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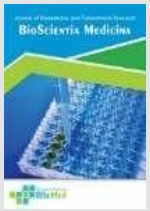

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Fatal Feline Rabies: A Case Report of Encephalitis Following a Cat Scratch in an Endemic Indonesian Region

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A B S T R A C T

Background: Rabies, a viral zoonosis caused by Lyssavirus, remains a significant public health threat, particularly in Asia and Africa, with an almost invariably fatal outcome once clinical symptoms manifest. While dogs are the primary vector, transmission via cats, especially through scratches, is an under-recognized risk in endemic areas like Indonesia. Delayed post-exposure prophylaxis (PEP) and gaps in surveillance contribute to ongoing fatalities. **Case presentation:** We report the case of a 45-year-old Indonesian male from a rural, rabies-endemic area in Jembrana Regency, Bali, who developed fatal encephalitis. Approximately one month prior to symptom onset, he sustained a superficial scratch on his right hand from a free-roaming domestic cat. He did not seek immediate medical attention or PEP. His illness commenced with prodromal symptoms of fever and headache, rapidly progressing to agitation, dysphagia, severe hydrophobia, and aerophobia. Neurological examination revealed fluctuating consciousness, hyperactive reflexes, and marked autonomic dysfunction. Despite intensive supportive care in an isolation unit, his condition deteriorated, leading to death two days after admission. A clinical diagnosis of rabies encephalitis was made based on the characteristic symptoms, a clear history of exposure to a potential vector, and the epidemiological context. **Conclusion:** This case underscores the critical importance of recognizing cats as significant vectors for rabies transmission, even via non-bite exposures like scratches, particularly in endemic settings. It highlights the urgent need for increased public awareness regarding prompt wound management and PEP for all potential rabies exposures, including those from felines. Furthermore, comprehensive rabies control strategies must incorporate feline vaccination and improved surveillance to effectively mitigate this fatal disease in regions like Indonesia.

1. Introduction

Rabies is an acute progressive viral encephalomyelitis caused by neurotropic RNA viruses of the genus *Lyssavirus*, family *Rhabdoviridae*. It is one of the oldest recognized infectious diseases, and despite being 100% vaccine-preventable, it continues to impose a considerable public health burden globally, particularly in resource-limited countries across Asia and Africa, which account for over 99% of all human rabies deaths. The World Health Organization (WHO) estimates that rabies causes approximately 59,000 human deaths annually, though this figure is widely considered an underestimation due to challenges in surveillance and reporting systems, especially in remote and rural areas where the disease is most prevalent. Once clinical symptoms become apparent, rabies is almost invariably fatal, underscoring the critical importance of preventive measures, primarily post-exposure prophylaxis (PEP) and animal vaccination.[1,2]

The primary mode of rabies virus transmission to humans is through the saliva of infected mammals, typically via bites. Scratches contaminated with saliva or direct contact of infectious material (saliva, neural

tissue) with mucous membranes or broken skin are also recognized, albeit less common, routes of transmission. Globally, dogs are the principal reservoir and vector for human rabies, responsible for up to 99% of all human cases. Consequently, rabies control efforts have predominantly focused on canine vaccination and population management. However, other domestic and wild mammals can also transmit rabies to humans. Among domestic animals, cats are increasingly recognized as important secondary vectors of rabies, particularly in regions where canine rabies is endemic and feline vaccination coverage is low. Cats, as natural predators, may contract rabies through encounters with rabid wildlife (bats, foxes) or other infected domestic animals, including dogs. Once infected, cats can transmit the virus to humans through bites or scratches, the latter being a significant mode of exposure that is often underestimated by the public and healthcare professionals.[3,4]

Indonesia, an archipelagic nation in Southeast Asia, remains a country with a high incidence of rabies. Data from the Indonesian Ministry of Health indicated 182,775 cases of Rabies-Transmitting Animal Bites (GHPR) in 2023, with a concerning upward trend in recent years. Provinces such as Bali, East Nusa Tenggara, and North Sumatra consistently report high GHPR figures. In the first quarter of 2025 alone (January to March 7th), 13,453 GHPR cases and 25 rabies-related deaths were recorded nationally. The island of Bali, in particular, has faced significant challenges with rabies since its re-emergence in 2008, with ongoing transmission despite control efforts. These efforts have included mass dog vaccination campaigns, the establishment of Rabies Centers, and community engagement initiatives. However, delays in seeking PEP, incomplete PEP regimens, limited availability of rabies immunoglobulin (RIG) in some areas, and insufficient surveillance contribute to the persistence of human rabies fatalities.[5,6]

