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Review Article

Public Health Section

Kyasanur Forest Disease: A Narrative Review of Clinical Features, Transmission, Diagnosis and Public Health Strategies

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ABSTRACT

Kyasanur Forest Disease (KFD) is an emerging tick-borne viral haemorrhagic illness which is endemic to the Western Ghats of southern India, caused by the Kyasanur Forest Disease Virus (KFDV), a Flavivirus transmitted primarily by *haemaphysalis* ticks. KFD presents after a 3-8 day incubation with abrupt fever, headache, myalgia and gastrointestinal symptoms and may progress to haemorrhagic and neurological complications. Spread of KFD to other neighbouring states underscores challenges to control the disease. Diagnosis of KFD depends upon Reverse Transcription Polymerase Chain Reaction (RT-PCR) during early viremia, which is then followed by Immunoglobulin M (IgM) Enzyme-Linked Immunosorbent Assay (ELISA), with emerging isothermal amplification techniques showing promise for field use. Measures for prevention of KFD are vaccination, carcass disposal, avoidance of ticks, along with community education. Treatment is mainly supportive, which focuses on intensive care for severe cases. Strengthening of surveillance methods, vaccinations and integrated one health strategies are essential for reducing burden of the disease.

Keywords: Arboviral infections, Disease surveillance, Flavivirus, Haemorrhagic fever, Tick-borne diseases

INTRODUCTION

Kyasanur Forest Disease (KFD) is a tick-borne zoonotic illness which is caused by KFDV, a member of the genus Flavivirus [1,2]. Humans are infected primarily through bites of infected hard ticks (notably *Haemaphysalis* spinigera) and the clinical spectrum ranges from an acute febrile illness with myalgia, headache and haemorrhagic manifestations to neurological complications in a subset of patients [2,3]. The disease has been classically associated with the Western Ghats of southern India and remains focal to forest-fringe communities and people who enter endemic forested areas, where enzootic transmission cycles between ticks and mammalian hosts maintain the virus [4].

The KFD was first recognised in March 1957 following an epizootic of sudden deaths among monkeys and a cluster of severe febrile illness in humans in the Kyasanur forest range (Shimoga district), Karnataka; subsequent virological investigations isolated a novel virus that was shown to be related to the Russian spring-summer encephalitis complex [5,6]. Over the ensuing decades the disease has persisted in the Western Ghats and particularly in the last 15-20 years has been reported outside the original district into adjoining states (Kerala, Goa, Maharashtra and Tamil Nadu), highlighting both ecological change and gaps in control measures [7]. Control efforts have included an inactivated tissue-culture vaccine and tick-avoidance strategies, but recurring outbreaks, variable vaccine uptake/effectiveness and expanding geographic reports underscore ongoing public-health challenges [8,9].

Search Methodology

The present narrative review was developed through a comprehensive search of electronic databases which was inclusive of PubMed, Scopus, Web of Science and Google Scholar. Relevant literature which was published up to January 2026 was identified using combinations of keywords like KFD, tick-borne flavivirus, clinical features, pathogenesis, diagnosis, RT-PCR, vaccination, public health and one health approach. Peer-reviewed original research articles, review articles, surveillance reports, outbreak investigations and publications from recognised public health agencies were also considered. Additional relevant studies were identified through manual screening of reference lists of selected articles. Articles were selected based upon their relevance to scope of the present review with emphasis on recent advances in clinical presentation, laboratory diagnosis, prevention strategies as well as public health interventions related to KFD.

Clinical Features of Kyasanur Forest Disease (KFD)

The KFD typically begins after an incubation period of almost 3-8 days with a sudden, influenza-like febrile illness [10]. Early symptoms in such cases are high fever along with having severe frontal headache, which is further marked by myalgia and prostration, chills and often nausea, vomiting with other gastrointestinal complaints [11,12]. Conjunctival infection and photophobia are also frequently reported in patients suffering with KFD [11]. KFD is classically described as biphasic in a subset of patients an acute viremic phase followed, in few of the cases, by a second “toxic” phase with new or worsening symptoms [8,13,14].

