REVIEW ARTICLE

Dengue fever-like illnesses: How different are they from each other?

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Abstract
In tropical countries and possibly elsewhere, dengue fever can be confused with other common tropical infections like enteric fever, leptospirosis, typhus fever, malaria, etc. Many of these illnesses can present in significant numbers after rains, and because of similar early presentations, can cause confusion in decision-making. With global warming, these diseases can assume significant proportions even in non-endemic areas. Identifying these illnesses in a non-immune returning traveller is equally challenging. Recognition of these diseases is important to diagnose them and treat them early, in order to avoid potentially fatal complications. This review is an attempt to highlight important clinical and laboratory differences among dengue fever-like illnesses.

Keywords: Dengue fever, enteric fever, leptospirosis, dengue fever-like illnesses (DFLI), other febrile illnesses, acute undifferentiated febrile illness

Dengue fever—an introduction
Dengue fever initially presents with an ‘abrupt onset’ of high fever with headache, retro-orbital pain, malaise, nausea, vomiting, and myalgia. This acute febrile stage lasts for 2–7 days, followed by recovery. The majority of patients make a rapid uneventful recovery without sequelae in the convalescent stage. During defervesence some patients may develop haemorrhagic manifestations, which may be severe. Soft, tender hepatomegaly is common. Thrombocytopenia (platelet count less than 100 × 10^9/l) and rising haematocrit (an increase in haematocrit 20% above average for age, sex, and population) due to plasma leakage are usually detectable before the onset of the subsequent stage of shock, which is an important differentiating characteristic amongst dengue fever-like illnesses (DFLI). Plasma leakage also results in pleural effusion or ascites. Some patients with bleeding or anaemia may not have a rising haematocrit. Dengue shock syndrome (DSS) is associated with varying degrees of circulatory disturbances lasting for 24–48 h. A number of atypical manifestations of dengue have also been reported in recent publications [1]. As against 4 grades of dengue haemorrhagic fever, the World Health Organization (WHO) has recently characterized dengue as with or without warning symptoms. Warning symptoms portend a high mortality.

By looking at the WHO definition of a probable case of dengue fever, any case of febrile illness with 2 or more of the non-specific symptoms of headache, retro-orbital pain, myalgia, arthralgia, rash, haemorrhagic manifestations, and leukopenia pending a serology or isolation of virus, will qualify as having DFLI (Table I). A number of common tropical illnesses may present early with similar clinical manifestations [2,3], calling for recognition of important characteristics (both clinical and laboratory) to differentiate between them. This diagnostic confusion in DFLI is greater during a dengue outbreak. Epidemiological studies have demonstrated that during outbreaks of dengue, 10.5% to 42.7% of cases can be detected by virus isolation, 46.3% by IgM/IgG enzyme-linked immunosorbent assay (ELISA), and 41.8% by haemagglutination inhibition tests [4,5]. Thus, the remaining patients may actually be suffering from DFLI rather than dengue. Suharti et al. [6] evaluated 118 patients who fulfilled the clinical WHO criteria for dengue fever/dengue haemorrhagic fever,
Leptospirosis

Leptospirosis is an emerging infectious disease of global importance, as illustrated by recent large global outbreaks. The clinical manifestations of leptospirosis range from a mild self-limiting febrile illness to a severe and potentially fatal illness characterized by jaundice, renal failure, thrombocytopenia, and haemorrhage (Weil’s disease).

The issue of distinguishing leptospirosis from dengue has been the subject of various studies. During a dengue epidemic not all DFLI test positive for dengue virus [6,7]. Leptospirosis should always be suspected in peculiar epidemiological settings and in certain occupational groups. There is an increase in the number of cases of leptospirosis after rains. Rat infested areas also contribute towards the maintenance of leptospirosis in the community. Occupational groups at high risk are veterinarians, agricultural workers, sewage workers, slaughterhouse employees, and workers in the fishing industry. Recreational water activities can also result in outbreaks [8].

The initial clinical features like fever, headache, chills, myalgia, and arthralgia are not helpful in differentiating dengue fever and leptospirosis [7,9]. Patients with leptospirosis report intermittent and a slightly longer duration of fever, while patients with dengue have continuous fever with chills [9,10]. A rash, petechiae, positive tourniquet test result, and bleeding gums are more common with dengue fever [7,9,10]. Patients with leptospirosis can present with a combination of fever, non-oliguric renal failure, and near normal platelet counts [11]. The median temperature and heart rate at physical examination are higher in leptospirosis patients than in dengue patients [10]. Subconjunctival haemorrhage, which may be confused with conjunctival inflammation, is more commonly reported in patients with leptospirosis [10]. Hepatomegaly has been observed to be present commonly in dengue fever [11].