While dog-mediated rabies is the primary focus in Indonesia, the role of cats in the local epidemiology of rabies is less well-defined but of growing concern. Free-roaming and unvaccinated domestic cat populations are common in many Indonesian communities, including rural areas of Bali. These animals often have close contact with humans, increasing the potential for transmission events. Cat scratches, often perceived as trivial injuries, may not prompt individuals to seek timely medical attention or PEP, thereby increasing the risk of developing clinical rabies. The clinical presentation of rabies in humans typically involves a variable incubation period (usually 1-3 months, but ranging from weeks to years) followed by a prodromal phase with non-specific symptoms such as fever, malaise, headache, and paraesthesia or pain at the exposure site. This progresses to an acute neurological phase, which can manifest as either furious rabies (characterized by hyperactivity, agitation, hydrophobia, aerophobia, and fluctuating consciousness) or paralytic rabies (characterized by ascending paralysis and less prominent autonomic signs). Both forms ultimately lead to coma and death, usually within 7-10 days of symptom onset, due to respiratory failure or other complications.[7,8]

The novelty of this case report lies in its detailed presentation of a fatal human rabies case unequivocally linked to a cat scratch in a well-documented rabies-endemic region of Indonesia, highlighting a specific mode of transmission that may be underappreciated in public health messaging and clinical practice. While feline rabies transmission is known, detailed case reports from specific endemic localities like Jembrana, Bali, focusing on scratch-only exposure and the subsequent clinical course, are crucial for reinforcing awareness and guiding local prevention strategies.[9,10] This study aims to describe the clinical progression, diagnostic challenges based on clinical presentation in the absence of immediate laboratory confirmation, and management of a fatal case of rabies encephalitis following a cat scratch in Jembrana, Bali. Furthermore, it seeks to emphasize the critical need for heightened public and healthcare provider awareness regarding the risk of rabies transmission from cats, the importance of appropriate PEP for all feline-inflicted injuries in endemic areas,

and the necessity of including cats in comprehensive rabies control and elimination programs. By presenting this case, we intend to contribute to the body of knowledge on feline rabies in Southeast Asia and advocate for a more inclusive One Health approach to rabies control that adequately addresses all relevant domestic animal vectors.

2. Case Presentation

A 45-year-old Balinese male, a farmer by occupation from a rural village in Jembrana Regency, Bali, was brought to the Emergency Department (ED) of Negara General Hospital due to acute agitation and inability to swallow, which had manifested hours prior. The patient's history revealed that approximately one month before this acute presentation, he had sustained scratches on his right hand from a free-roaming domestic cat. He had considered the injury minor, performing only a cursory wash with water, and crucially, did not seek any medical attention or post-exposure prophylaxis (PEP). The cat was reportedly unvaccinated and had not been seen for several weeks prior to the patient's admission. The prodromal phase of his illness began two days before admission, characterized by chest discomfort radiating to the neck, fever, and headache. He had no significant past medical illnesses or relevant family history. His village was known to have cases of rabies in dogs, indicating an endemic area (Table 1).

Upon physical examination in the ED (Day 1), the patient was highly agitated, with fluctuating consciousness (Glasgow Coma Scale E4V4M5, fluctuating). Vital signs showed hypertension (BP 160/90 mmHg), a normal pulse rate (88/min), normal respiratory rate (20/min), a slight fever (axillary temperature 37.5°C), and normal oxygen saturation (SpO_2 98% on room air). Distinctive neurological signs included pronounced photophobia, hypersalivation, and severe dysphagia. The hallmark signs of furious rabies were strikingly evident: extreme hydrophobia, where attempts to swallow or even the sight/sound of water triggered intense pharyngeal spasms and panic, and aerophobia, with excessive reactions to air currents. Deep tendon reflexes were symmetrically hyperactive, and fine tremors were noted in his digits. Healed superficial scars, consistent with the reported cat scratch, were visible on the dorsum of his right hand. Other systemic examinations, including cardiovascular, respiratory, and abdominal, were largely unremarkable. Initial laboratory investigations (Day 1) revealed leukocytosis (WBC 13.8 × 10^3/µL) and hyperglycemia (random blood glucose 284 mg/dL). Renal function tests showed elevated BUN (57 mg/dL) and slightly elevated serum creatinine (1.3 mg/dL), while serum electrolytes were within normal limits. A clinical diagnosis of rabies encephalitis (furious form) was made based on these comprehensive findings.

