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Case Report

Internal Medicine Section

Dengue Fever Complicated by Intracerebral Haemorrhage, Acute Respiratory Failure and Nosocomial Pneumonia: A Case Report

RONAK SHAH[1], PARTH SHAH[2], MITTAL SINDHAV[3]

ABSTRACT

Dengue fever is a mosquito-borne viral illness caused by the dengue virus. Majority of cases have self-limiting course, rarely severe dengue causes fatal neurological complications like encephalitis and haemorrhage. This is case report of a 38-year-old male who presented with three days of high-grade fever, one day of altered sensorium, headache, and generalised weakness. Serological testing confirmed dengue IgM positivity. The clinical course was complicated by a rapid decline in Glasgow Coma Scale (GCS) from 15 to 11, necessitating endotracheal intubation on day two in the setting of blood-stained pulmonary secretions. High-resolution Computed Tomography (CT) of the thorax suggested consolidation with pulmonary oedema. CT of the brain revealed a 3.9×3.8×3.5 cm Intracerebral Parenchymal Haemorrhage (IPH) with surrounding oedema causing significant mass effect. Laboratory parameters documented severe thrombocytopaenia (nadir 0.6 lac/cumm), coagulopathy, hepatocellular injury with peak SGOT/SGPT of 227/276 IU/L, and elevated C-reactive protein at 85.6 mg/L. Endotracheal aspirate cultures grew extensively drug-resistant Acinetobacter baumannii on day five, requiring escalation of antimicrobial therapy to meropenem and polymyxin. Supportive management included platelet and Fresh Frozen Plasma (FFP) transfusion. The patient was successfully extubated on day nine and discharged home on day twelve. Notably, the simultaneous occurrence of dengue-associated Intracerebral Haemorrhage (ICH), acute hypoxaemic respiratory failure, and Multi-Drug Resistant (MDR) nosocomial pneumonia in a young immunocompetent adult represents a triad that is rarely documented in the published literature. Early recognition of neurological deterioration, timely neuroimaging, aggressive blood product support, and targeted antimicrobial therapy with conservative management of ICH remains critical determinants of a favourable outcome.

Keywords: Mechanical ventilation, Multi-organ dysfunction, Thrombocytopaenia

CASE REPORT

A 38-year-old male with no significant prior medical history presented to the emergency department with a three-day history of high-grade continuous fever (measured up to 103°F), associated with severe bi-frontal headache, myalgia, and generalised weakness. On the day of admission, the family noted progressive confusion and inappropriate behaviour, suggesting acute alteration in sensorium.

On examination at presentation, the patient was afebrile (temperature 37.2°C), blood pressure was 140/80 mmHg, pulse was 90 beats per minute with regular rhythm, respiratory rate was 20 breaths per minute, and oxygen saturation (SpO_2) was 94% on room air. Neurological examination revealed a GCS of E4M6V5 (15/15). Power was 5/5 in all four limbs, deep tendon reflexes were normal, and bilateral plantar responses were flexor. There were no signs of meningism. Chest auscultation was clear. Abdominal examination was unremarkable at this stage.

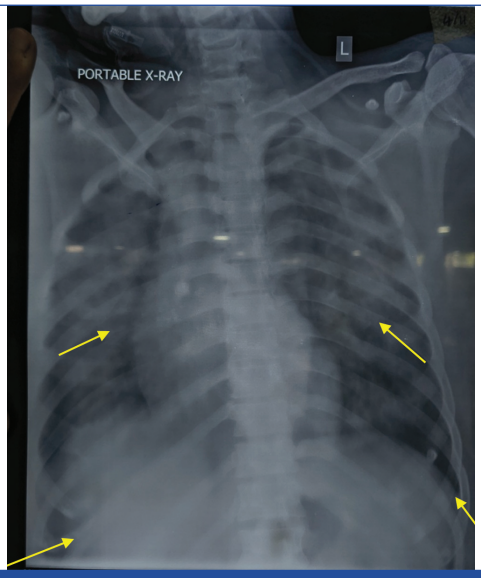
Initial investigations confirmed dengue IgM positivity on the NS1 antigen and IgM/IgG ELISA panel, establishing the diagnosis of dengue fever. Haematological parameters revealed profound thrombocytopaenia with a platelet count of 0.6 lac/cumm on day one. Haemoglobin was 14.5 g/dL. The white cell count was initially normal at 7500/cumm. Liver function tests showed mild hyperbilirubinaemia (total bilirubin 2.8 mg/dL, direct 1.2, indirect 1.6 mg/dL) with mildly elevated transaminases (SGOT 52, SGPT 45 IU/L). Renal function and serum electrolytes were within normal limits. Prothrombin time was mildly prolonged at 16 seconds (INR 1.2). Serum LDH was 395 IU/L (day two). Serum uric acid was 7.8 mg/dL. CRP was 18 mg/L on day one, rising to 85.6 mg/L by day five [Table/Fig-1]. Electrocardiography and transthoracic echocardiography were normal. Chest X-ray suggestive of bilateral pleural effusions suggestive of pulmonary oedema with superimposed infective consolidation [Table/Fig-2]. Ultrasonography of the abdomen and pelvis demonstrated hepatomegaly (liver span 17 cm) and bilaterally raised renal echogenicity.