Haemorrhagic manifestations (epistaxis, haematemesis, melena and other bleeding) were prominent in early descriptions of KFD and it still remains an important aspect of severe presentation in few cases [14,15]. Neurological involvement is also increasingly recognised which may occur in either phase presenting as altered sensorium, seizures, focal deficits, aseptic meningitis or postinfectious cerebellitis [16]. Other severe complications in cases of KFD are circulatory shock, myocarditis, acute respiratory distress and hyperinflammatory syndromes (for example, haemophagocytic lymphohistiocytosis) in only few selected patients [8,17]. Clinical features and complications of KFD are briefed in [Table/Fig-1] [8,10-18].

Aetiology and Pathogenesis of Kyasanur Forest Disease (KFD)

The KFD is caused by KFDV which is a zoonotic, tick-borne pathogen: humans are incidental hosts who typically acquire infection following the bite of infected hard ticks (primarily *Haemaphysalis spp.*), whereas non human primates (especially macaques) and various small mammals act as amplifying hosts in enzootic cycles [18]. Transmission of KFD is complex and supports both horizontal and

Category	Details	References
Incubation period	3-8 days	[10]
Initial Phase - early symptoms	- Sudden onset, influenza-like febrile illness - High fever - Severe frontal headache - Myalgia and prostration - Chills - Nausea, vomiting, gastrointestinal complaints - Conjunctival infection - Photophobia	[10-13]
Biphasic pattern	Seen in a subset of patients: acute viremic phase followed by second "toxic" phase with new/worsening symptoms	[8,14,15]
Haemorrhagic manifestations	Epistaxis, haematemesis, melena and other bleeding tendencies	[15,16]
Neurological involvement	Altered sensorium, seizures, focal neurological deficits, aseptic meningitis, postinfectious cerebellitis	[17]
Other severe complications	Circulatory shock, myocarditis, acute respiratory distress, hyperinflammatory syndromes (e.g., haemophagocytic lymphohistiocytosis)	[8,18]

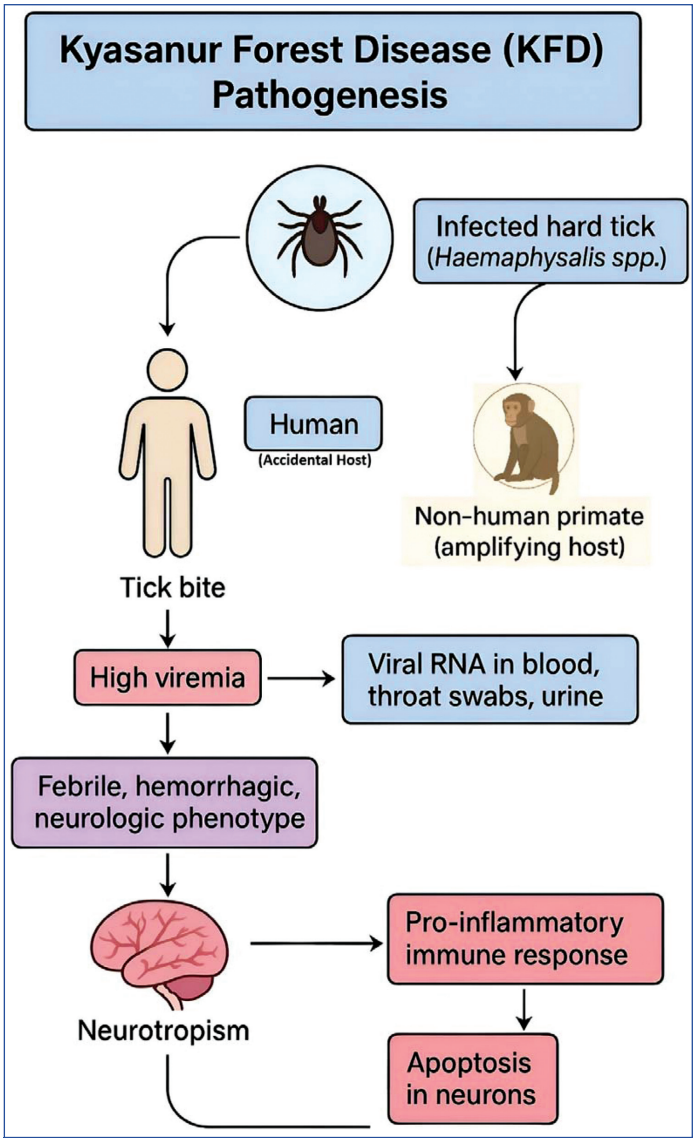
[Table/Fig-1]: Clinical features and complications of Kyasanur Forest Disease (KFD) [8,10-18].