The patient was immediately admitted to a quiet isolation room to minimize external stimuli (Table 2). Management was primarily supportive. This included intravenous hydration with 0.9% NaCl, IV Paracetamol for fever, IV Ondansetron as an antiemetic, IV Ranitidine for gastric protection, IV Dexamethasone empirically for potential cerebral inflammation, and IV Ceftriaxone for prevention of secondary infections. His hypertension was managed with oral Candesartan, and his hyperglycemia was addressed with Insulin Glargine and Insulin Lispro following an internal medicine consultation. Severe agitation necessitated mechanical restraints and pharmacological sedation with IV Diazepam, initially as a bolus and then as a continuous infusion, titrated to effect.

Despite these interventions, the patient's clinical condition progressively worsened. Throughout the evening of Day 1 and into Day 2, he experienced persistent episodes of agitation, and the hydrophobia and aerophobia remained profound. His level of consciousness steadily declined. On Day 2, he developed increasing autonomic dysfunction, with more pronounced temperature instability, tachycardia, and labile blood pressure. His respiratory efforts became increasingly labored. The focus of care remained palliative, aiming to ensure comfort and dignity. After approximately 53 hours from the onset

of specific neurological symptoms, and on the second day of hospitalization, the patient suffered a cardiorespiratory arrest and was pronounced dead. A post-mortem brain biopsy for definitive laboratory confirmation of rabies was declined by the family.

Table 1. Summary of patient's clinical findings.

Category	Details
Demographics	45-year-old male, Balinese ethnicity, farmer, resident of rural Jembrana Regency, Bali.
Anamnesis	
Chief complaints	Acute agitation, aggressive behavior, inability to swallow water (dysphagia), choking sensation. Onset ~4 hours prior to admission.
History of exposure	Scratches on the dorsum of the right hand from a free-roaming domestic cat ~1 month prior. Cat unowned, unvaccinated, status unknown (disappeared). No PEP sought.
Prodromal symptoms	~2 days prior to admission: chest discomfort radiating to the neck, fever, headache, increasing anxiety, and restlessness.
Past medical history	No history of hypertension, diabetes mellitus, stroke, or heart disease. No regular medications.
Family history	No similar history in the family.
Social history & epidemiological context	Routinely cared for cattle, frequent interaction with dogs and cats. Resided in a known rabies-endemic area (reported cases in dogs).
Physical examination (Day 1)	
General appearance & mental status	Visibly distressed, highly agitated, uncooperative. Fluctuating consciousness (GCS E4V4M5, intermittent disorientation). Anxious, fearful facial expression.
Vital Signs	BP: 160/90 mmHg; Pulse: 88/min; RR: 20/min; Temp (axillary): 37.5°C; SpO_2: 98% (room air).
Head & neck	Pupils ERL (3mm), photophobia. No nuchal rigidity. Hypersalivation. Oral cavity difficult to inspect.
Cardiovascular	S1S2 normal, regular rhythm, no murmurs.
Respiratory	Symmetrical expansion, resonant percussion, clear vesicular breath sounds, no added sounds.
Abdominal	Soft, non-tender, not distended, normal bowel sounds, no hepatosplenomegaly.
Integumentary	Healed, superficial linear scars on dorsum of right hand. Warm peripheries.
Neurological examination	
Cranial nerves	Photophobia. Prominent dysphagia.
Motor system	Generally increased tone, hyperactive spontaneous movements. Strength 4-5/5 where assessable. Fine tremors in digits.
Reflexes	Deep tendon reflexes symmetrically hyperactive (3+ to 4+). Plantar responses flexor. No pathological reflexes.
Sensory system	Reactive to painful stimuli; detailed exam limited.
Specific signs	Pronounced hydrophobia (panic, pharyngeal spasms with water). Marked aerophobia (excessive reaction to air movement).
Autonomic signs	Labile BP (initially high), profuse sweating (intermittent), hypersalivation.
Laboratory findings (Day 1)	
Complete blood count	WBC: $13.8\times10^3/\mu L$ (Leukocytosis); Hb: 12.9 g/dL; Hct: 39.9%; Platelets: $263\times10^3/\mu L$.
Blood chemistry	Random Blood Glucose: 284 mg/dL (Hyperglycemia); BUN: 57 mg/dL (Elevated); Serum Creatinine: 1.3 mg/dL (Slightly elevated).
Serum electrolytes	Sodium: 136 mEq/L; Potassium: 4.4 mEq/L; Chloride: 98.5 mEq/L (Normal).
Clinical diagnosis	Rabies Encephalitis (Furious Form).