Serial haematological data documenting the evolving pattern of thrombocytopaenia, coagulopathy, hepatic injury, and inflammatory markers are detailed in [Table/Fig-1].

Parameters	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11
Hb (g/dL)	14.5	14.6	14.2	13.1	12.7	13.4	14.0	13.0	14.3	14.9	14.8
WBC (/cumm)	7500	8400	12970	14180	17300	18000	20000	18000	11000	7800	6100
Platelets (lac/cumm)	0.6	0.6	1.05	1.54	1.92	2.62	2.6	2.7	2.7	2.68	2.7
PT/INR (sec)	16/1.2	17/1.22	17.5/1.2	21/1.5	14.3/1.03	14.6/1.04	21.5/1.6			15/1.02	
aPTT (sec)	37	38	37.7	40	34	36	34			34	
Creatinine (mg/dL)	0.6	0.5	0.6	0.8	1.0	1.0	1.1	0.8	0.9	1.0	
Urea (mg/dL)	42	23	22	44	37	24	25	22	22	20	
Bilirubin T/D/I (mg/dL)	2.8/1.2/1.6	2.0/1.5/0.5	2.1/0.8/1.3	2.6/1.0/1.6	2.7/1.2/1.5	2.6/1.2/1.4	1.6/0.8/0.8	1.5/0.7/0.8	1.0/0.5/0.5	1.2/0.5/0.7	
SGOT (IU/L)	52	77	79	101	198	227	172	105	128	60	40
SGPT (IU/L)	45	53	63	76	236	276	220	205	198	87	52

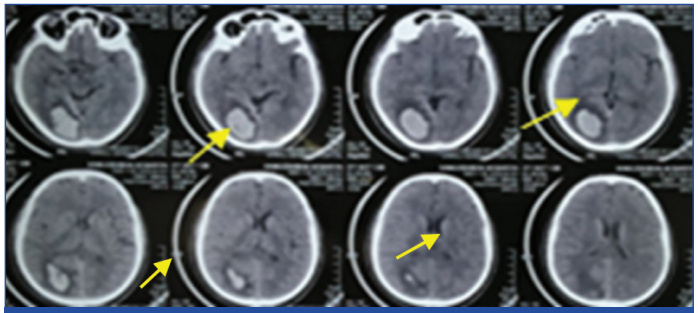
LDH (IU/L)		395								200	
CRP (mg/L)	18				85.6					14	
Na+ (mEq/L)	136	142	139	146	139	139	145	148	146	146	
K+ (mEq/L)	3.8	4.3	4.2	3.9	3.9	5.0	4.4	3.9	3.9	3.7	

[Table/Fig-1]: Serial laboratory investigations during hospital stay.
Hb: Haemoglobin; WBC: White blood cell count; PT: Prothrombin time; INR: International normalised ratio; aPTT: Activated partial thromboplastin time; LDH: Lactate dehydrogenase; CRP: C-reactive protein



[Table/Fig-2]: Bilateral symmetrical airspace opacification with perihilar predominance and interstitial shadowing is noted, accompanied by mild blunting of bilateral costophrenic angles consistent with pleural effusions suggestive of pulmonary oedema with superimposed infective consolidation.