Differences in laboratory profiles, particularly haematological and liver function tests, have been the subject of interest in various studies. Haemoglobin at the time of admission was not found to be different among dengue and leptospirosis groups [7], but haemoconcentration is significantly found in dengue fever [10,11]. The erythrocyte sedimentation rate (ESR) is often elevated in leptospirosis, as against a normal or low ESR in dengue fever. In fact a normal/low ESR in DSS can be used to differentiate this condition from septic shock in which ESR will be high [8,12]. The most significant single laboratory value independently associated with leptospirosis compared to dengue is the absolute neutrophil count (ANC); patients with leptospirosis have relative neutrophil predominance and absolute neutrophilia,
with a few circulating immature neutrophils (band forms). The percentage of typical and atypical lymphocytes is higher in dengue than leptospirosis, but an absolute lymphopenia remains. Thrombocytopenia is found in both; however the platelet nadir is lower in dengue as compared to leptospirosis [7].

Jaundice is more commonly seen in leptospirosis, although this difference was found not to be statistically significant [10]. Mean plasma aspartate aminotransferase (AST) levels have been found to be slightly higher in dengue compared to leptospirosis, with no differences in alanine aminotransferase (ALT) levels. Mean plasma albumin levels on admission are slightly lower in leptospirosis compared to dengue [7]. The jaundice of Weil’s syndrome is usually not associated with severe hepatic necrosis. Patients with leptospirosis typically have elevated serum levels of bilirubin and alkaline phosphatase, as well as mild increases (up to 200 U/l) in serum levels of aminotransferases. In Weil’s disease, the prothrombin time may be prolonged, but can be corrected with vitamin K [8]. Based on these observations, a predictive model to distinguish leptospirosis from dengue was generated using 3 variables: ANC, plasma albumin, and AST in the first 72 h of illness. A higher probability of leptospirosis compared to dengue is independently associated with a higher ANC, lower albumin levels, and AST levels between 30 and 80 IU/ml on presentation [7]. Varying degrees of renal dysfunction as manifested by the excretion of albumin, cells, and casts may be seen in patients with leptospirosis [13].

**Malaria**

Malaria should be considered in the differential diagnosis of any acute febrile illness until it can be excluded by a definite lack of exposure, by repeated examination of blood smears, or by a therapeutic trial of antimalarials [14]. The early symptoms of malaria are non-specific. A lack of a sense of well being, headache, fatigue, abdominal discomfort, and muscle aches, followed by fever, are all similar to the symptoms of a minor viral illness. Fever can present typically as a recurring paroxysm consisting of chills, fever, and sweats. Myalgia may be prominent, but it is usually not as severe as that in dengue fever and the muscles are not tender as in leptospirosis or typhus. Rash and petechial haemorrhages are rarely seen in malaria. Abdominal symptoms cannot be used to differentiate between dengue fever and malaria [15].

The particular combination of anaemia with thrombocytopenia should raise a clinical suspicion of malaria in the tropics, particularly in the rainy season [16,17]. Thrombocytopenia usually correlates with the degree of parasitaemia, both in Plasmodium vivax and Plasmodium falciparum infections [18,19]. The importance of the peripheral smear cannot be overlooked; this not only confirms the diagnosis but also determines the level of parasitaemia and the species involved. The accuracy of the peripheral smear is operator-dependant and decreases with low levels of parasitaemia. Peripheral smear examination is recommended every 6–8 h until malaria remains a differential.

The coagulation profile, i.e. prothrombin time (PT), activated partial thromboplastin time (Kaolin) (aPTTK), and thrombin time, are normal in P. vivax infection, but they may be prolonged in a P. falciparum infection. Coagulopathy correlates well with the level of parasitaemia [18]. When compared to other tropical infections, leukocyte counts and red cell distribution width (RDW) have been found not to be predictive of malaria [17], while the median leukocyte count has been found to be lower in dengue when compared to other infections [16]. However leukocytosis can be present in complicated falciparum malaria [20].

