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Case Report: Re-Emergence of Rabies Cases in Denpasar

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ABSTRACT

Rabies is a fatal zoonotic disease caused by *Lyssavirus* from the *Rhabdoviridae* family, which primarily affects the central nervous system. Once symptoms manifest, the case fatality rate exceeds 99%, making it one of the deadliest viral infections. The high mortality rate emphasizes the urgency of preventive measures. This case report aims to highlight the clinical features, diagnostic challenges, and management of symptomatic rabies in humans and reinforce the importance of post-exposure prophylaxis (PEP). A descriptive clinical case report is presented of a 41-year-old male who developed rabies symptoms three months after a dog bite, having not received PEP. Clinical presentation included hydrophobia, vomiting, restlessness, and paresthesia. The patient was treated symptomatically with intravenous antibiotics, antipyretics, gastric protection, and psychiatric support, alongside monitoring in a special care unit. The results show that despite comprehensive symptomatic management—including ceftriaxone, paracetamol, omeprazole, and haloperidol—rabies remained untreatable at the symptomatic stage. This case underscores the critical window for prevention before the onset of symptoms. Rabies remains incurable once neurological symptoms appear. Thus, in any suspected exposure to potentially rabid animals, prompt and complete post-exposure prophylaxis—comprising wound care, anti-rabies vaccine, and rabies immunoglobulin—is essential. This case reinforces the public health importance of early intervention to prevent fatal outcomes.

Keywords: *Rabies, rabies virus transmission, bite wound management.*

INTRODUCTION

Rabies is an acute zoonotic disease that attacks the central nervous system and can be fatal (Bilal, 2021). Rabies is estimated to cause 59,000 deaths annually in 150 countries, with 95% of cases occurring in Africa and Asia. 99% of rabies cases are caused by dog bites, and occur more in lower economic class communities. It is estimated that half of the total cases occur in children under the age of 15 (Organization, 2012). Keep in mind that rabies is a neglected tropical disease that is widespread around the world, and there are still many cases of rabies that go unreported or underreported, so the extent of the spread of the disease remains unclear. Based on data from the Ministry of Health in 2011 – 2018, in Indonesia the average number of rabies-infected animal bite cases per year is 78,413 cases and an average of 63,534 cases receive anti-rabies vaccine (VAR) (Kemenkes, 2019).

The rabies virus has a wide variety of abilities to invade humans. This virus can survive on heating for a long time (Rather & Vasavi, 2024; Tarantola, 2017). However, the rabies virus is easily killed by sunlight and ultraviolet rays, the influence of acidic and alkaline states, fat solvents such as ether and chloroform, Na deoxychlorate, and soapy water (Akoso, 2007). The virus infects humans through bite and non-bite by infected animals. The incubation period of this virus also varies depending on the type/strain of the rabies virus, the number of viruses, the depth of the bite wound, the location of the bite wound, the amount of neuration in the wound area, and the patient's immunity.1 After infection, the patient will go through several stages of clinical symptoms consisting of the prodromal stage, sensory stage, excitation stage, and paralysis stage with their respective manifestations (Bilal, 2021; Kemenkes, 2019).

The enforcement of rabies cases is not much different from the enforcement of other diseases that require anamnesis, physical examinations, and support. Despite having clear guidelines, rabies bite

cases are a widespread case in Bali and require strict clinical attention because they can be fatal. This case report will discuss a 41-year-old male patient infected with rabies from diagnosis enforcement to clinical management.

RESEARCH METHOD

This study adopts a case report design to examine the clinical management of a 41-year-old male patient with suspected symptomatic rabies following a dog bite. The research aims to describe the patient's clinical presentation, diagnostic process, and treatment interventions to highlight the consequences of delayed or absent post-exposure prophylaxis (PEP). The population in this study includes individuals exposed to rabies-infected animals, with the subject selected through purposive sampling based on relevant clinical criteria. Data were collected through direct observation, detailed anamnesis, physical and neurological examinations, and laboratory investigations, supplemented by a review of medical records and treatment documentation. The data were analyzed using narrative descriptive methods to reconstruct the clinical progression from initial symptoms to final treatment outcomes. This approach allows the study to contextualize clinical decision-making in rabies management and emphasize the urgent need for timely PEP and improved awareness in endemic regions.

Case Report

A man with the initials ME, 41 years old, came with complaints of vomiting since 2 days before entering the hospital. Vomiting more than 7 times, the complaints are getting more and more severe. In addition, patients were seen forcing themselves while drinking water, accompanied by choking and coughing while drinking water. The patient also complained of tingling and a feeling of thickness in both legs since a few days ago. Patients complain that sometimes there is a shortness of a missing nature. The patient feels even tighter when exposed to air (fanned). There is no photophobia. 3 months ago there was a history of a stray dog bite on the index finger of the left hand which caused an open wound. When after the incident, the patient did not wash the wound, only cleaned the wound with povidone iodine. There is no history of getting the anti-rabies vaccine.