At the time of admission, neurological examination revealed a GCS of 15/15 (E4M6V5), with no focal motor deficits, no cranial nerve palsies, and intact cerebellar signs. On day two, the patient experienced an acute decline in GCS to 11/15 (E3M5V3) with no new focal motor deficits and no cranial nerve palsies, with pupils remaining equal, round, and bilaterally reactive, and cerebellar signs remaining elicitable. In view of the rapidly deteriorating neurological status, blood-stained secretions from the oropharynx and inability to protect the airway, the patient was emergently intubated and placed on invasive mechanical ventilation. The patient was placed on volume-controlled assist-control ventilation with the following initial settings: tidal volume 4 mL/kg ideal body weight (lung-protective strategy), respiratory rate 14 breaths per minute, Positive End-Expiratory Pressure (PEEP) 6 cmH_2O, and FiO_2 0.6, targeting SpO_2 ≥94% and plateau pressure below 30 cmH_2O. Head of bed elevation was maintained at 30–45 degrees throughout. CT of the brain demonstrated an IPH measuring 3.9 × 3.8 × 3.5 cm in the right basal ganglia region with surrounding perihaematomal oedema, causing mass effect in the form of effacement of ipsilateral sulci and gyri. No midline shift or intraventricular extension was identified [Table/Fig-3]. Patient developed IPH on day 5 of illness placing the event within the critical thrombocytopaenic window typically observed between days four and six of dengue illness, when platelet nadir is most profound. Based on WHO dengue classification



[Table/Fig-3]: CT head: Axial CT demonstrates a hyperdense intracerebral haematoma measuring 3.9×3.8×3.5 cm within the right basal ganglia, with surrounding perihaematomal oedema and ipsilateral sulcal effacement. No midline shift or intraventricular extension is identified.

criteria, this patient was categorised as severe dengue on account of severe organ impairment specifically, dengue-associated ICH with neurological compromise, acute hypoxaemic respiratory failure requiring mechanical ventilation, and hepatic involvement with transaminase elevation, in the context of confirmed dengue seropositivity and profound thrombocytopaenia [1]. The decision to manage the intracranial haemorrhage conservatively was reached following multidisciplinary discussion with the neurosurgical team, based on the following considerations. First, the estimated haematoma volume of approximately 27 mL, calculated using the ABC/2 method, fell below the 30 mL threshold above which surgical evacuation is generally favoured in basal ganglia haemorrhage. Second, the haematoma was located within the deep basal ganglia, a surgically eloquent region where operative intervention carries significant risk of iatrogenic neurological morbidity. Third, there was no intraventricular extension, no transtentorial herniation, and no progressive midline shift on serial imaging. Fourth, the concurrent profound thrombocytopaenia and coagulopathy substantially elevated operative haemorrhagic risk, making surgical intervention disproportionately hazardous in the acute phase. Clinical neurological monitoring served as the primary surrogate for haematoma progression assessment as repeated CT head was not done due to logistic issues. HRCT of the thorax revealed bilateral symmetrical air-space consolidation with surrounding ground-glass opacification in a peribronchovascular distribution, smooth interlobular septal thickening, mild bilateral pleural effusions with underlying segmental collapse, appearances consistent with pulmonary oedema alongside an infective aetiology.

Initial antimicrobial therapy was commenced with intravenous ceftriaxone one gram 12 hourly and clindamycin 600mg three times a day targeting community-acquired respiratory pathogens. Given the profound thrombocytopaenia, coagulopathy, and active haemorrhage from the endotracheal tube, the patient received 4 random donor platelet transfusions on Day 1 and 2 targeting a platelet count above 1.5 lac/cumm, as well as four units of Fresh-Frozen Plasma (FFP) to correct the coagulopathy (peak INR 1.5 on day four). Fluid management followed a cautious restrictive strategy given the competing risks of cerebral oedema, pulmonary oedema, and dengue-associated plasma leakage. Intravenous fluid administration was guided by serial haematocrit monitoring, urine output targets of 0.5-1 mL/kg/hour, clinical assessment of hydration status and inferior venacava diameter and collapsibility index assessed by bedside point-of-care ultrasonography Isotonic crystalloid (0.9% normal saline) alternate with ringer's lactate was used as the primary resuscitation fluid, administered at maintenance rates of 1-1.5 mL/kg/hour and titrated against haemodynamic parameters.

Quantitative endotracheal aspirate culture obtained on day three was reported on day five as growing Acinetobacter baumannii resistant to ceftriaxone, piperacillin-tazobactam, and carbapenems on conventional sensitivity, but intermediate sensitive only to polymyxin suggestive of Extensively Drug Resistant (XDR) organism. Antimicrobial therapy was escalated to intravenous meropenem 2 g every eight hours (extended infusion) and intravenous polymyxin B as per the culture and sensitivity report, in keeping with hospital antimicrobial stewardship protocols.