Table 2. Procedure of treatment and follow-up.

Aspect	Details
Initial setting	Admitted to a quiet, darkened isolation room in Negara General Hospital to minimize external stimuli.
Pharmacological & supportive therapy (Day 1 onwards)	
IV fluids	0.9% Sodium Chloride at 20 drops per minute.
Antipyretic	IV Paracetamol 1 g three times daily.
Antiemetic	IV Ondansetron 4 mg three times daily.
Gastric protection	IV Ranitidine 50 mg twice daily.
Anti-inflammatory	IV Dexamethasone 5 mg four times daily (empirical).
Antibiotics	IV Ceftriaxone 2 g twice daily (prophylactic).
Antihypertensive	Oral Candesartan 8 mg once daily.
Hyperglycemia management	Internal Medicine consultation. Insulin Glargine 10 units once daily, Insulin Lispro 6 units three times daily. Regular blood glucose monitoring.
Management of agitation	Mechanical restraints (soft limb restraints). Pharmacological: IV Diazepam 10 mg bolus, followed by continuous infusion of 20 mg Diazepam in 500 mL 0.9% NaCl over 8 hours, titrated to effect.
Consultations	Internal Medicine (for hyperglycemia). Local public health authorities notified.
Follow-up & outcome	
Hospital course	Despite sedation, persistent agitation, hydrophobia, and aerophobia. Progressive decline in consciousness (Day 1 evening - Day 2). Developed increasing autonomic dysfunction (Day 2). Palliative care focus.
Final outcome	Cardiorespiratory arrest on Day 2 of hospitalization (~53 hours after onset of specific neurological symptoms). Pronounced dead after brief, futile resuscitation efforts.
Post-mortem confirmation	Declined by family.

3. Discussion

This report details a tragic case of human rabies encephalitis in a 45-year-old male from Jembrana, Bali, an area known for its rabies endemicity. The diagnosis was established based on a classic clinical presentation of furious rabies, including the pathognomonic signs of hydrophobia and aerophobia (Table 1), coupled with a history of exposure—a scratch from a free-roaming domestic cat approximately one month prior to symptom onset—and the critical absence of post-exposure prophylaxis (PEP) (Table 1). The patient's rapid neurological decline and subsequent death within two days of hospitalization, despite supportive management (Table 2), are characteristic of this almost invariably fatal disease. This case provides a crucial opportunity to delve into the intricate interplay between the clinical findings and the underlying pathophysiology of rabies, the specific risks associated with feline transmission, diagnostic complexities, management limitations, and the broader public health imperative for enhanced control strategies in endemic regions like Indonesia.