The detection of intraleukocytic hemozoin during an automated full blood count is a promising new way to avoid misdiagnosis of clinically unsuspected malaria. It has also been demonstrated that the presence of malaria pigment in neutrophils is a marker for disease severity and is better than parasite density. Peripheral parasitaemia does not necessarily reflect the burden of sequestered parasites, especially in P. falciparum. The presence of pigmented neutrophils indicates a recent heavy parasite burden and provides a prognostic marker of disease, while longer-lived pigmented monocytes with longer clearance rates may reflect a more protracted infection or repeated infection [21]. The proportion of circulating monocytes containing hemozoin is associated with anaemia and reticulocyte suppression, independent of the level of circulating cytokines [22]. Hemozoin-containing white blood cells (WBCs) may show the atypical light scattering pattern and thus record a pseudo-eosinophilia on automated counters. Therefore, it has been suggested that in cases of malaria, haematologists should confirm the WBC differential count, particularly the eosinophil count, by microscopy [23].

The median C-reactive protein (CRP) level has been found to be rather low (<5 mg/dl) in dengue as compared to malaria [15]. Malaria has been strongly predicted with a ‘combination’ of enlarged spleen, thrombocytopenia (platelet count <150 ×10^3/μl), fever without localizing symptoms, and hyperbilirubinaemia (total bilirubin level ≥1.3 mg/dl); the highest likelihood ratio of dengue fever was in the combination of leukopenia (leukocytes <4 ×10^3/μl), skin rash, and thrombocytopenia [15]. Atypical lymphocytes
often greater than 5% has been noticed in peripheral smears of malaria [16,24]. Atypical lymphocyte counts are also found in dengue fever. An increase in atypical lymphocytes predicts that within the next 24 h the patient will have no fever and will enter a critical phase if they develop dengue haemorrhagic fever [25]. A high lactate dehydrogenase (LDH) also suggests malaria [16]. Urinalysis can show proteinuria, microscopic haematuria, haemoglobinuria, and red cell casts.

In patients with severe malaria, jaundice is reported in 2.58% of patients [26]. Serum bilirubin, mainly the conjugated fraction, is elevated in malarial hepatopathy. Liver enzymes are usually elevated 2–3-times the normal. The presence of malarial hepatopathy with P. falciparum malaria indicates more severe illness with a higher incidence of complications and a poor prognosis [27]. In contrast, increased levels of alkaline phosphatase and bilirubin have also been found in a smaller proportion of dengue fever cases [28]. Amongst the liver transaminases, there is a greater elevation in AST levels than ALT levels in dengue fever [29]. Liver damage, and consequently increases in aminotransferase levels, is more frequent among females and in patients with dengue haemorrhagic fever [30]. Several cases of fulminant hepatitis in dengue haemorrhagic fever with a high mortality have been reported [31].

Complications associated with malaria caused by P. falciparum are severe anaemia, renal failure, acute respiratory distress syndrome, shock, hypoglycaemia, disseminated intravascular coagulation, and neurological manifestations. Finally, it is also worth mentioning that malaria cannot be ruled out by a single negative peripheral smear or a history of chemoprophylaxis. In fact chemoprophylaxis can alter the symptomatology and can confuse the clinical scenario.

**Enteric fever**

Typhoid and paratyphoid fevers are endemic in the Indian subcontinent, Southeast and Far East Asia, the Middle East, Africa, and Central and South America. These regions are also home to other DFLI. In the first week of the illness, the clinical features are rather non-specific, with headache, malaise, and a rising remittent fever. This step ladder type of fever may not be seen in all patients. Constipation and a mild non-productive cough are common, but some patients may present with diarrhoea. Bottieau et al. [15] found that cough was frequent in enteric fever as compared to other febrile illnesses. Even in the presence of respiratory complaints, the chest X-ray is usually normal. Splenomegaly is usually appreciable by the second week of illness. Out of all febrile illnesses, splenomegaly is usually appreciable in malaria and enteric fever [15]. An enlarged spleen (assessed clinically) and an elevated ALT level were found to be the strongest predictors of enteric fever in ‘non-malaria’ febrile patients [15]. Rose spots, characterized by crops of 2–4-mm diameter pink papules that fade on pressure, develop on the upper abdomen and lower chest between the 7th and 12th days. Relative bradycardia is common during the first 2 weeks of illness.

