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The Attenuation Paradox in Bundibugyo Virus Disease: Case Fatality, Ascertainment, and Delayed Outbreak Recognition

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ABSTRACT

Bundibugyo virus has caused three recognised outbreaks, and each was recognised late. The usual reading of that record treats the delay as a failure of investment, diagnostics or governance. This review sets out a partly mechanistic alternative: the low case fatality ratio observed early in a Bundibugyo virus outbreak is itself one of the conditions that produces the delay, and the delay in turn depresses the observed ratio further. The evidence is organised around three comparison tables: the burden, severity and recognition interval of each outbreak; the presenting clinical features set against a matched Ebola virus series; and the point at which species assignment failed in each event. A schematic draws the relationships together. Two findings emerge. The clinical syndrome does not discriminate between species: haemorrhage was documented in roughly a quarter of confirmed patients in both the 2007–08 Bundibugyo series and a 2014 Ebola virus series, so the presenting picture cannot carry the explanatory weight often placed on it. The discriminating failures are instead genomic and diagnostic, and the early case fatality ratio operates as a surveillance trigger that is biased downward precisely when ascertainment is narrowest. The 2026 epidemic, in which the confirmed ratio rose from 12.1% to 44.3% while the virus doubled every 5.5 days, fits this account better than it fits a change in virulence. A fatality ratio from the first weeks of a filovirus outbreak should therefore be read as an index of surveillance maturity rather than of danger, and escalation should be governed by species-inclusive confirmation.

مفارقة انخفاض الضراوة في مرض فيروس بونديبوغيو: معدل وفيات الحالات، ورصد الحالات، وتأخر اكتشاف تفشّي المرض

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الكلمات المفتاحية:
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الملخص

تسبب فيروس بونديبوغيو في ثلاث فاشيات معروفة، وجرى التعرّف على كل منها متأخرًا. وتُفسَّر هذه التأخّرات عادةً بأنها قصور في الاستثمار أو التشخيص أو الحوكمة. تطرح هذه المراجعة تفسيرًا بديلًا ذا طابع آلي جزئيًا: إن انخفاض معدل إماتة الحالات المرصود مبكرًا في فاشية فيروس بونديبوغيو هو أحد الشروط التي تُنتج التأخّر، وأن التأخّر بدوره يزيد من خفض المعدل المرصود. نُظِّمت الأدلة حول ثلاثة جداول مقارِنة: العبء والشدة وفترة التعرّف في كل فاشية؛ والسمات السريرية مقارنةً بسلسلة مناظِرة لفيروس إيبولا؛ والنقطة التي أخفق عندها تحديد النوع، مع مخطط يجمع العلاقات. وتبرز نتيجتان. الأولى أن المتلازمة السريرية لا تميّز بين النوعين: فقد وُثِّق النزف لدى نحو ربع المرضى المؤكَّدين في كل من سلسلة بونديبوغيو 2007–2008 وسلسلة فيروس إيبولا لعام 2014، ومن ثَم لا تحمل الصورة السريرية الثقل التفسيري الذي يُسنَد إليها غالبًا. والثانية أن مواطن الإخفاق المميِّزة جينومية وتشخيصية، وأن معدل الإماتة المبكر يعمل كمُحفِّز للترصّد يتحيّز نحو الانخفاض تحديدًا حين يكون تحرّي الحالات في أضيق نطاقاته. ووباء 2026، الذي ارتفع فيه المعدل المؤكَّد من 12.1% إلى 44.3% بينما تضاعف الفيروس كل 5.5 أيام، يتسق مع هذا التفسير أكثر من اتساقه مع تغيّر في الفوعة. ولذلك ينبغي قراءة معدل الإماتة في الأسابيع الأولى من فاشية فيروس خيطي بوصفه

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مؤشرًا على نضج الترصّد لا على الخطورة، وأن يكون التصعيد محكومًا بالتأكيد الشامل للأنواع.