vertical maintenance of the virus in populations of ticks [19]. KFDV is detected during various field and molecular studies in multiple *Haemaphysalis* species and importantly have provided direct evidence of transovarial (female tick → eggs → larvae) transmission, which further helps explain how KFDV persists in nature across seasons and in locations where mammalian hosts are intermittently available [2,19,20]. Human infection therefore often clusters in forested or peri-forested settings with high tick exposure [20].

Pathogenesis of KFD in humans begins with a short incubation period which is later followed by high viremia during the first week of illness; viral RNA is detectable in blood and, in some patients, in throat swabs and urine, aligning with the systemic spread seen in severe cases [18,21]. KFDV is pantropic but shows neurotropism in severe or late disease, inducing apoptosis in neurons of cerebrum and hippocampus in animal models and contributing to the neurological manifestations reported clinically [13,22]. Host innate and adaptive immune responses which include strong pro-inflammatory signalling are observed in infected cells which thereby mediate both viral clearance and immunopathology, thus together produce febrile, haemorrhagic and occasional neurologic phenotype of KFD [8,23]. Pathogenesis of KFD is depicted through [Table/Fig-2].

Host Immune Responses to Kyasanur Forest Disease Virus (KFDV)

Host immune responses play an important role into determination of severity and outcome of KFDV infection [24]. Following viral entry, the innate immune system is activated through pattern recognition receptors which detect viral RNA resulting into induction of interferon-mediated antiviral responses as well as production of pro-inflammatory cytokines [25]. KFDV infection also stimulates cytokines such as tumour necrosis factor-α, interleukin-6 and interferon-γ which contribute into viral clearance but it can also drive systemic inflammation associated with severe disease manifestations [24,25]. Adaptive immune responses further contribute into host defense usually through generation of neutralising antibodies directed against viral envelope proteins which limit viral replication which provides protective immunity following natural infection or vaccination [24,26]. In addition, T-cell mediated immune responses are believed to support viral control through cytotoxic activity, cytokine signalling although excessive immune activation can further contribute into immunopathological effects observed in severe cases of KFD [26].



[Table/Fig-2]: Pathogenesis of Kyasanur Forest Disease (KFD).

Phylogenetic and Sequence Similarity Insights into KFD Virus

Genomic sequencing as well as phylogenetic studies helped to provide insights into the dynamics of KFDV in the Indian regions [27]. Comparative analyses of viral isolates collected over several decades have also showed remarkably low genetic diversity among KFDV strains thereby suggesting that virus has remained genetically stable since its initial identification [27]. Whole-genome phylogenetic investigations analysing isolates from 1957 to recent year outbreaks have showed only minimal nucleotide divergence (approximately 1-2%) among strains thereby indicating slow evolutionary change as compared to many other types of Ribonucleic Acid (RNA) viruses [27,28]. Such genetic stability can thus partly explain persistence of similar clinical as well as epidemiological characteristics of KFD across successive outbreaks in endemic regions of Western Ghats [27].

More recent phylogeographic analyses which is done using genome sequencing of isolates from newly affected regions have provided evidence of regional lineage diversification associated with geographic spread of the virus [27]. Viral strains which are detected in areas like Kerala, Goa and Maharashtra cluster closely along with earlier Karnataka strains thus supporting hypothesis that emergence of KFD in these locations represents geographic expansion rather than emergence of genetically distinct variants [27,29,30]. Sequence analyses of structural genes particularly envelope glycoprotein gene have also showed very high sequence similarity (>99%) between historical as well as contemporary isolates suggesting that antigenic determinants remain largely

conserved [31]. All these findings highlight about importance of continued genomic surveillance for monitoring viral evolution to assess whether emerging mutations can influence transmission dynamics, virulence as well as effectiveness of vaccines in endemic regions of KFD [27,31].