In enteric fever, the extent of liver involvement varies from mild elevation of transaminases and alkaline phosphatase in almost all patients in the second to third week of illness, to a more dramatic presentation with a picture indistinguishable from viral hepatitis in 1% to 26% of cases [32]. After an analysis of 27 cases of Salmonella hepatitis in comparison to acute viral hepatitis, El-Newihi et al. concluded that Salmonella hepatitis is associated with lower peak serum ALT and serum AST, and a higher peak of serum alkaline phosphatase. AST was found to be higher than ALT in two-thirds of Salmonella hepatitis patients. The admission ALT/LDH ratio, when levels of both enzymes were expressed as multiples of the upper limit of normal value for each, was significantly lower in Salmonella hepatitis. All Salmonella hepatitis cases had a ratio \(< 4\) and all viral hepatitis patients had a ratio \(> 5\). The admission ALT/LDH is the best discriminator between viral hepatitis and Salmonella hepatitis [33]. The ratio suggested by Balasubramanian et al. was \(< 9\) in typhoid hepatitis and \(> 9\) in viral hepatitis. The ALT/LDH ratio has not been evaluated in other DFLI [34]. Other clues that raise the possibility of Salmonella hepatitis include high fever, relative bradycardia, and left shift of WBC [33].

Haematological observations in enteric fever are anaemia, leukopenia, eosinopenia, thrombocytopenia, and sub-clinical disseminated intravascular coagulation. An absolute eosinophil count of 0% can be used as an important diagnostic marker of enteric fever [35,36]. The bone marrow of enteric fever patients shows myeloid maturation arrest, and a decrease in the number of erythroblasts and megakaryocytes [37]. Neutropenic or leukopenic patients may show evidence of haemophagocytosis in the bone marrow. There is an increased number of histiocytes, which phagocytose neutrophils, red blood cells, and platelets, thus explaining the pancytopenias [37,38]. On the other hand, self-limiting haemophagocytosis is an uncommon presentation of dengue infection [39,40].

**Hantavirus**

Outside endemic areas, hantavirus infections may present atypically, the manifestations of the disease depending on the infecting serotype. Demographics
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Chikungunya

Chikungunya virus (CHIKV) has resurfaced after a gap of almost 50 y, with a recent large-scale outbreak. CHIKV is responsible for a 2-stage disease, consisting of an intense acute stage commonly followed by a long-lasting disabling polyarthritis. The incubation period is short (2–6 days), and is followed by an acute stage characterized by a sudden onset high fever, incapacitating polyarthritis, and skin manifestations. In tropical areas, confusion with dengue fever is common, although the 2 diseases present characteristic clinical signs. The general manifestations of CHIKV, which include high fever, intense asthenia, and diffuse myalgia, are not specific. Patients are often confined to bed because of coexisting multiple inflammatory arthralgias or arthritides. This acute rheumatism is mostly bilateral and cumulative within a few days, and especially involves the peripheral joints: hands, wrists, feet, and ankles. Polyarthritis commonly involves more than 10 joint groups. It can be oedematous, asymmetric, or atypical (Baker’s cyst), and is frequently associated with disabling acute tenosynovitis [49]. Some unusual locations are possible, e.g., sternoclavicular and temporomandibular joints, whereas hips are relatively spared. Axial involvement is common at any level.

Skin manifestations usually start after 2–4 days and last for 3 days. They can include a maculopapular rash, diffuse hyperaemia, and oedema of the face and extremities. Less frequently there can be itching, peeling, or epidermolysis. All types can be followed by persisting dyschromic patches on dark skin [49]. Minor and transient mucosal bleeding is possible at this stage.

The second stage of the disease is not constant and is affected by age and underlying diseases, notably rheumatic or traumatic diseases. Pain or stiffness is more severe and more prolonged in older patients and in those who previously had rheumatism. This stage is characterized by persisting polyarthritis, which can severely incapacitate the patient for weeks to more than 1 y. CHIKV-induced chronic rheumatism consists of 3 clinical components: finger and toe polyarthritis with morning pain and stiffness, severe sub-acute tenosynovitis/bursitis in hands, wrists, and ankles, and exacerbation of pain on movement in
previously injured joints and bones [49]. Transitory peripheral vascular disorders, such as Raynaud’s syndrome, can be seen in about a sixth of CHIKV-infected travellers [49]. Chronic hypertrophic tenosynovitis is also common and sometimes induces nerve tunnel syndromes in wrists or ankles. Brighton et al. found that 88% of CHIKV-infected patients declared themselves cured 3 y after disease onset, whereas 12% mentioned persistent symptoms including occasional discomfort, persistent joint stiffness or pain, and effusion [50]. Patients frequently complain of unpredictable relapses, which include the sensation of fever, asthenia, and exacerbation of joint pain and stiffness, and often require intensification of symptomatic treatment.