The patient demonstrated progressive clinical improvement from day six onwards with stabilisation of GCS, reduction in ventilator support requirements, improvement in platelet count (reaching

2.62 lac/cumm by day six), and normalisation of coagulation parameters. Liver enzymes peaked on day six (SGOT 227, SGPT 276 IU/L) and trended downward thereafter. Inflammatory markers improved with CRP declining to 14 mg/L by day ten and LDH returning to 200 IU/L. The ventilator support was gradually shifted from volume-controlled assist-control ventilation to continuous positive airway pressure mode and FiO_2 was weaned progressively from 0.6 on day two to 0.35 by day eight. The patient passed a spontaneous breathing trial on day nine and was successfully extubated to supplemental oxygen via face mask, with subsequent weaning to room air within 24 hours. He was transferred to the general ward on day ten and discharged home on day twelve in a clinically stable condition with a GCS of 15/15, resolved thrombocytopaenia, and normalising hepatic function. The patient was reviewed at an outpatient clinic two weeks following discharge. At this visit, he was ambulatory and independent in all activities of daily living, with no residual motor deficits, speech disturbance, or cognitive complaints reported by the patient or family. Repeat complete blood count demonstrated normalised platelet count at 2.8 lac/cumm and resolving transaminase elevation.

DISCUSSION

Dengue fever, caused by DENV 1-4 and primarily transmitted by *Aedes aegypti*, is the world's fastest-spreading mosquito-borne infection, with WHO estimating 100-400 million cases annually across tropical and subtropical regions. While most infections are self-limiting, severe dengue can manifest as plasma leakage, shock, haemorrhage, and organ impairment in a subset of patients [1,2].

This case report represent an unusual case of dengue fever in a 38-year-old immunocompetent male presented with dengue fever complicated by a 3.9×3.8×3.5 cm right basal ganglia ICH, acute hypoxaemic respiratory failure requiring nine days of mechanical ventilation, and XDR Acinetobacter baumannii ventilator-associated pneumonia. Profound thrombocytopaenia with a nadir platelet count of 0.6 lac/cumm, coagulopathy, and acute respiratory failure complicated the condition of the patient. Management included conservative neurosurgical care, serial platelet transfusions, IVC-guided fluid resuscitation, lung-protective ventilation, and targeted combination antimicrobial therapy with meropenem and polymyxin B. Despite the severity and multi-system nature of the illness, the patient was discharged neurologically intact on day twelve, underscoring the importance of early recognition, timely neuroimaging, and coordinated multidisciplinary intensive care in severe dengue.

Neurological complications, including encephalitis, encephalopathy, Guillain-Barré syndrome, and intracranial haemorrhage, occur in 0.5-6% of dengue cases, with haemorrhage primarily driven by thrombocytopaenia and endothelial dysfunction [3]. It occurs via direct neurotropism, immune-mediated mechanisms, or haemorrhagic events secondary to thrombocytopaenia and endothelial dysfunction. The dengue virus directly infects endothelial cells, disrupting capillary integrity and increasing vascular permeability, while immune-mediated platelet destruction produces profound thrombocytopaenia [4]. In this patient, a platelet nadir of 60,000/cumm, peak INR of 1.5, and mildly prolonged aPTT collectively impaired haemostasis, reflecting dengue-induced hepatocellular dysfunction. Neurological deterioration on day two of admission, corresponding to day five of illness, coincided with the critical thrombocytopaenic window (days four to six), when spontaneous haemorrhage risk is highest.

Carod-Artal FJ et al., conducted a comprehensive review of neurological complications in dengue and reported that dengue-associated ICH predominantly occurred in young adults from Southeast Asian and South Asian endemic zones associated with severe thrombocytopaenia and deranged coagulation [4], which aligns with the demographic profile of this 38-year-old male patient. However, a key distinction in this case is the size of the haematoma (3.9×3.8×3.5 cm) causing appreciable mass effect managed by conservative approach, whereas Carod-Artal FJ et al., noted smaller dengue-related ICH cases spontaneously resolving without significant mass effect on medical management [4].