1. Introduction

Bundibugyo virus is one of the orthoebolaviruses pathogenic for humans, and it is the least studied of them. It was identified in late 2007 during an outbreak of haemorrhagic fever in Bundibugyo District in western Uganda, where laboratory investigation of the first 29 suspect specimens by antigen capture and immunoglobulin assays, followed by random-primed pyrosequencing, revealed a species sufficiently divergent from all previously recognised ebolaviruses that the investigators flagged the finding as consequential for the design of future diagnostic assays [1]. The virus reappeared in Isiro in the Democratic Republic of the Congo in 2012, one of four filovirus outbreaks in the region that year [2], and then again in Ituri Province in 2026 in an epidemic that was declared a public health emergency of international concern and that became the largest on record for the species [3,4].

Research on this virus has concentrated in two places. The first is the countermeasure gap: no vaccine or therapeutic is licensed for Bundibugyo virus, although recombinant vaccines and cross-reactive monoclonal antibodies show protection in animals, and several of the broadly neutralising antibodies now in development were isolated from survivors of this species [5–7]. The second is preparedness architecture, where recent commentary has proposed auditing diagnostic, vaccine and therapeutic systems for species breadth rather than for coverage of Ebola virus alone [8], and where scoping work has mapped the asymmetry of the evidence base and found that fewer than a quarter of the studies retrieved under a Bundibugyo search actually concern the species [9]. Alongside these, the 2026 epidemic has generated rapid genomic characterisation [3,10,11], transmission modelling [12], and a quantitative reconstruction of how long the outbreak ran before anyone knew it was running [13].

None of this work asks whether the severity of the disease is causally related to the lateness of its recognition. Every outbreak of Bundibugyo virus disease has been characterised, at the moment decisions about escalation were being taken, by a case fatality ratio lower than the figure clinicians and surveillance officers associate with Ebola disease, and every outbreak has been confirmed late. Treating that coincidence as incidental leaves a mechanism unexamined; treating it as causal runs into the fact that the 2026 epidemic eventually reported a ratio near 47% [4], which is not a mild disease by any measure. Resolving the tension requires separating the ratio observed early in an outbreak from the ratio established after it. This review organises the reported evidence on Bundibugyo virus disease by the relationship between observed severity and recognition interval, in order to determine whether low early case fatality functions as a cause of detection delay, a consequence of it, or both.

The evidence considered here was identified by searching OpenAlex and Semantic Scholar, with the medRxiv and Research Square preprint servers, from January 2007 to November 2026, combining Bundibugyo, orthoebolavirus and filovirus with terms for case fatality, detection delay, ascertainment and diagnostic assay, and by screening the reference lists of the reviews retrieved. Primary reports of the three outbreaks, clinical series reporting features at the level of the individual patient, and quantitative analyses of epidemic growth and case ascertainment were prioritised. Comparative material on Ebola virus was admitted only where it was needed to test whether a feature attributed to Bundibugyo virus is specific to that species. Preprints are cited where they carry data not available elsewhere and are identified as such throughout. This is a narrative review, and no attempt was made at exhaustive retrieval.

2. Severity and recognition across three outbreaks

The three outbreaks differ in almost every respect that matters epidemiologically, which makes the one feature they share worth attention. Table 1 sets the reported burden, severity and recognition interval of each event side by side.

Table 1. Reported burden, severity and recognition interval across the three recognised outbreaks of Bundibugyo virus disease. Figures are as reported in the cited sources.

Feature	Uganda, 2007–08	Democratic Republic of the Congo, 2012	Democratic Republic of the Congo and Uganda, 2026
Location	Bundibugyo District, western Uganda	Isiro, Orientale Province	Ituri Province, extending to five provinces, with locally transmitted cases in Uganda
Cases	192 suspected; 42 laboratory-confirmed; 74 probable [14]. A separate clinical series reports 93 putative and 56 confirmed [15]	57–59 [4]	4,566 laboratory-confirmed at 11 August [4]; 8,728 cases at 6 October [16]
Deaths	37 [15]	29–34 [4]	2,128 at 11 August [4]; 4,205 at 6 October [16]