Unusual manifestations of CHIKV infection include encephalopathy/encephalitis/meningoencephalitis, seizures, neuropathy, Guillain–Barre syndrome, cerebellar syndrome, myocarditis/pericarditis, heart failure, arrhythmias, unstable blood pressure, ischaemic heart disease, nephritis, acute renal failure, pigmentation, genital ulcers, bullous dermatosis, optic neuritis, iridocyclitis, episcleritis, retinitis, antepartum foetal deaths, disseminated intravascular coagulation, pneumonia, respiratory failure, hepatitis, pancreatitis, syndrome of inappropriate secretion of antidiuretic hormone, and hypoadrenalism [51].

As early as 1955, Robinson proposed that CHIKV infection can be distinguished from other epidemic illnesses by lack of pain when moving the eyes and extended duration of joint pain [52]. In chikungunya disease the onset of symptoms is more abrupt, the febrile course shorter, and arthralgia more common than in dengue [53,54]. More chikungunya patients have acute arthritis and they also develop chronic arthritic disabilities, unlike in dengue fever [55,56]. Some 40% of patients present early to hospital because of the abrupt onset of high-grade fever [54]. Nimmanitya et al. noted that conjunctival injection was present more frequently in the chikungunya group than the dengue group [53]. Convulsions associated with high-grade fever have been observed 3 times more frequently in chikungunya fever as compared to dengue fever [54]. In contrast, headache, myalgia, shock, and gastrointestinal haemorrhage are seen more commonly in dengue patients [53–56]. Rashes have been variably found in both groups and may not distinguish them definitively [53,54,56]. Simon et al. noted that in chikungunya rashes appear between days 1 and 4, and in dengue fever they appear between days 3 and 7 [56]. Leukopenia is found in both diseases; neutropenia is characteristic of dengue fever [57]. Thrombocytopenia is found in both, but is more pronounced in dengue fever. However, Kularatne et al. found a positive correlation between duration of illness and platelet counts in chikungunya as compared to a negative correlation in dengue fever [55]. In chikungunya fever, anti-CHIKV IgG and IgM antibodies are detected soon after the onset of symptoms. They are present in all patients in whom an acute infection is confirmed after 5 days. An explanation for early IgG production would be the presence of unusually high presymptomatic viraemia, which provides an early antigen stimulus for antibody formation. This course of antibodies and viraemia is different from those of dengue virus infections, the most important difference being a clear delay in IgG (appearing after IgM) in cases of primary dengue virus infections [58].

**Scrub typhus**

Scrub typhus is an infectious disease caused by Oriens tsutsugamushi, transmitted by infected chigger mites (Leptotrombidium species) and characterized by focal or disseminated vasculitis and perivasculitis, which may involve the lungs, heart, liver, spleen, and central nervous system. The symptoms are usually mild and the clinical course self-limited, with spontaneous recovery after a few days; however, some cases are more severe and protracted, and the disease may be fatal.

The clinical signs and symptoms (fever, 100%; myalgia, 95%; headache, 85%; rash, 85%) of rickettsiosis were found to be similar to those of dengue fever patients in the study of Zavala-Velazquez et al. [59]. In another study, the most common gastrointestinal symptoms reported in patients of scrub typhus were vomiting (65%), nausea (60%), diarrhoea (45%), and haematemesis or melaena (25%). Gastrointestinal signs included hepatomegaly (40%), jaundice (35%), and abdominal pain (20%) [60]. Acute reversible hearing loss in scrub typhus has been described, which has been attributed to vasculitis-induced cochlear damage, either by damage due to direct invasion of the central nervous system by rickettsiae or by an immune-mediated mechanism [61–63]. It has been suggested that scrub typhus should always be suspected in endemic areas if patients present either with pyrexia of unknown origin and hearing loss, or with fever, rash, and acute sensorineural hearing loss/otalgia without otoscopic abnormalities [61,63].