Seasonal Fluctuations of Kyasanur Forest Disease (KFD)

The KFD shows a marked seasonal pattern which is closely linked having ecological dynamics of its tick vectors as well as climatic conditions in Western Ghats [32]. Human cases usually occur during dry season from November to May along with a peak incidence between January and March corresponding to period of heightened activity of the nymphal stage of *Haemaphysalis* ticks [32]. These nymphs are highly anthropophilic which further readily attach to humans when they enter forested environments for activities such as agriculture, firewood collection, grazing livestock [32]. Increased human-forest interaction during this season enhances risk of exposure to infected ticks as well as it facilitates spillover of the virus from wildlife reservoirs to human beings [32].

The seasonal dynamics of KFD are influenced by climatic factors usually rainfall and temperature which regulate tick population density and survival [29,32]. Endemic region studies such as Shivamogga (Karnataka), Wayanad (Kerala) and Goa have showed that transmission of KFD begins soon after cessation of monsoon in cases when environmental conditions favor proliferation of host-seeking nymphal ticks [29,33]. During monsoon period (which is June-October months), heavy precipitation suppresses tick activity also it further interrupts transmission cycles [33]. Conversely, post-monsoon period is usually characterised by moderate temperatures, reduced rainfall (<500 mm cumulative precipitation) which creates optimal conditions for survival of ticks as well as an increased risk for human exposure [33]. All these climatic correlations highlight about important role of environmental factors into shaping of seasonal epidemiology of KFD [33].

In addition to climatic determinants, life cycle of the vector ticks also contributes into seasonal pattern of KFD transmission [34]. After the monsoon season; larval ticks emerge which further feed on small mammals and birds subsequently moulting into nymphs which become highly active during winter as well as early summer months [34,35]. This stage is usually significant because nymphs have broader host range frequently infest humans thereby acting as principal stage responsible for transmission of KFD virus [34]. Studies which focus on tick ecology in endemic areas have showed that density of questing nymphal ticks increases substantially from December to February paralleling rise in human cases during same time period [34,35]. Understanding all these seasonal fluctuations is an essential aspect for timely planning preventive interventions inclusive of vaccination campaigns before the transmission season, intensified tick surveillance, community awareness programmes and early warning systems based on ecological indicators such as monkey deaths [35].

Emerging Diagnosis of Kyasanur Forest Disease (KFD)

Diagnosis of KFD mainly depends upon combination of clinical suspicion in the appropriate epidemiological setting and laboratory confirmation [36]. During the early (viremic) phase, detection of KFD viral RNA by Reverse-Transcription PCR (RT-PCR) from acute-phase blood (serum or plasma) is the diagnostic method of choice because it directly demonstrates the virus and gives the highest sensitivity early on [36,37]. Laboratory-based real-time RT-PCR and nested RT-PCR assays are developed for NS5/non coding regions which are widely used in reference laboratories and were shown to identify cases that serology alone would miss in the acute period of KFD [38].

Serology becomes useful later in illness: anti-KFDV Immunoglobulin M (IgM) {capture Enzyme-Linked Immunosorbent Assay (ELISA)} usually appears after the viremic phase and is employed for diagnosis from about day 5-7 onward or on convalescent sera; paired acute and convalescent phase sera can thereby confirm recent infection when PCR is negative [39]. Routine isolation of virus is possible but is slow and requires high-containment facilities, so it is rarely used for rapid diagnosis of case scenario [39]. Cross-reactivity with other flaviviruses also can affect some serological assays, so interpretation must consider clinical timeline of disease along with local arboviral epidemiology [40]. Due to occurrence of KFD in remote, forested areas, recent work has focused on bringing diagnostics closer to the field: validated point-of-care molecular platforms (for example, Truenat KFD) and simplified isothermal/dry-down RNA detection methods have shown promise for faster, decentralised testing with reasonable agreement to laboratory RT-PCR [40,41].

Emerging diagnostic innovations for KFD include Reverse Transcription-Polymerase Spiral Reaction (RT-PSR) combined with magnetic bead-based RNA extraction in a dry-down format [42]. This portable and equipment-light method performs isothermal amplification at 63°C for about 60 minutes, detecting as few as 10 viral RNA copies per reaction [42]. Results are visually interpreted using colour change via Hydroxy Naphthol Blue dye [42]. Its performance and lack of cross-reactivity with other similar flaviviruses make it promising to be used in low-resource, field-based settings [42].