Feature	Uganda, 2007–08	Democratic Republic of the Congo, 2012	Democratic Republic of the Congo and Uganda, 2026
Reported case fatality ratio	25% in the confirmed clinical series [15]; 34% in the outbreak investigation [14]; ≈40% among the 56 laboratory-confirmed cases [17]	34–58% [4]	12.1% early, rising to 44.3% [13]; ≈47% at 11 August [4]
Interval to recognition	Laboratory confirmation lagged outbreak verification by three months [14]	Not separately reported in the sources retrieved	Back-calculated onset 19 April; 16 days before the alert and 25 days before laboratory confirmation [13]
Early growth	Doubling time 15–18 days (retrospective) [13]	Doubling time 15–18 days (retrospective) [13]	0.1261 per day; doubling time 5.50 days (95% CI 4.24–7.83) [13]
Reproduction number	Not reported	Not reported	1.71, range 1.4–2.1 [4]; 3.67, 95% highest-density interval 2.0–5.7 [12]
How the species was assigned	Classical antigen capture and ELISA, then random-primed pyrosequencing; a new species [1]	Genomic analysis across four concurrent outbreaks [2]	An Ebola-virus-focused assay was negative; broader testing and sequencing were required [8]; 22 genomes form a separate cluster [3,10]

CI, confidence interval. Entries marked "not reported" indicate that the parameter was not given in the sources retrieved for this review, not that it is unobtainable.

Read across the rows, the table gives no support to the idea of a mild virus. The reported case fatality ratio spans 25% to 58%, and the 2026 epidemic settled near the upper part of that range [4,13]. A pathogen that kills close to half of those it infects cannot be called attenuated. What the table does show is that the figure available at the moment of decision was consistently at the lower end of the range. In 2007–08 the investigation reported that Bundibugyo virus was less fatal than the ebolaviruses that had previously caused outbreaks in the region, and in the same sentence recorded that laboratory confirmation had lagged outbreak verification by three months [14]. A dedicated assessment of that outbreak identified 131 cases, 44 suspect, 31 probable and 56 confirmed, attributed the prolonged person-to-person transmission to the same three-month lag, and concluded that filovirus surveillance, reporting and diagnostics in endemic African settings required improvement [18]. In 2026 the confirmed ratio began at 12.1% and only later rose to 44.3% [13]. The severity eventually established was therefore not the severity that informed the response.

The growth row sharpens the cost of that gap. The 2026 epidemic doubled every 5.50 days, roughly threefold faster than the 15 to 18 days reconstructed for the earlier outbreaks, and back-calculation placed transmission onset 16 days before the alert and 25 days before laboratory confirmation [13]. Twenty-five days at a doubling time of 5.5 days is more than four doublings. The two reproduction-number estimates in the table disagree substantially, 1.71 against 3.67 [4,12], and the disagreement is unresolved: the lower figure derives from a narrative synthesis of situation reports and the higher from a compartmental model fitted to Ituri surveillance data that explicitly accounts for undetected community infection. The discrepancy is itself informative, because models that allow for unobserved transmission recover faster spread than summaries that do not.

3. Does the clinical syndrome explain the delay?

The most intuitive explanation for repeated late recognition is that Bundibugyo virus disease looks different from Ebola virus disease, and in particular that it bleeds less, so that case definitions anchored on haemorrhage fail to capture it. This explanation is widely implied and worth testing directly. Table 2 places the only detailed Bundibugyo clinical series against a comparably reported Ebola virus series.

Table 2. Presenting clinical features in the 2007–08 Bundibugyo virus series compared with a 2014 Ebola virus series from the Democratic Republic of the Congo. The two studies used different instruments and are not formally comparable; the comparison is indicative.

Presenting feature	Bundibugyo virus, Uganda 2007–08 (26 hospitalised confirmed patients) [15]	Ebola virus, Democratic Republic of the Congo 2014 (25 admitted confirmed patients) [19]	Discriminates between the two species?
Non-bloody diarrhoea	81%	Gastrointestinal symptoms in 76% (≥1 symptom)	No
Severe headache	81%	Not reported in comparable form	Cannot be assessed
Asthenia	77%	Not reported in comparable form	Cannot be assessed
Any haemorrhagic manifestation	7 of 26 (27%), of whom 6 died	28% (≥1 symptom)	No
Case fatality in the series	25%	48% in the admitted series; 74.2% outbreak-wide	Yes
Authors' own characterisation	"Clinical findings are congruous with previously reported" Ebola haemorrhagic fever [15]	"Clinical features were mainly generic" [19]	—

Dashes indicate that no corresponding figure was reported. Percentages are as published and have not been recalculated.