Even though rash is considered the hallmark of rickettsial disease, it may not be seen at presentation or in all patients. Thus it should be remembered that spotted fevers may be spotless too! The rash usually becomes apparent at 3–5 days after the onset of symptoms. Initially the rash is in the form of pink, blanching, discrete macules, which subsequently becomes maculopapular, petechial, or haemorrhagic. Sometimes palpable purpura (typical of vasculitis) is seen.
Occasionally petechiae enlarge to ecchymosis and gangrenous patches may develop. Rarely, gangrene of the digits, earlobes, scrotum, limbs, or nose occurs secondary to vasculitis and thrombosis. The distribution of the rash is initially near the ankles, lower legs, and wrists. Thereafter, the rash spreads centrifugally to involve the whole body. The presence of a rash on the palms and soles, considered so typical of rickettsial disease, can be seen in other diseases like infective endocarditis, syphilis, meningococcaemia, enteroviral diseases, and adverse drug reactions.

Within 2 days after the chigger feeds, a skin papule appears that slowly becomes a small bulla, which sloughs to leave a flat shallow ulcer. By the time the patient is symptomatic, the ulcer is covered with a black eschar 1–2 cm in diameter. The location of eschars ranges widely: the axillae, umbilicus, waist, buttocks, and foot. For this reason it is difficult to search for evidence of an eschar unless a thorough examination of the body is done [64]. Besides, patients are usually unaware of the bite as the eschar is painless and does not itch [62]. Previous studies from different new epidemic areas in northern China have shown that the percentages of patients with scrub typhus with eschars vary from 15% to 100% [64]. Complications of scrub typhus usually develop after the first week of illness and may include pneumonitis, meningocerephalitis, adult respiratory distress syndrome (ARDS), acute renal failure, jaundice, myocarditis, and septic shock.

During the early course of the disease, the total leukocyte count is normal to low normal, with a marked shift to the left. Leukocytosis can be seen in the later stage of disease. Low platelet counts are present in about 60% cases [65]. The ESR is usually high [65]. Rarely, haemophagocytic syndrome has also been described in scrub typhus [66,67]. Thrombocytopenia, hyponatraemia, and normal to low leukocyte counts are certain clues in the early diagnosis of scrub typhus [65]. With regard to liver function testing, elevations of bilirubin, AST, ALT, and alkaline phosphatase levels associated with low serum protein levels have been noted in various studies [60,68,69]. Granulomatous hepatitis has also been reported in scrub typhus [70]. It has been pointed out that amongst the liver transaminases, the rise in AST is higher than ALT [68]. Higher levels of AST and ALT and lower levels of serum albumin were found to be significantly correlated with the severity of scrub typhus [68]. Scrub typhus should be considered in the differential diagnosis if patients are found with fever of unknown origin and abnormal liver function tests [60,69,71].

The early diagnosis of scrub typhus often depends on recognition of the eschar, rash, lymphadenopathy, and exposure history of the patient. However, this combination may not be evident in all patients [72,73]. Because of the similar manifestations, scrub typhus is an important cause of acute undifferentiated fever [59,64,73,74]. Watt et al. have recommended that physicians should consider the possibility of scrub typhus infection in leptospirosis patients who respond poorly to treatment or who have atypical disease manifestations [75].

Four other arboviral diseases have a dengue-like course but without rash: Colorado tick fever, sand fly fever, Rift Valley fever, and Ross River fever. In adults, Ross River fever often produces protracted and crippling arthralgia involving weight-bearing joints [76]. As mentioned, atypical cases of the mosquito-borne dengue can closely resemble sand fly fever. However, a dengue outbreak is usually more explosive in character than a phlebotomus fever epidemic. Rashes are also present more frequently (70%) in dengue patients as compared to sand fly fever patients [77]. Dengue virus infection and hantavirus-associated HFRS can be differentiated from scrub typhus by the presence of haemorrhagic manifestations, particularly bleeding from the gums [64,72]. Conjunctival suffusion has also been found more commonly in HFRS as compared to scrub typhus [64].

Summary
To summarize, dengue fever-like illnesses are usually rampant after rains. It may be difficult to differentiate DFLIs in the early course of the diseases. Diagnostic difficulties arise because of atypical manifestations and overlapping clinical features. Recognition of these diseases is important in order to make correct treatment decisions. Knowledge about the clinical, epidemiological, and laboratory features of individual diseases, as discussed in this review, may help in their early recognition.

Declaration of interest: No conflicts of interest.

References


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