In the present case, patient had haematoma confined to a single compartment within the right basal ganglia without subdural extension, intraventricular involvement, or significant midline shift; the estimated volume of approximately 27 mL fell below the surgical threshold so conservative management was sustained throughout without operative intervention. In contrast, Sam JE et al., reported a 47-year-old male who presented on day seven of dengue illness with generalised tonic-clonic seizure, and a GCS of E1V1M3 with anisocoria, in whom CT brain revealed a right frontotemporoparietal acute subdural haemorrhage of 30 mm thickness with a concurrent left thalamic bleed, 20 mm midline shift, and severe coagulopathy {International Normalised ratio (INR) 2.63, Activated Partial THromboplastin Time (APTT) 40.5 seconds} and thrombocytopaenia (platelet count 31×10^9/L), with NS1 antigen positivity and IgG positivity consistent with dengue infection. Despite haematological correction with FFP and platelet concentrate- achieving a platelet count of 56×10^9/L and INR of 1.2- bilateral fixed dilated pupils developed prior to surgery. Emergency right decompressive craniectomy, clot evacuation, external ventricular drainage, and duraplasty were performed eight hours after admission; however, postoperative CT demonstrated generalised cerebral oedema, bilateral thalamic bleeds, and persistent midline shift, and the patient died on day three of admission from haemodynamic collapse [5].

Verma R et al., in a prospective case series from a tertiary care centre in India, identified thrombocytopaenia (<50,000/cumm) as the most consistent predictor of haemorrhagic neurological events, with platelet nadirs of 8,000-45,000/cumm in ICH patients, all of whom survived with conservative management and platelet transfusion [6]. In the present case, a nadir of 60,000/cumm reflected comparable haematological compromise, yet full neurological recovery was achieved, underscoring the importance of aggressive and timely haemostatic support. The intracranial hemorrhage in dengue results from vasculopathy, thrombocytopenia, and platelet dysfunction-induced bleeding diathesis [7].

Wiwanitkit V noted that mechanical ventilation-requiring respiratory failure occurs in fewer than 5% of hospitalized dengue patients, attributable to plasma leakage, myocarditis-induced cardiogenic oedema, or secondary bacterial pneumonia [8]. The present patient's High-Resolution Computed Tomography (HRCT) demonstrated bilateral air-space consolidation, ground-glass opacification, interlobular septal thickening, and pleural effusions, consistent with mixed cardiogenic and infective aetiology, further complicated by haemorrhagic diathesis evidenced by blood-stained tracheal secretions. A normal echocardiogram excluded primary cardiogenic contribution, directing management toward capillary leak and infective mechanisms.

Hsieh CC et al., in a cohort of 75 Intensive Care Unit (ICU)-admitted dengue patients during Taiwan outbreak, reported a 41.3% in-hospital case fatality rate, with 76% requiring mechanical ventilation and independent mortality predictors comprising pre-ICU cardiac arrest, APTT exceeding 48 seconds, and acute kidney injury on admission [9]. The present case shares several risk features with this cohort, including mildly prolonged APTT, significant hepatic transaminase elevation, and mechanical ventilation requirement of nine days- broadly consistent with Hsieh CC et al., reported means. However, the patient was younger at 38 years, immunocompetent, and without pre-existing comorbidities, contrasting with the older, comorbidity-laden non-survivors in Hsieh CC et al., cohort. The absence of all three independent mortality predictors identified by Hsieh CC et al., despite concurrent intracerebral haematoma and XDR nosocomial pneumonia, likely underpinned the favourable outcome, with full neurological recovery and discharge achieved by day 12 [9].

In this patient, the organism MDR Acinetobacter baumannii was identified on day five of admission from an endotracheal aspirate culture, was resistant to cephalosporins and carbapenems, and was sensitive only to polymyxin, prompting escalation to a meropenem-polymyxin B combination. Peleg AY et al., reported that Ventilator Associated Pneumonia (VAP) due to MDR Acinetobacter baumannii in mechanically ventilated patients carries attributable mortality rates of 35-75%, with polymyxin-based combination regimens representing the primary therapeutic option in carbapenem-resistant strains [10]. This patient's favourable outcome with successful extubation on day nine and discharge on day twelve- is notably more optimistic than the high mortality reported by Peleg AY et al., which may reflect a shorter duration of ventilation (9 days), the absence of prior antimicrobial exposure limiting further resistance emergence, and the timely institution of appropriate salvage therapy [10].

Garnacho-Montero J et al., reported combination colistin plus carbapenem achieved superior cure rates over monotherapy (57% vs. 33%) in A. baumannii VAP, with delayed appropriate therapy beyond 48 hours identified as an independent mortality predictor [3]. Although initial empirical therapy in this patient did not cover MDR Acinetobacter, prompt culture-guided escalation by day five likely contributed to the favourable outcome, reinforcing the critical importance of timely microbiologically-directed therapy.