The comparison does not support the intuition. Haemorrhagic manifestations were documented in 7 of 26 confirmed Bundibugyo patients, about 27%, against 28% of the admitted Ebola virus patients [15,19]. Both series describe a predominantly gastrointestinal and

constitutional illness, and both sets of authors said so explicitly: the Bundibugyo investigators concluded that their clinical findings were congruous with previously reported Ebola haemorrhagic fever, and the Ebola virus investigators described their patients' features as mainly generic [15,19]. If a case definition weighted toward bleeding under-triggers for Bundibugyo virus, it under-triggers for Ebola virus to a very similar degree. A separate analysis of the same Ugandan outbreak reached the same conclusion from the laboratory-confirmed cases alone: signs and symptoms were largely non-specific and resembled those of outbreaks caused by Zaire and Sudan ebolaviruses, while the proportion of deaths among the 56 confirmed patients was about 40% [17]. The syndrome is non-specific, but it is not differentially non-specific, and the data now available do not sustain an argument resting on a distinctive Bundibugyo presentation.

Two caveats qualify this conclusion. The Bundibugyo series comprises 26 hospitalised patients from a single outbreak, recruited under field conditions that its own authors describe as constraining data quality, and the comparator series comprises 25 patients managed in two treatment centres under a different protocol, in another country and six years later [15,19]. Neither was designed for cross-species comparison, and the apparent concordance in haemorrhage frequency could be an artefact of differing ascertainment of a symptom that is notoriously variably recorded. The available clinical evidence therefore provides no support for a distinctive Bundibugyo syndrome, although it does not exclude one. Removing that explanation directs attention to the factors that do differ by species.

4. Species-restricted confirmation as the operative filter

The genome differs by species, and so, in consequence, does the behaviour of the assays. Bundibugyo virus differs from the species against which vaccines and diagnostics have been matched by as much as 43% of its genome sequence [6], and the original description of the virus warned that this divergence carried implications for assay design [1]. Table 3 traces what happened at the point of confirmation in each outbreak.

Table 3. The point at which species assignment failed or was delayed in each outbreak, and the method ultimately required.

Outbreak	Method first applied	Method ultimately required	Reported consequence
Uganda, 2007–08	Antigen capture and IgM/IgG ELISA on the initial 29 suspect specimens [1]	Random-primed pyrosequencing [1]	A new species was identified; the authors warned that the divergence had implications for future assay design [1]. Confirmation lagged verification by three months [14]
Democratic Republic of the Congo, 2012	Not separately reported	Genomic analysis distinguishing filoviruses across four concurrent outbreaks [2]	Species assignment among co-circulating filoviruses
Democratic Republic of the Congo and Uganda, 2026	An Ebola-virus-focused molecular assay, which returned a negative result [8]	Broader molecular testing with genomic sequencing [8]; 22 genomes defining a distinct cluster [3]	25 days from back-calculated onset to laboratory confirmation [13]; cross-border spread, including an importation detected in Kenya [11]
Standing recommendation	—	Pan-orthoebolavirus assays with decentralised diagnostic and sequencing capacity [3,8,20]	Not yet implemented at the time of writing [5,9]

Entries drawn from preprints are cited as such in the reference list and should be weighted accordingly.

The pattern across the three rows is a single failure mode recurring over nineteen years. In 2007 classical immunoassays on the first specimens could not resolve the agent and a sequencing approach was needed [1]. In 2026, with the species long since described, an Ebola-virus-focused molecular assay was applied to the index testing and returned a negative result, and only broader molecular testing with sequencing established what was circulating [8]. A recent synthesis states the general form of the problem plainly, concluding from the 2007–08 experience that assays optimised for known filoviruses can miss divergent ebolaviruses, and arguing that Bundibugyo virus cannot be reduced to an Ebola-virus-like model [5]. The recommendation that follows, pan-orthoebolavirus assays with decentralised sequencing capacity, is now made by genomic, diagnostic and preparedness analyses alike [3,8,20]; it has become uncontroversial, and it has yet to change practice.

Published work is almost entirely descriptive about the magnitude of this failure. It is established that an Ebola-virus-focused assay returned a negative result in 2026; it is not established how many assays in routine use would do the same, by how much, or at which nucleotide positions. No study retrieved for this review has systematically evaluated the inclusivity of published orthoebolavirus assays against Bundibugyo virus genomes. The claim that diagnostics are species-restricted is therefore well evidenced as an anecdote and poorly evidenced as a quantity.