From the physical examination carried out on April 3, 2025, the awareness of the Glasgow Coma Scale (GCS E4 V5 M6) with a temperature of 36.3 degrees Celsius, respiration 20 x/min, pulse 123 x/min, blood pressure 128/88 mmHg, and 95% saturation in room water was obtained. It was found that the eye conjunctiva was not anemic and the sclera were not icteric, plaque and redness of the pharynx were absent, the heart of S1 S2 was single regular and murmur was absent. Lung examination found vesicular sounds, no rhonki and no wheezing. Abdominal examination, normal bowel noise is heard, there is no distension. Examination of the acral extremities was warm and no edema was found. Neurological clinical examination, no meningeal signs, 3mm/3mm isocor spherical pupils, light reflex +/+, corneal reflex +/+, cranial nerve paresis (-), motor strength of the upper and lower extremities normal, sensory within normal limits, negative pathological reflexes, photophobia (-), hydrophobia (+), aerophobia (+).

The laboratory results were obtained on April 2, 2025, leukocytes (WBC) 16.51 × 10^3/µL, neutrophils 14.57 × 10^3/µL, lymphocytes 0.97 × 10^3/µL, hemoglobin (HGB) 16.7 g/dL, hematocrit (HCT) 49.5%, platelets (PLT) 302 × 10^3/µL, mean corpuscular volume (MCV) 81.8 fl, mean corpuscular hemoglobin (MCH) 27.6 pg, urea 27mg/dL, serum creatinine 0.69 mg/dL, Sodium 139mmol/L, Potassium 3.8 mmol/L, blood sugar at 154mg/dL.

The patient was diagnosed with suspected rabies and bacterial encephalitis. The patient was given ceftriaxone 2 grams every 12 hours IV, paracetamol 1 gram every 8 hours IV, omeprazole 40mg every 12 hours IV, psychiatric TS consul due to anxiety and then given haloperidol 1/2 IM ampoule (can be repeated 1 hour post the first injection, maximum 1 ampoule) then do a rest strain, TS anesthesia consul acc special treatment room.

RESULTS AND DISCUSSION

Rabies is an acute zoonotic disease caused by a virus of the genus Lyssavirus, family Rhabdoviridae. This virus is made up of RNA nuclei, then in humans this virus attacks the central nervous system. Rabies is almost always fatal if post-exposure prophylaxis is not given before the onset of severe symptoms. In this case illustration, a 41-year-old male patient came in with complaints of vomiting, forcing himself while drinking water, tingling and a feeling of thickness in both limbs, fear of air, shortness of breath, and restlessness. Accompanied by a history of being bitten by a stray dog 3 months ago on the index finger of the left hand and not getting rabies-infected animal bite prevention procedures which puts the patient at high risk of being infected with rabies. Cases like this are very relevant to be discussed in depth considering the importance of knowing the transmission of rabies virus transmission and the management of rabies-infected animal bite wounds to prevent mortality due to rabies-infected animal bites.

The rabies virus is an RNA virus that comes from the genus lyssa virus, family Rhabdoviridae. The three major subfamilies of rhabdoviridae consist of alpharhabdovirinae, betarhabdovirinae, and gammarhabdovirinae. Cases of rabies infection in humans are often associated with the specific subfamily Alpharhabdovirinae of the genera Lyssavirus (Shepherd et al., 2023). The rabies virus virion is encased by host cells and is shaped like a bullet with a length of 130–250nm by a diameter of 60–100nm. It has 2 functional parts, namely the inner part is made up of nucleocapsids including RNA, and the outer part is made up of 2 lipid layers with prominent parts (like spines) derived from viral glycoproteins. The viral genome is made up of 5 proteins: nucleoprotein (N), phosphoprotein (P), matrix protein (M), glycoprotein (G), and polymerase (L) (Fisher et al., 2018). Viral proteins have a wide range of functions.

