.771-8. https://pubmed.ncbi.nlm.nih.gov/26699788/

[44] Eswarappa M, Reddy SB, John MM, Suryavara S, Madhyashatha RP. Renal manifestations of dengue viral infection. Saudi J Kidney Dis Transpl.2019; 30:pp.394-400. http://www.sjkdt.org/text.asp?2019/30/2/394/256847

[45] Finsterer J, Kongchan KJ. Severe, persisting, steroid-responsive Dengue myositis. J Clin Virol.2006; 35:pp.426-428.

[46] Ramachandran B, Balasubramanian S, Abhishek N, Ravikumar KG, Ramanan AV. Profile of hemophagocytic lymphohistiocytosis in children in a tertiary care hospital in India. Indian Pediatr.2011; 48:pp.31–5. https://pubmed.ncbi.nlm.nih.gov/20972297/

[47] Ray S, Kundu S, Saha M, Chakrabarti P. Hemophagocytic Syndrome in Classic Dengue Fever. J Glob Infect Dis.2011;3(4):pp.399–401. http://www.jgid.org/text.asp?2011/3/4/399/91068

[48] Karras A, Hermine O. Macrophage activation syndrome. Rev Med Interne.2002; 23:pp.768–78. http://www.sciencepublishinggroup.com/journal/paperinfo?journalid=235&doi=10.11648/j.ajlm.20200501.14

[49] Henter JI, Horne A, Aricó M, Egeler RM, Filipovich AH, et al. HLH-2004: Diagnostic and therapeutic guidelines for hemophagocytic lymphohistiocytosis. Pediatr Blood Cancer.2007; 48(2):pp.124-31. https://pubmed.ncbi.nlm.nih.gov/16937360/

[50] de Azeredo EL, Monteiro RQ, de-Oliveira Pinto LM. Thrombocytopenia in Dengue: Interrelationship between Virus and the Imbalance between Coagulation and Fibrinolysis and Inflammatory Mediators. Mediators Inflamm.2015; 2015: 313842. https://pubmed.ncbi.nlm.nih.gov/25999666/

[51] Huang YH, Liu CC, Wang ST, Lei HY, Liu HL, et al. Activation of coagulation and fibrinolysis during dengue virus infection. J Med Virol.2001; 63(3):pp.247-51. https://pubmed.ncbi.nlm.nih.gov/11170065/

[52] Rao IS, Loya AC, Ratnakar KS, Srinivasan VR. Lymph node infarction -a rare complication associated with disseminated intravascular coagulation in a case of dengue fever. BMC Clin Pathol.2005;5:11. https://bmcclinpathol.biomedcentral.com/articles/10.1186/1472-6890-5-11

[53] Karakus A, Banga N, Voorn GP, Meinders AJ. Dengue shock syndrome and rhabdomyolysis. Neth J Med.2007; 65:pp.78-81. https://pubmed.ncbi.nlm.nih.gov/17379934/