5. A self-reinforcing severity–recognition loop

Taken together, the three tables describe something more specific than a list of preparedness failures. Table 2 removes the clinical syndrome as a discriminator. Table 3 identifies species-restricted

confirmation as the operative filter on who enters the confirmed case count. Table 1 shows that the ratio of deaths to confirmed cases, early in an outbreak, is low, and that recognition is late. These three observations connect, and the connection is circular.

The mechanism runs as follows. When filovirus disease is not suspected and the available assay is matched to the wrong species, the cases that do get confirmed are not a random sample: they are the most severe, the most clustered, or those sampled late enough for the diagnostic question to have been reopened. This is the surveillance pyramid problem in its standard form, in which the composition of the confirmed denominator determines the precision and the accuracy of any severity estimate derived from it [21], compounded by the well-described downward bias that the direct ratio of cumulative deaths to cumulative cases carries early in an epidemic before case-to-death intervals have elapsed [22,23]. The resulting figure is then read, by the people deciding whether to escalate, as a measure of how dangerous the event is. A low figure does not meet the threshold that the word Ebola carries, escalation is deferred, suspicion stays low, and the narrow ascertainment that produced the low figure persists. Transmission continues through the interval. Only when the case base is large enough, and the response mature enough, does ascertainment broaden and the observed ratio converge on the true one, by which point the epidemic has amplified. Figure 1 sets out this loop with the reported values at each stage.