The N protein plays a major role in lining the viral genome to protect it from the RNAse activity of the host cell, as well as interacting with the L protein and P protein in the process of transcription and replication. Protein P, which is a polymerase cofactor but is not catalytic, helps to place the L protein to match the N-RNA mold, as well as acting as a chaperone in the synthesis of N proteins. The M protein binds to the nucleocapsids as well as the cytoplasmic domain of the G protein, and functions in facilitating the process of viral release (budding). Protein G itself is a component of the virus located on the surface of the virion. It plays a role in binding to the receptors of the host cell, triggering the process of endocytosis, and aiding in fusion between the virus and the endosome membrane. Because it is the only external component, the G protein also stimulates the formation of viral neutralizing antibodies (VNAs), which then trigger an immune response from the host cell. Meanwhile, the L protein has many domains and plays an important role in the transcription process as well as the replication of the viral genome (Rupprecht et al., 2018).

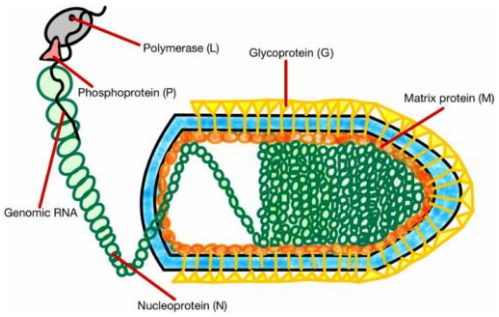

Figure 1. Rabies Virus Structure (Rupprecht et al., 2018)

The main characteristics of the rabies virus are the ability to attack the nervous system (neuroinvasiveness), the tendency to live inside nerve cells (neurotropism), and the ability to cause nerve damage (neurovirulence). The G protein in the rabies virus plays an important role in neuroinvasive processes, including the entry of the virus into cells, its spread, and the synthesis of RNA (Dietzschold et al., 2008). Once a person is bitten, the G protein receptor helps the virus attach to target cells such as muscle cells (myocytes) and local sensory or motor nerves. The virus then replicates inside myocytes and macrophages before spreading to the central nervous system through muscle spindles (sensory) or neuromuscular (motor) connections (Singh et al., 2017). The virus enters the peripheral nerve through the bite wound, then travels to the terminal neuron and spreads retrograd (towards the nerve center) until it reaches the spinal medulla. This deployment is carried out through a mobile transportation mechanism. The virus is carried to the nerve cell body with the help of the dynein motor complex through microtubules. Once it reaches the cell body, the virus is replicated and channeled anterograd (to the periphery) using the kinesin motor complex. From the spinal cord, the virus spreads to various parts of the brain such as the cerebral cortex, cerebellum, diencephalon, midbrain, pons, and medulla oblongata. The virus passes through the dorsalis cornea to the cerebral cortex and conducts synapses in the nucleus of the dorsal column (medulla oblongata) and thalamus (diencephalon). Other pathways are through the spinotachalamic tract (to the cerebral cortex with synapses in the thalamus and cornu dorsalis medulla spinalis), the spinocerebellar tract (to the cerebellum), and the corticospinal tract (to the cerebral cortex with synapses in the ventral corneus medulla spinalis) (Begeman et al., 2018).

The rabies virus in large quantities (high inoculum) can directly infect the motor nerve endings without the need to replicate first in the muscles, unlike previous ones that are transmitted through inoculation directly into the nerves. This explains why the incubation period can be shorter (Hemachudha et al., 2013). The virus spreads from one neuron to another via the p75 neurotrophin receptor (p75NTR). The L protein aids in the transport of viruses by modifying microtubules, while the M protein enhances viral transcription and replication through the process of depolymerization of microtubules. The retrograd spread lasts about 50–100 mm per day. The mechanism of anterograd spread is not yet known, but the virus can spread to the salivary glands through terminal axons. The rabies virus can also spread anterogradously to peripheral tissues, including non-nervous organs (Scott & Nel, 2021). The

rabies virus can survive on heating for a long time. In dry heating reaching a temperature of 100oC can still last for 2-3 minutes. Inside saliva with hot air temperature can last for 24 hours. In a freeze-dried state with storage at 4oC the virus can survive for many years, this is the basis why the anti-rabies vaccine must be stored at a temperature of 4oC – 8oC. The rabies virus is easily killed by sunlight and ultraviolet rays, the influence of acidic and alkaline states, fatty solvents such as ether and chloroform, Na deoxychlorate, and soapy water (Akoso, 2007). It is therefore very important to wash the wound with soap for 15 minutes as soon as possible after the bite to kill the rabies virus that is around the bite wound. This virus infects humans through contact with the saliva of suspected rabies animals. Contact can be through bite and non-bite (scratching or licking of the mucosa or open skin) by infected animals (dogs, monkeys, cats, wolves, bats). The incubation period for rabies varies greatly between 2 weeks - 2 years, but is generally 3 - 8 weeks. According to WHO, it is stated that the incubation period is on average 30 - 90 days. The difference in the incubation period is influenced by the type/strain of the rabies virus, the amount of virus entered, the depth of the bite wound, the location of the bite wound, the number of nerves in the wound area, and the immunity of the sufferer (Organization, 2012).