In this case report, patient demonstrated a hepatocellular pattern with peak SGPT (276 IU/L) exceeding SGOT (227 IU/L) on day six, accompanied by mild hyperbilirubinaemia and hepatomegaly, suggesting direct viral hepatic insult potentiated by systemic inflammation and sepsis-related hypoperfusion. Reassuringly, progressive normalisation of transaminases by day ten (SGOT 60, SGPT 87 IU/L) confirmed the typically reversible nature of dengue-associated hepatitis with effective infection control. In contrast, Wahid SF et al., reported disproportionately elevated SGOT over SGPT in dengue haemorrhagic fever, reflecting systemic muscle breakdown and haemolysis alongside hepatocellular injury [11].

CONCLUSION(S)

Dengue fever, while frequently regarded as a self-limiting febrile illness, can precipitate life-threatening multi-system complications in a subset of patients. This case documents a rare and challenging clinical scenario encompassing simultaneous dengue-associated IPH, acute hypoxaemic respiratory failure requiring prolonged mechanical ventilation, and MDR Acinetobacter baumannii ventilator-associated pneumonia. The favourable outcome in this patient highlights that prompt neuroimaging, targeted haemostatic support early airway protection, culture-directed antimicrobial escalation, and meticulous intensive care are the pillars of successful management.

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Written informed consent was obtained from the patient for publication of this case report. Patient confidentiality has been maintained throughout. This report is approved by institutional ethics committee (SVIEC/ON/MEDI/RP/NOC/APRIL 261264).

REFERENCES

[1] World Health Organization. Dengue and severe dengue [Internet]. Geneva: WHO; 2023 [cited 2024].
[2] Bhatt S, Gething PW, Brady OJ, Messina JP, Farlow AW, Moyes CL, et al. The global distribution and burden of dengue. Nature. 2013;496(7446):504-07.
[3] Garnacho-Montero J, Ortiz-Leyba C, Jimenez-Jimenez FJ, Barrero-Almodóvar AE, García-Garmendia JL, Bernabeu-Wittell M, et al. Treatment of multidrug-resistant Acinetobacter baumannii ventilator-associated pneumonia with intravenous colistin: A comparison with imipenem-susceptible VAP. Clin Infect Dis. 2003;36(9):1111-18.
[4] Carod-Artal FJ, Wichmann O, Farrar J, Gascon J. Neurological complications of dengue virus infection. Lancet Neurol. 2013;12(9):906-19.
[5] Sam JE, Gee TS, Wahab NA. Fatal intracranial hemorrhage in a patient with severe dengue fever. Asian J Neurosurg. 2018;13(1):56-58.
[6] Verma R, Sharma P, Garg RK, Atam V, Singh MK, Mehrotra HS. Neurological complications of dengue fever: Experience from a tertiary center of North India. Ann Indian Acad Neurol. 2011;14(4):272-78.
[7] Murthy JMK. Neurological complications of dengue infection. Neurology India. 2010;58(4):581-84.
[8] Wiwanitkit V. Dengue fever: Diagnosis and treatment. Expert Rev Anti Infect Ther. 2010;8(7):841-45.
[9] Hsieh CC, Cia CT, Lee JC, Sung JM, Lee NY, Chen PL, et al. A cohort study of adult patients with severe dengue in Taiwanese intensive care units: The elderly and APTT prolongation matter for prognosis. PLoS Negl Trop Dis. 2017;11(1):e0005270.
[10] Peleg AY, Seifert H, Paterson DL. Acinetobacter baumannii: Emergence of a successful pathogen. Clin Microbiol Rev. 2008;21(3):538-82.
[11] Wahid SF, Sanusi S, Zawawi MM, Ali RA. A comparison of the pattern of liver involvement in dengue hemorrhagic fever with classic dengue fever. Southeast Asian J Trop Med Public Health. 2000;31(2):259-63.

PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Critical Care Medicine, Smt. B.K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.
2. Associate Professor, Department of Anaesthesiology, Ananya College of Medicine and Research, Kalol, Gujarat, India.
3. Assistant Professor, Department of Emergency Medicine, Smt. B.K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Ronak Shah,
104, Nakshtra Enclave Apartment, Behind Bright School, Near Amit Complex,
VIP Road, Karelibaugh, Vadodara-390018, Gujarat, India.
E-mail: drronakshah88@gmail.com

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