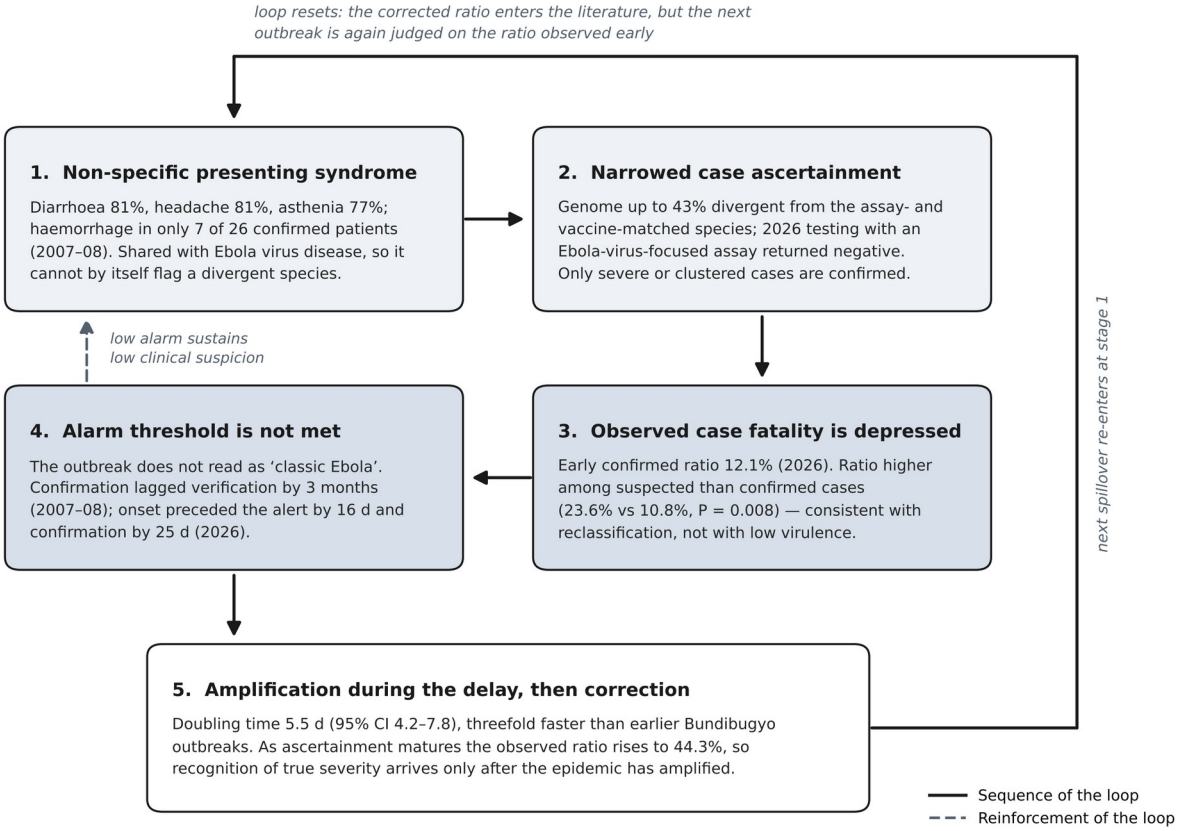


Fig. 1. The severity–recognition feedback loop in Bundibugyo virus disease. Solid arrows trace the sequence; the dashed arrow marks the limb by which a low alarm state sustains the narrow ascertainment that produced it. Values are those reported in the sources cited in Tables 1 to 3.

The strongest evidence that this loop is real, rather than merely plausible, comes from the internal structure of the 2026 data. If the rising ratio reflected a virus becoming more virulent, the ratio among confirmed cases should have risen independently of case classification. Instead, on 21 May the ratio was higher among suspected than among confirmed cases, 23.6% against 10.8%, and the difference was statistically significant; the authors read this as progressive reclassification rather than increasing virulence [13]. That is the signature the loop predicts: deaths were occurring among people the system had not yet confirmed, so the confirmed ratio understated severity for as long as confirmation lagged. Independent support comes from an analysis of fatality ratios by health zone, which found that a difference of only 1.25-fold in the ratio of death ascertainment to case ascertainment between provinces would suffice to generate the apparent geographical contrast in lethality, and concluded that zone-level ratios were not yet interpretable as fatality at all [16]. An appraisal of the 2026 alert chain places the constraint on the surveillance side rather than in the data: ten days separated the initial alert of 5 May from laboratory confirmation on 15 May, although several non-laboratory signals were already active, which identifies the interpretive integration of concurrent signals as the limiting step [24].

The loop also explains why the problem recurs rather than resolving. Each outbreak eventually produces a corrected, higher ratio, and the species is now routinely described with a range of 25% to 51% [9]. But the next outbreak is judged on the ratio observed in its own first weeks, which is again depressed by the same mechanism. Accumulating an accurate species-level figure does not protect the next response, because the next response reads a different number. Low observed severity is therefore a hazard, and the claim here concerns surveillance rather than virology.

One further implication follows from the modelling work. If recognition is the binding constraint, then interventions acting on time-to-detection should dominate interventions acting on transmission once detected. That ordering is what the one available compartmental analysis of this epidemic reports: shortening the time to isolation averted the largest share of infections, 73.4%, ahead of reducing leakage from isolation facilities or re-isolating those who had left [12]. That study is unreviewed and its parameter choices are contestable, but its ranking is consistent with the loop described here.

6. Interpretation and alternative explanations

The account set out here remains an explanatory hypothesis, supported by converging evidence from a clinical comparison, a diagnostic history and two quantitative analyses of the same epidemic. The evidence base is thin and unevenly distributed: two of the three outbreaks involved fewer than 200 cases, the 2012 event has almost no separately reported clinical or detection-interval data, and the detection intervals in Table 1 are drawn from two outbreaks rather than three. Much of the quantitative material concerning 2026 is preprint or situation-report derived and has not completed peer review [11,12,16]; the central back-calculation of the detection interval is peer-reviewed [13], and the argument stands without the preprints, although several of the supporting figures do not. Case and death counts from an active epidemic also revise, and the two cut-offs reported here, 11 August and 6 October 2026, differ substantially [4,16].

The direction of causation has not been established, only its plausibility and its consistency with the data. A rival explanation holds that delay in all three outbreaks is sufficiently explained by conflict, displacement, weak laboratory infrastructure and the absence of prepositioned capacity, all of which are documented for eastern Democratic Republic of the Congo and none of which require any reference to severity [4,25,26]. A scoping review of the two countries' preparedness instruments gives that account quantitative shape: Uganda's national instruments addressed 47 of 52 components against the Democratic Republic of the Congo's 38, mean Joint External Evaluation indicator scores were 2.75 and 1.63 respectively, and Uganda's outbreak ended with 20 confirmed cases while the Democratic Republic of the Congo had reported 7,890 by 23 September; its authors nonetheless state that a documentary review cannot establish that these differences caused the different courses of the outbreak [27]. The two accounts are not mutually exclusive, and the data assembled here cannot apportion weight between them. The severity pathway has the advantage of yielding a prediction that can be tested prospectively, which the structural account, for all that its components can be scored, does not.