The clinical manifestations observed in this patient (Table 1) are direct consequences of the rabies virus's profound neurotropism and its devastating impact on the central nervous system (CNS). Following the cat scratch on the patient's hand, the rabies virus, presumably present in the cat's saliva, contaminating its claws, gained entry into the patient's peripheral nerves. The virus binds to acetylcholine receptors (like nAChR) or other neuronal receptors (NCAM, p75NTR) at the neuromuscular junction or nerve endings within the dermis and muscle. Once internalized, the virus embarks on a relentless journey via rapid retrograde axonal transport along peripheral nerves towards the spinal cord and brain. The incubation period, approximately one month in this case, is variable and depends on several factors: the site of

inoculation (richly innervated areas like hands and face are associated with shorter incubation), the severity of the wound (deeper wounds with higher viral load shorten incubation), the distance from the CNS, the variant of the rabies virus, and the host's age and immune status. During this period, the virus replicates within dorsal root ganglia before extensively invading the CNS. The initial prodromal symptoms of fever, headache, and localized discomfort radiating from the chest to the neck likely coincided with early CNS involvement and inflammatory responses, or perhaps aberrant sensations related to viral activity in spinal ganglia. Paresthesia or pain at the site of the bite/scratch is a common early symptom, reflecting local nerve involvement, though not explicitly detailed as the primary prodromal complaint in this instance. The development of furious rabies, as seen in this patient with extreme agitation, hydrophobia, and aerophobia (Table 1), reflects widespread viral infection and dysfunction in specific brain regions. These are perhaps the most agonizing and characteristic symptoms. They result from severe, involuntary, and painful spasms of the pharyngeal, laryngeal, and diaphragmatic muscles upon attempts to swallow liquids or exposure to air currents. This is thought to be due to viral-induced dysfunction of brainstem nuclei controlling swallowing and respiration, including the nucleus ambiguus and surrounding pontine and medullary circuits. The virus disrupts inhibitory neurotransmission (GABAergic pathways) and sensitizes reflex arcs, leading to these exaggerated responses. The terror associated with these spasms reinforces avoidance of water and air. The patient's panic and struggling when offered water, and jumping at the sound of running water, vividly illustrate this profound neurological derangement.

These symptoms are indicative of diffuse encephalitis affecting higher cortical centers, the limbic system (involved in emotion and behavior), and neurotransmitter systems (dopamine, serotonin, acetylcholine). The rabies virus does not typically cause widespread neuronal destruction in the same way as some other encephalitides (Herpes Simplex Encephalitis). Instead, it primarily causes profound neuronal dysfunction. This "non-cytolytic" nature of infection, where neurons remain structurally relatively intact but functionally impaired, contributes to the unique clinical picture. The virus can alter ion channel function, neurotransmitter release, and receptor activity, leading to neuronal hyperexcitability or inhibition in different brain regions. The fluctuating consciousness, with periods of lucidity interspersed with confusion and aggression, is a hallmark of this widespread but unevenly distributed neuronal dysfunction. Rabies virus infection frequently involves autonomic nervous system pathways, leading to signs such as hypersalivation, pupillary changes (photophobia was noted), cardiac arrhythmias, labile blood pressure (hypertension was noted), and abnormal sweating. These features arise from viral involvement of brainstem autonomic centers, hypothalamic nuclei, and peripheral autonomic ganglia. The initial hypertension in this patient could reflect sympathetic overactivity. As the disease progresses, severe autonomic instability often contributes to cardiovascular collapse. A peculiar aspect of rabies is the virus's ability to evade early and robust immune detection within the CNS. Several viral proteins, particularly the phosphoprotein (P protein), interfere with host innate immune responses, including interferon pathways. This "stealth" mechanism allows the virus to replicate and spread widely before a significant adaptive immune response is mounted within the CNS. By the time antibodies are detectable in the CSF (if at all), the infection is usually too advanced. Histopathologically, rabies encephalitis is characterized by relatively mild inflammation (perivascular cuffing, microglial activation, and variable neuronal degeneration) despite profound clinical deficits. The presence of Negri bodies (intracytoplasmic eosinophilic inclusions composed of viral nucleocapsids) in neurons is pathognomonic, but they are not found in all cases and require specific staining and expertise for identification, usually post-mortem. The primary cause of death is usually respiratory failure due to brainstem dysfunction or

cardiovascular collapse from autonomic instability.[11,12]