Rabies has several stages of its clinical symptoms, namely the prodromal stage, sensory stage, excitation stage, and paralyzed stage. In the prodromal stage, the initial symptoms are fever, malaise, nausea and pain in the throat for several days. In the sensory stage, complaints of pain, heat, tingling at the site of the scar are obtained, followed by symptoms of anxiety and excessive reaction to sensory stimuli. Meanwhile, in the excitation stage, the patient appeared confused, restless, experienced hallucinations, appeared frightened accompanied by changes in behavior to aggression, increased muscle tone and sympathetic activity, hyperhidrosis, hypersalivation, hyperlacrimation, and pupil dilation. This stage is also accompanied by various phobias (such as hydrophobia, aerophobia, photophobia). At this stage, apnea, cyanosis, convulsions, and tachycardia can occur. As many as 80% of rabies sufferers will experience the excitation stage and the duration is 7 days with an average of 5 days. In the last stage, the paralysis stage, is another form of paralytic rabies. Symptoms at this stage are muscle paresis that occurs progressively starting from the scar to causing paralytic respiratory muscles and heart. Patients with paralytic symptoms are often misdiagnosed and unreported. The duration is usually 13 days, longer than the excitation type (Kemenkes, 2019).

In accordance with what was found in this case, the patient's anamnesis has a history of biting, scratching or contact with dogs, cats, or other animals that are positive for rabies (the results of the brain examination of the suspect animal), dead within 10 days of biting instead of being killed, cannot be observed after biting (killed, run, and so on), or suspected rabies (animals change their nature, are lazy to eat, etc.). On physical examination, the bite wound had healed and even been forgotten. Then symptoms were found according to the stage of excitation in the patient. The diagnosis is established with a history of bites and the biting animal dies within 1 week. Symptoms of the early phase are not typical such as flu symptoms, malaise, anorexia, sometimes paresthesia is found in the bite area. Further symptoms include agitation, fluctuating consciousness, persistent high fever, pain in the pharynx sometimes such

as suffocation (inspiratory spasm), hypersalivation, seizures, hydrophobia and aerophobia (Perdossi, 2016).

Prevention of rabies in humans is to manage rabies-infected animal bite wounds by washing wounds, administering antiseptics, administering VAR and SAR. Wound washing is carried out with the aim of killing the rabies virus that is around the bite wound. The outer sheath of the rabies virus is dissolved by soap so that the virus can be inactivated. Wound washing is carried out as soon as possible with soap under running water for approximately 15 minutes. Wound washing does not use equipment because it is feared that it can cause new wounds where the virus will increasingly enter inside. After that, patients with HPR bite wounds are immediately taken to the health center or hospital that becomes the Rabies Center to get further management. The administration of antiseptics is carried out to kill the rabies virus that still remains around the bite wound. Antiseptics that can be given include povidone iodine, 70% alcohol, and other antiseptic substances. The administration of VAR aims to awaken the immune system in the body against the rabies virus and it is hoped that the antibodies formed will neutralize the rabies virus. However, if the rabies virus has reached the central nervous system, VAR administration will no longer provide benefits. The provision of VAR and SAR requires several considerations according to the flowchart in Figure 2 (Kemenkes, 2019).

High-risk wounds are licks/wounds on the mucosa, wounds above the shoulder area (neck, face and head), wounds on the fingers and toes, wounds in the genitalia area, wide/deep wounds, or multiple wounds. Low-risk wounds are licks on open skin or scratches/bites that cause abrasions (excoriation) in the body, hands and feet area (Kemenkes, 2019). SAR is intended for people with high-risk injuries or category III injuries caused by animals that are indicated to be high in rabies. The goal is to provide passive immunity in the first 7 days where at that time immunity to the rabies virus has not been formed. The award was carried out at the same time as the award of VAR on day 0. There are 2 types of SAR, namely homologous serum and heterological serum. Homologous serum (derived from human serum) was given as much as 20 IU/kgBB injected infiltrated into as many wounds as possible, the rest was injected IM. If the heterologous serum (derived from horse serum) is 40 IU/kgBB, a skin test needs to be done first (Perdossi, 2016).