Reverse-Transcription Loop-Mediated Isothermal Amplification (RT-LAMP) assay can be used for detection of KFDV [43]. In this method viral RNA is amplified at a constant temperature in one step (60 min at 63°C) and has a detection limit of just 10 copies [43]. Evaluated across human, primate and tick samples, RT-LAMP demonstrated 100% concordance with conventional TaqMan qRT-PCR and showed high specificity with no cross-reactivity to other haemorrhagic/flaviviruses [43,44]. All these makes RT-LAMP, an ideal for rapid, high-throughput screening during outbreaks, especially where laboratory infrastructure is minimal [43]. Diagnostic methods for KFD are depicted in [Table/Fig-3] [36,37,39-44].

Differential Diagnosis of Kyasanur Forest Disease (KFD)

The KFD shares several clinical features along with other acute febrile illnesses endemic to tropical regions particularly dengue, leptospirosis and malaria, which often complicates early clinical diagnosis [14]. Patients having KFD usually present with abrupt onset of high fever, severe headache, myalgia, gastrointestinal symptoms and thrombocytopenia features which significantly overlap with those observed in dengue and other viral haemorrhagic fevers [14,45]. In dengue infection, however, patients usually show retro-orbital pain, rash and marked haemoconcentration due to plasma leakage which are less commonly observed in KFD [45]. Similarly, haemorrhagic manifestations can occur in both diseases but KFD often shows a biphasic illness pattern which may progress to neurological complications such as meningitis or encephalitis in the later stage which are less usual in uncomplicated-type of dengue infection [14]. Laboratory confirmation which is done using molecular assays inclusive of RT-PCR, virus-specific serological testing are essential for distinguishing KFD from similar arboviral infections in endemic areas [41].

Leptospirosis and malaria are also important considerations in differential diagnosis of KFD because they usually present with acute febrile illness which is accompanied by headache, myalgia and systemic involvement [46]. Leptospirosis can also mimic KFD having symptoms such as fever, myalgia and conjunctival suffusion. However severe cases often show jaundice, renal impairment and pulmonary haemorrhage which are not characteristic findings in KFD [46,47]. Malaria, usually caused by

Diagnostic method	Principle/ Target	Timing and use	Advantages	Limitations/ Notes	References
RT-PCR (Conventional, real-time, nested)	Detects KFDV RNA (e.g., NS5/non coding regions)	Early (viremic phase, acute-phase serum/ plasma)	Highest sensitivity early; confirms infection before antibodies appear; identifies cases missed by serology	Requires laboratory setup; limited field availability	[36,37]
Anti-KFDV IgM capture ELISA	Detects virus-specific IgM antibodies	From ~Day 5-7 onwards; useful in convalescent sera	Confirms recent infection when PCR is negative; easy to implement	Cross-reactivity with other flaviviruses; less useful early	[39]
Virus isolation	Culture of KFDV from clinical sample	Any stage; research/ reference use	Definitive proof of infection	Slow; requires high-containment (BSL-4) facilities; not suitable for rapid diagnosis	[39]
Point-of-care molecular platforms (e.g., Truenat KFD)	Portable RT-PCR-based detection	Early phase; field-based	Faster, decentralised testing; reasonable agreement with lab RT-PCR	May have slightly reduced sensitivity vs. reference lab methods	[40,41].
RT-PSR (Reverse Transcription–Polymerase Spiral Reaction) with Magnetic bead RNA extraction	Isothermal amplification at 63 °C (~60 min), visual colour change (Hydroxy naphthol blue)	Early phase; low-resource settings	Portable, equipment-light; detects as few as 10 RNA copies; no cross-reactivity	New/emerging method; limited large-scale validation	[42]
RT-LAMP (Reverse Transcription Loop-Mediated Isothermal Amplification)	Isothermal amplification at 63 °C (60 min)	Early phase; human, primate and tick samples	Detection limit 10 copies; 100% concordance with qRT-PCR; high specificity; field-friendly	Requires trained personnel for primer preparation; emerging method	[43,44]

[Table/Fig-3]: Diagnostic methods for Kyasanur Forest Disease (KFD) [36,37,39-44].