The transmission of rabies via a cat scratch, as occurred in this case, warrants detailed consideration. While dogs are the global mainstay of rabies transmission to humans, cats are important secondary vectors, especially in rabies-endemic areas with significant free-roaming cat populations and insufficient canine rabies control. Cats frequently lick their paws and claws as part of grooming. If a cat is rabid, its saliva contains the virus. A scratch that breaks the skin can then become contaminated with this infected saliva, inoculating the virus into the victim. The depth of the scratch and the amount of viral inoculum are critical factors. Even superficial scratches that cause minor abrasions can be sufficient for viral entry if saliva is present. The patient's healed scars on the dorsum of the hand indicate a breach of the skin barrier. Cats often contract rabies from rabid dogs or wildlife (foxes, raccoons, skunks, bats, depending on the region). Unvaccinated, free-roaming cats are at the highest risk. In areas like Bali, where canine rabies is endemic, the spillover to the feline population is a recognized concern. The implicated cat in this case was described as a "free-roaming pet cat" , suggesting it was not strictly confined and likely unvaccinated, fitting the high-risk profile. The cat's disappearance is also consistent with the behavior of rabid animals, which may wander off, become reclusive, or die. Rabies in cats can manifest in furious or paralytic ("dumb") forms, similar to dogs. Furious rabies in cats may involve unprovoked aggression, irritability, attacking objects or other animals/humans, restlessness, and vocal changes. Paralytic rabies often presents with lethargy, ataxia, and progressive paralysis. Hypersalivation can occur in both forms. Cat scratches are often perceived as less dangerous than dog bites. This perception can lead to delays or failures in seeking PEP, as was tragically the case here. Public health campaigns must explicitly address the risk of rabies from cat exposures (both bites and scratches) and emphasize that any break in the skin caused by a potentially rabid animal warrants medical evaluation for PEP.[13,14]

The diagnosis of human rabies in this case was made on strong clinical grounds (Table 1), a common scenario in many regions where advanced laboratory confirmation is not readily available or results are delayed. The combination of a compatible exposure history (cat scratch in an endemic area, no PEP) followed by a characteristic prodrome and the development of pathognomonic neurological signs (hydrophobia, aerophobia, agitation) is highly indicative of rabies. In such clear-cut presentations, clinical diagnosis can have a high positive predictive value in endemic settings. While hydrophobia is nearly pathognomonic for rabies, other conditions must be considered in cases of acute encephalitis or rapidly progressive neurological disease. These include: Herpes Simplex Encephalitis (HSE): Can cause fever, altered consciousness, seizures, and focal neurological signs. However, prominent hydrophobia and aerophobia are not typical features of HSE. Brain imaging (MRI) and CSF PCR for HSV are key for diagnosis. Other Viral Encephalitides: Arboviruses (Japanese Encephalitis in parts of Asia) can cause severe encephalitis. Clinical features and epidemiological context (season, mosquito exposure) are important. Tetanus: If the wound was contaminated, tetanus could be a remote consideration, causing muscle spasms. However, the dysphagia in tetanus (trismus, risus sardonicus, opisthotonus) differs from rabies-induced hydrophobia, and the sensorium is usually clear until late stages. The cat scratch here was superficial. Acute Psychiatric Conditions/Delirium: Agitation and altered behavior can mimic psychiatric illness. However, the specific neurological signs like hydrophobia, aerophobia, and progressive focal deficits point away from a primary psychiatric disorder. Metabolic Encephalopathies or Toxin Exposure*:* These can cause altered mental status but usually lack the specific brainstem signs of rabies. The patient's hyperglycemia and elevated BUN/creatinine were noted but were more likely consequences of the acute illness/stress or dehydration rather than

primary causes of the encephalitic picture.[15,16]

Definitive diagnosis relies on detecting rabies virus, viral antigens, or nucleic acids in clinical specimens (ante-mortem: saliva, CSF, nuchal skin biopsy for RT-PCR or virus isolation; post-mortem: brain tissue for direct fluorescent antibody (DFA) test, RT-PCR, or virus isolation) or detecting specific rabies virus-neutralizing antibodies in the CSF of an unvaccinated individual. RT-PCR on saliva samples can be positive from early in the clinical course. Nuchal skin biopsy (containing nerve endings at hair follicles) for RT-PCR is also a sensitive ante-mortem test. CSF analysis in rabies may show mild pleocytosis and protein elevation, but these are non-specific. Detection of rabies virus RNA by RT-PCR or rabies virus-neutralizing antibodies in CSF (in an unvaccinated person) is diagnostic. Serum antibodies typically appear later in the illness (after the first week), and their presence in an unvaccinated individual is diagnostic. However, their appearance often coincides with irreversible neurological damage. DFA test on brain tissue is the gold standard for post-mortem diagnosis. In this case, the extreme agitation and rapid deterioration of the patient made ante-mortem sample collection (especially LP or skin biopsy) highly challenging (Table 1: Imaging; implicitly, why CSF was not obtained). The lack of immediate availability of specialized rabies diagnostic tests at the local hospital is a common issue in many endemic areas. Family refusal for post-mortem examination further precluded definitive laboratory confirmation, a frequent cultural and emotional barrier.[17,18]