7. Conclusions

Bundibugyo virus is not a mild pathogen, and the 2026 epidemic should end any residual impression that it is. The reviewed evidence nonetheless supports a narrower and more useful claim: the case fatality ratio observed in the opening weeks of a Bundibugyo virus outbreak has been consistently and predictably lower than the ratio established afterwards, and that early figure, not the later one, is what has informed decisions about escalation. The depression is produced by the same species-restricted confirmation that delays recognition, so severity and detection are not independent variables.

The clinical presentation does not explain the pattern. Haemorrhage was documented at closely similar frequencies in Bundibugyo and Ebola virus series, and both sets of authors characterised their patients' illness as generic. The discriminating factors are genomic divergence of up to 43% from the assay-matched species and the diagnostic consequences that follow from it, demonstrated in 2007 by the need for sequencing to identify the agent and repeated in 2026 by a negative result from an Ebola-virus-focused assay.

The operational consequence is a change in how one number should be read. A fatality ratio reported during the first weeks of a filovirus outbreak is an index of how mature the surveillance is, not of how dangerous the pathogen is, and it should not be permitted to govern escalation. Escalation should instead be triggered by species-inclusive confirmation capacity and by signals that are insensitive to ascertainment, of which unexplained deaths compatible with viral haemorrhagic fever, and deaths among healthcare workers, are the most defensible candidates.

8. Future directions

Three lines of work follow directly. The first is the most tractable and the most overdue. The inclusivity of published orthoebolavirus molecular assays against Bundibugyo virus genomes has never been measured. The materials for such a study are already public: the primer and probe sequences of the assays in published use, and the genomes from the 2007, 2012 and 2026 outbreaks, including the 22

deposited from the current epidemic [3]. Aligning the former against the latter and reporting per-assay exact-match rates, with the position of each mismatch relative to the primer 3′ terminus and the probe centre, would convert the present anecdote into a quantity and would yield a table that reference laboratories could act on. The method is established in other viral systems and requires computation rather than containment-level facilities, which places it within reach of groups that cannot work with the agent itself.

The second is to test the loop prospectively. The prediction is specific and falsifiable: in any filovirus outbreak, the ratio of deaths to confirmed cases should rise monotonically as ascertainment broadens, and the gap between the ratio among suspected and the ratio among confirmed cases should close as the interval from onset to sampling shortens. Both quantities are derivable from routine surveillance data that are already collected, and the 2026 epidemic furnishes at least one dataset in which the relationship has been observed in cross-section [13,16]. The obstacle is access, because line-list data carrying event dates rather than reporting dates are rarely released.

The third is to decide what should replace severity as an escalation trigger, and to evaluate the candidates rather than assert them. Sentinel alerts based on unexplained deaths compatible with viral haemorrhagic fever, with healthcare-worker deaths treated as a high-priority special case, have been proposed on the basis of the 2026 growth dynamics [13], and healthcare workers accounted for 7.2% of confirmed cases in the Democratic Republic of the Congo and 20% in Uganda in that epidemic [4]. Whether such a trigger would have fired earlier in 2007 or 2012 is answerable from the existing case records and has not been attempted.

A fourth issue falls outside the detection window but deserves naming. Every interval of unchecked amplification generates a survivor cohort, and the long-term outcomes of Bundibugyo virus survivors rest on two small studies: a retrospective cohort from the 2007–08 outbreak and a cross-sectional follow-up of 40 survivors sixteen years later [28,29]. An epidemic of the size reached in 2026 will produce survivors by the thousand, and the evidence base for their care consists of those two studies. Some of the required capacity was improvised during the 2026 response, when Uganda and the Democratic Republic of the Congo set up a joint incident management team and joint mobile treatment centres at Aru and Kasenyi, supported by mobile laboratories able to confirm cases close to affected communities [30]. The difficulty throughout is that the interval these proposals seek to compress is measured in days, while the capacity they require, decentralised and species-inclusive confirmation, takes years to build and has been recommended, without being funded to completion, since the species was first described [1,5,9].

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