Plasmodium falciparum can present with fever, thrombocytopenia along with systemic complications that resemble early KFD, but periodic fever patterns, splenomegaly as well as detection of parasites on peripheral blood smear or rapid antigen tests aid in differentiation [48]. Epidemiological history is very crucial aspect in distinguishing these infections; exposure to forested areas having known tick activity as well as reports of monkey deaths are strong epidemiological indicators suggestive of KFD [47]. Therefore, integration of clinical presentation, exposure history and confirmatory laboratory investigations is also critical for accurate diagnosis as well as appropriate management in endemic regions [47]. The differential diagnoses of KFD are summarised in [Table/Fig-4] [14,41,45-48].

Differential diagnosis	Similarity	Differentiating feature	References
Dengue fever	Acute febrile illness, headache, myalgia, gastrointestinal symptoms, thrombocytopenia, haemorrhagic manifestations	Retro-orbital pain, skin rash, marked haemoconcentration due to plasma leakage; neurological complications and biphasic course less common in uncomplicated dengue	[14,45]
Leptospirosis	Fever, myalgia, headache, systemic involvement	Conjunctival suffusion, jaundice, renal impairment, pulmonary haemorrhage more typical; these are uncommon in KFD	[46,47]
Malaria	Fever, thrombocytopenia, systemic symptoms	Periodic fever pattern, splenomegaly, detection of parasites on peripheral smear or rapid antigen tests	[48]
Viral haemorrhagic Fever	Fever, thrombocytopenia, bleeding manifestations	KFD often shows biphasic illness and may progress to neurological complications like meningitis or encephalitis; epidemiological link to tick exposure and monkey deaths	[14,41]

[Table/Fig-4]: Differential diagnosis of Kyasanur Forest Disease (KFD) [14,41,45-48].

Vaccination and Community-Based Interventions for Prevention of KFD

Vaccination has traditionally used as a preventive strategy against KFD [49]. Field studies which are conducted in endemic districts of Karnataka have demonstrated that inactivated KFD vaccine provides moderate protection when administered before transmission season usually when the recommended primary doses, booster schedules are completed [14,30]. Observational studies have further suggested about effectiveness of vaccine which improves with timely booster doses as well as adequate coverage in high-risk populations inclusive of forest workers and tribal communities residing in endemic regions [35,50]. However, challenges such as incomplete vaccination schedules, delayed booster administration as well as logistical barriers in remote forest areas have been reported for reducing overall protective impact of vaccination program [35,47]. The formalin-inactivated vaccine also offers effectiveness with two doses and have more effectiveness with an additional third dose [49].

During 2022 outbreak of KFD in Shivamogga, Karnataka, it was found that no statistically significant difference is there in disease incidence between vaccinated and unvaccinated individuals [30]. These observations suggest that vaccine coverage, timing, matching of strain, logistical challenges, or waning immunity can further undermine protective benefits, thus it highlights an urgent need to revisit vaccination strategies and improving surveillance of vaccine effectiveness [30,49].

Complementing efforts of immunisation, environmental and community-based interventions have vital roles in disease prevention [30]. The study from Shivamogga emphasised the importance of timely and safe disposal of monkey carcasses, since dead monkeys may harbour infected ticks which can further pose an indirect risk to nearby populations [30]. Additionally, targeted behaviour change communication like wearing full-body, light-coloured clothing, using tick repellents such as Dimethylphthalate (DMP)/N -Diethyl Phenylacetamide (DEPA) oil, conducting self-checks for ticks after exposure to forest area and avoiding areas with recent deaths of monkey is essential in endemic communities [51,52]. Integrating all these practices with active surveillance, rapid monkey necropsy and community education especially in vulnerable tribal populations relying on forests supports a multidisciplinary One Health approach to mitigate risk of having KFD [52,53].