The interventions provided to this patient—IV fluids, antipyretics, sedation, empirical antibiotics/dexamethasone, and management of comorbidities like hyperglycemia and hypertension — were aimed at maintaining physiological stability and providing comfort. Isolation in a quiet, dark room is crucial to minimize stimuli that can trigger spasms and agitation. Sedation with benzodiazepines (diazepam, lorazepam) and/or antipsychotics (haloperidol, chlorpromazine) is a cornerstone of symptomatic relief. The goal is to reduce agitation, anxiety, and the intensity of spasms, thereby alleviating suffering. Continuous infusions may be necessary. The Milwaukee Protocol, an aggressive experimental treatment involving therapeutic coma, antiviral agents, and intensive neurocritical care, was first reported in 2004 with one survivor. However, subsequent attempts to replicate this success have been largely disappointing, with very few survivors, often with severe neurological sequelae. The protocol is highly resource-intensive, and its efficacy remains unproven and controversial. Most rabies experts do not recommend it outside of highly specialized research settings with full informed consent, and many consider it to be ethically problematic given its low success rate, high cost, and potential to prolong suffering. Other antiviral drugs and immunomodulatory therapies have also been tried without consistent success. The fundamental challenge is that by the time clinical symptoms appear, the virus has already caused extensive neuronal dysfunction, which may be irreversible. Given the near-100% fatality, a palliative care approach is often the most humane and appropriate strategy once rabies is clinically established. This involves meticulous attention to symptom control (pain, dyspnea, agitation, seizures, hypersalivation), psychological and spiritual support for the patient (if conscious) and family, and clear communication about the prognosis. Withholding or withdrawing futile life-sustaining interventions, in accordance with patient/family wishes and ethical guidelines, is an important aspect of palliative care in rabies. The decision to provide comfort-focused care for this patient was appropriate given the diagnosis and grim prognosis.[19,20]

This case underscores the ongoing public health challenge posed by rabies in Indonesia, particularly in endemic areas like Bali, and the critical need for robust, multi-pronged control strategies. Post-Exposure Prophylaxis (PEP), Timely and appropriate PEP is the most effective way to prevent rabies in humans after exposure. Immediate and thorough washing of the bite/scratch wound with soap and

water for at least 15 minutes is crucial to remove and inactivate virus particles. Application of a virucidal antiseptic (povidone-iodine, alcohol) is also recommended. The patient's cursory wound washing without soap was inadequate. A course of modern, potent cell-culture rabies vaccine should be administered according to WHO-recommended schedules (intramuscular Essen regimen of 4 or 5 doses, or intradermal regimens which are dose-sparing). Rabies Immunoglobulin (RIG), For Category III exposures (transdermal bites/scratches, contamination of mucous membranes, licks on broken skin, or any exposure to bats) and for Category II exposures in immunosuppressed individuals, RIG (human or equine) should be infiltrated into and around the wound if feasible, with any remainder given intramuscularly at a distant site. Availability and affordability of RIG can be major challenges in resource-limited settings. The failure of this patient to seek PEP highlights critical gaps in public awareness about the seriousness of animal scratches and the importance of PEP. The cornerstone of rabies prevention and elimination at the source is sustained mass vaccination of reservoir animal populations, primarily dogs, aiming for at least 70% coverage. This case strongly advocates for the inclusion of domestic cats in these mass vaccination campaigns, especially in areas with significant free-roaming cat populations interacting with dogs and humans. Indonesian rabies control programs, including those in Bali, have focused on dog vaccination, but specific strategies for feline rabies control may need strengthening. Effective surveillance in both human and animal populations is essential to monitor trends, identify high-risk areas, detect outbreaks, and evaluate the impact of control interventions. This includes laboratory capacity for rabies diagnosis. Underreporting of animal bites/scratches and suspected rabies cases is a common problem.