There are several options for post exposure prophylaxis in rabies cases. The first PEP is Purified Vero Rabies Vaccine/PVRV (Verorab ®) with a dose of 0.5 ml for both adults and children. This PEP is injected intramuscularly (IM) in the upper arm area (deltoid) or in the anterolateral thigh region (children under 1 year of age). On day-0, 2 doses were given either on the right and left upper arm or on the right and left thigh in children < 1 year old, followed by 1 dose on day 7 and 1 dose on day 21. Another PEP is the Purified Chick Embriyo Cell-culture vaccine/PCECV (Rabipur®) with a dose of 1 ml injected intramuscularly. The time for giving PEP is the same as PVRV. If the patient with complete VAR has been bitten with a bite time of <3 months, then the patient does not need vaccination. If the bite time is 3-12 months ago, even though the VAR is complete, then 1 dose of vaccination is given. Meanwhile, if the patient with complete VAR is bitten >12 months, the patient needs a complete vaccination. 2] Suspected rabies sufferers were immediately referred to the hospital. Before being referred to

Ringer lactate or NaCl 0.9% infusion. If necessary, give anticonvulsants and the patient should be fixed during the trip. Be aware of the sufferer's irrational and sometimes aggressive behavior. In the hospital, the patient is treated in an isolation room. Medical measures and medication administration are symptomatic and supportive, including antibiotics when needed. To avoid the possibility of transmission from the sufferer, when treating rabies patients, health workers should wear gloves, goggles and masks and fix the patient in bed. If the medical officer or paramedic who treats rabies patients, has never received the anti-rabies vaccine and does not wear personal protective equipment and then gets vomit or saliva from the patient on the open skin or mouth/eye mucosa, it is recommended to get rabies prevention procedures (Kemenkes, 2019).

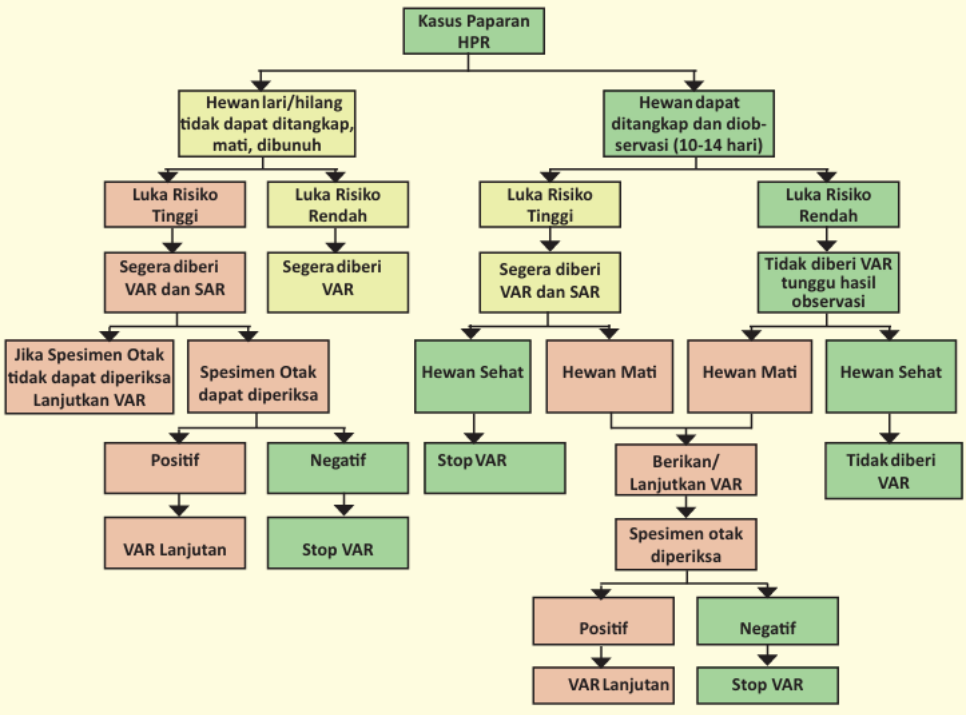

Figure 2. VAR and SAR *Flowchart* (Kemenkes, 2019)

CONCLUSION

This case describes a 41-year-old male who developed classic rabies symptoms—including recurrent vomiting, dysphagia with hydrophobia, paresthesia, aerophobia, and agitation—three months after a dog bite to his left index finger, without receiving any post-exposure prophylaxis (PEP). The absence of wound care, rabies vaccination, or rabies immunoglobulin placed him at high risk for developing rabies, ultimately resulting in a fatal outcome. This case underscores the urgent need for increased public and clinical awareness about rabies transmission and the critical importance of immediate wound management and PEP after exposure. It highlights the necessity for standardized protocols at all healthcare levels and advocates for enhanced public education and improved access to rabies vaccines and immunoglobulin, especially in primary care settings. Future research should focus on evaluating the effectiveness of targeted educational interventions and access-improving strategies in reducing rabies incidence and mortality in high-risk populations.

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