Management of Kyasanur Forest Disease (KFD)

Treatment of KFD is currently mainly supportive and symptomatic because there is no proven, specific antiviral therapy [54]. Early care focuses on fluid balance, antipyretics, analgesia, close monitoring of haemodynamic status, blood counts and prompt management of bleeding [55]. Supportive care protocols also emphasise about regular monitoring of platelet counts, liver enzymes and renal parameters as haematological abnormalities such as thrombocytopenia, leukopenia are usually observed during acute phase of illness [17,40]. Careful assessment of hydration status as well as maintenance of electrolyte balance are important for prevention of complications associated having systemic inflammatory responses and gastrointestinal symptoms

[40]. In endemic regions, early hospitalisation, close observation during first week of illness are usually recommended for promptly identifying progression to severe KFD disease in patients [14,40]. Routine broad-spectrum antibiotics are not indicated unless a secondary bacterial infection is suspected in such cases; most uncomplicated patients improve with conservative in-patient care and observation [14].

Targeted supportive interventions along with intensive monitoring for haemorrhagic complications, correction of coagulopathy and intensive-care management for respiratory failure or shock are required in severe cases of KFD [8,33]. Patients having severe manifestations such as circulatory shock, significant haemorrhage, neurological complications can require admission to intensive care units for advanced supportive management of KFD [23,56,57]. Critical care measures are inclusive of haemodynamic stabilisation, blood product transfusion for severe bleeding, ventilatory support in cases of respiratory distress as well as close neurological monitoring [57]. Early recognition of complications, timely escalation of care has been reported for improvement of clinical outcomes in hospitalised patients having severe KFD [18,57]. Neurological involvement in KFD further requires early Intensive Care Unit (ICU) care, seizure control and neurocritical support; adjunctive immunomodulatory therapies such as corticosteroids [23,56]. Research must be carried out on broad-spectrum antivirals and immunotherapies, but at present clinical management remains supportive [56].

The overall case fatality rate of KFD has been reported to range in between 3% and 5% in most outbreaks although outcomes vary depending on severity of disease as well as access to timely medical care [58]. Most patients having uncomplicated illness recover completely with supportive treatment whereas severe cases presenting with neurological involvement, haemorrhagic complications can further require prolonged hospitalisation, intensive monitoring [33,58]. Strengthening of early detection, supportive management protocols thereby remains an essential aspect into improving patient outcomes in endemic regions of KFD cases [2,21].

Repurposed and Investigational Therapeutic Approaches

Due to the absence of a specific antiviral therapy for KFDV; recent research articles have further explored potential usage of repurposed antiviral drugs which helps targeting conserved flaviviral replication pathways [59]. Broad-spectrum antiviral agents which are inclusive of ribavirin which has shown inhibitory activity against several RNA viruses have been evaluated experimentally for their possible utility against KFDV and related tick-borne flaviviruses, although convincing clinical evidence still remains limited [18,59]. Additionally, studies investigating about antiviral compounds targeting viral RNA-dependent RNA polymerase as well as other replication-associated proteins have suggested that drugs originally developed for other viral infections can interfere with flaviviral replication [14,60]. High-throughput screening of existing drug libraries further identified several small-molecule inhibitors having activity against related flaviviruses thus highlighting about potential of drug repurposing strategies in identification of candidate therapeutics [61]. All these approaches remain largely experimental, continued research on repurposed antivirals as well as host-targeted therapies can provide promising directions for development of effective treatments for severe cases of KFD [14,61].

CONCLUSION(S)

The KFD remains an important public health challenge in India, particularly in forest living communities of the Western Ghats.Despite advances in diagnosis methods, vaccination strategies, preventive options, recurrent outbreaks and geographic spread highlight gaps in control measures. Supportive care remains the main aspect of management, as no specific antiviral exists. Enhancing disease surveillance, improving vaccine effectiveness and implementing integrated tick-control strategies alongside community-based interventions are crucial for effective disease control. A coordinated One Health approach is thus, essential for reducing disease burden to mitigate future outbreaks in endemic and emerging regions.

Authors contribution: The AA contributed to the conceptualisation, literature review, data collection and drafting of the manuscript. AS provided supervision, critically revised the manuscript for important intellectual content and approved the final version. BS contributed to literature review, data interpretation, manuscript drafting and formatting of tables and figures. All authors read and approved the final manuscript.

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