Continuous public education campaigns are vital to inform communities about rabies risks, responsible pet ownership (including vaccination and confinement of animals), how to avoid animal bites/scratches, appropriate first aid for wounds, and the critical need to seek prompt medical attention for PEP. These messages must be culturally appropriate and reach remote and vulnerable populations. The role of community health workers and local leaders is crucial. Rabies is a classic zoonotic disease that demands a One Health approach, integrating efforts across human health, animal health, and environmental sectors. This involves collaboration in surveillance, outbreak response, vaccination campaigns, public education, and policy development. Bali has made significant efforts in rabies control since the 2008 outbreak, yet challenges persist. These may include maintaining high dog vaccination coverage across all areas, managing large populations of free-roaming dogs and cats, logistical difficulties in reaching remote villages, ensuring consistent PEP availability, and addressing socio-cultural factors that influence human behavior towards animals and health-seeking practices. Specific data on feline rabies prevalence and cat vaccination coverage in Jembrana would be valuable for targeted interventions.

This case report, by detailing a fatal instance of rabies encephalitis transmitted via a cat scratch in a specific, rabies-endemic rural Indonesian setting (Jembrana, Bali), serves to: Reinforce to clinicians, particularly in endemic areas, the importance of maintaining a high index of suspicion for rabies in patients presenting with acute neurological syndromes, even if the exposure was a seemingly minor cat scratch. Highlight for public health authorities the ongoing risk posed by feline rabies and the need to include cats explicitly in rabies awareness campaigns and control strategies (vaccination drives). Emphasize the absolute criticality of prompt wound care and PEP for all potential rabies exposures, countering any public complacency regarding non-bite exposures from animals other than dogs. Contribute to the local epidemiological understanding of rabies transmission modes in Bali, providing a case-based example that can inform local educational materials and training for healthcare providers. Advocate for continued investment and strengthening

of the One Health approach to rabies control in Indonesia, addressing both canine and feline reservoirs, to progress towards the goal of zero human deaths from dog-mediated (and other domestic animal-mediated) rabies. The tragic outcome for this patient underscores the devastating potential of rabies when preventive measures are not taken. Each human rabies death is a preventable tragedy and a call to intensify efforts towards global rabies elimination.

4. Conclusion

This case of fatal human rabies encephalitis following a cat scratch in an endemic region of Bali, Indonesia, serves as a stark reminder of the persistent threat posed by this ancient disease and highlights the often-underestimated role of felines in its transmission dynamics. The classic presentation of furious rabies, characterized by hydrophobia and rapid neurological deterioration, tragically led to the patient's demise despite supportive hospital care. The key learning points from this case are multifaceted. Firstly, it underscores the critical importance for both the public and healthcare professionals to recognize that cats, not solely dogs, can be significant vectors of rabies, and that non-bite exposures such as scratches can effectively transmit the virus. Secondly, it emphasizes the life-saving importance of immediate and thorough wound care followed by prompt administration of post-exposure prophylaxis (PEP) for any potential rabies exposure, including all bites and scratches from potentially rabid animals, irrespective of the perceived severity of the wound. The failure to seek PEP in this instance was a fatal oversight. Thirdly, this case highlights the ongoing challenges in rabies control in endemic areas, including the need for enhanced surveillance, improved public awareness campaigns that specifically address feline rabies and non-bite exposures, and ensuring the accessibility and affordability of PEP, including rabies immunoglobulin. Finally, for effective rabies elimination, a comprehensive One Health approach is paramount. This necessitates robust, coordinated strategies that include sustained mass vaccination of primary (dogs) and significant secondary (cats) domestic animal reservoirs, responsible pet ownership, effective management of stray animal populations, and active community engagement. Continued vigilance, education, and integrated control efforts are essential to prevent such tragic and entirely avoidable deaths from rabies in Indonesia and other endemic regions worldwide. This case report contributes to the growing body of evidence calling for more inclusive rabies control programs that adequately address the risks posed by all relevant domestic animal species.

5. References

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