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A case of an atypical course of rabies

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The aim of this study was to perform a clinical analysis of an atypical course of rabies in a 68-year-old female patient.

Materials and methods. A retrospective analysis of the medical records of the 68-year-old woman who died from the paralytic form of rabies was conducted. The patient was hospitalized at the Municipal Non-Profit Enterprise "Regional Infectious Diseases Clinical Hospital" of the Zaporizhzhia Regional Council from April 19 to April 29, 2024. The diagnosis of rabies was confirmed post-mortem by detection of rabies virus RNA in brain tissue using polymerase chain reaction (PCR), as well as by morphological verification of Babes–Negri inclusion bodies in the brain tissue.

Results. This clinical case describes an atypical (paralytic) form of rabies in the 68-year-old patient. The initial clinical presentation lacked classical manifestations such as agitation, aerophobia, and hydrophobia. The disease onset was characterized by flaccid paralysis, which progressively evolved into tetraparesis and other neurological signs indicative of encephalitic brain involvement. This atypical presentation significantly complicated the diagnostic process and resulted in the establishment of only symptomatic diagnoses during life. Suspicion of rabies arose only after clarification of the epidemiological history, which revealed that 2–3 months before the onset of symptoms the patient had been bitten by a dog of unknown vaccination status that died two days after the incident. Definitive confirmation of rabies was obtained during the post-mortem pathological examination through detection of rabies virus RNA in brain tissue using PCR and identification of Babes–Negri inclusion bodies. In our opinion, the most likely cause of infection and the atypical disease course was the administration of an incomplete course of post-exposure rabies prophylaxis. This observation highlights the critical importance of strict adherence to the anti-rabies prophylaxis protocols recommended by the World Health Organization.

Conclusions. This clinical case demonstrates that an atypical (paralytic) course of rabies may mimic other neurological syndromes, particularly in the absence of a clearly established epidemiological history. Such circumstances pose significant challenges to timely and accurate diagnosis. Incomplete post-exposure rabies prophylaxis was identified as the cause of the fatal outcome and is considered the probable reason for the atypical clinical course of the disease.

Ключові слова: сказ, атиповий перебіг, постконтактна профілактика.

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Випадок атипового перебігу сказу

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Мета роботи – здійснити клінічний аналіз атипового перебігу сказу у пацієнтки віком 68 років.

Матеріали і методи. Ретроспективно проаналізовано історію хвороби померлої від паралітичної форми сказу (вік хворої – 68 років). Пацієнтка перебувала на лікуванні в КНП «Обласна інфекційна клінічна лікарня» ЗОР з 19.04.2024 року до 29.04.2024 року. Діагноз підтверджено посмертно за результатами виділення РНК вірусу сказу методом полімеразної ланцюгової реакції з тканини мозку та шляхом морфологічної верифікації в тканинах головного мозку тілець Бабеша–Негрі.

Результати. У наведеному клінічному випадку проаналізовано атиповий (паралітичний) варіант перебігу сказу у пацієнтки віком 68 років. Клінічні прояви сказу маніфестували без класичних ознак збудження, аеро- та гідрофобії. Виникнення в дебюті захворювання млявих паралічів із поступовим розвитком тетрапарезу та інших ознак енцефалітичного ураження головного мозку призвело до ускладненого діагностичного пошуку з формулюванням лише симптоматичних діагнозів. Діагноз сказу у пацієнтки припустили лише після того, як з'ясувалося, що за 2–3 місяці до розвитку клінічних симптомів її вкусив собака з невідомим вакцинальним статусом, який через два дні по тому помер. Діагноз сказу підтверджено під час патологоанатомічного дослідження тіла внаслідок виділення з тканин головного мозку померлої РНК вірусу сказу методом полімеразної ланцюгової реакції та морфологічної верифікаці тілець Бабеша–Негрі. Імовірною причиною розвитку інфекції та її атипового перебігу став незавершений курс постконтактної профілактики сказу, що вказує на критичну важливість дотримання рекомендованого експертами Всесвітньої організації охорони здоров'я протоколу антирабічної допомоги.

Висновки. Згідно з результатами аналізу клінічного випадку, в разі розвитку атипового (паралітичного) перебігу сказу його маскування під інші неврологічні синдроми при нез'ясованому епідеміологічному анамнезі хвороби значно ускладнює та відтерміновує встановлення правильного діагнозу. Недотримання чинного протоколу постконтактної профілактики сказу стало ймовірною причиною атипового перебігу захворювання та настання летального наслідку.

Rabies is one of the most dangerous zoonotic infectious diseases, reported in more than 150 countries worldwide, and results in approximately 60,000 deaths annually, about 40 % of which occur in children under 15 years of age [1].

The causative agents of rabies are RNA-containing viruses of the genus *Lyssavirus*, family *Rhabdoviridae*. Both domestic and wild animals can serve as reservoirs of infection, including dogs, cats, foxes, wolves, raccoons, badgers, and bats [2]. Globally, dogs, particularly stray animals, represent the primary threat to humans, accounting for nearly 99 % of human rabies cases [3].

In Ukraine, due to the ongoing full-scale military aggression, the epidemiological situation regarding rabies has significantly deteriorated and acquired specific features [4]. On the one hand, the destruction of veterinary and hunting infrastructure in war-affected regions, together with the migration of wild animals from combat zones, has disrupted the established ecological balance. This disruption has resulted in an almost threefold increase in reported rabies cases among animals and nearly a twofold rise in the number of identified disease foci between 2022 and 2024 [5]. The number of medical consultations related to bites inflicted by rabid animals increased from 1,845 cases in 2023 (4.5 per 100,000 population) to 2,427 cases in 2024 (5.86 per 100,000 population) [5,6].

Moreover, in 2024, four fatal human rabies cases were registered in Ukraine: three in the Kharkiv region and one in the Dnipropetrovsk region. This figure markedly exceeds the incidence observed in previous years [6,7]. For comparison, the same number of human rabies cases had been recorded in the Kharkiv region over the entire period from 2014 to 2024 [7].

On the other hand, systemic changes in Ukraine's ecological balance have led to a shift in the structure of animal rabies cases. In 2024, almost 50 % of all registered rabies cases among animals involved cats [7]. A distinctive "Ukrainian phenomenon" has therefore emerged: three out of four fatal human rabies cases reported in 2024 were caused by injuries inflicted by infected cats [7].

Rabies may manifest in two clinically distinct forms: the typical (encephalitic) form, characterized by pronounced psychoneurological excitation, and the atypical (paralytic) form, which may mimic poliomyelitis or Guillain–Barré syndrome [8]. In cases of the typical disease course, characteristic clinical manifestations, such as agitation, aerophobia, and hydrophobia, usually allow for a relatively straightforward clinical diagnosis. However, when rabies begins with flaccid paralysis and other signs of encephalitic involvement, including psychiatric disturbances, dizziness, and balance disorders, clinical diagnosis may be considerably complicated. Furthermore, in the absence of epidemiological evidence of traumatic contact with animals or bats, the diagnosis of rabies may not be established at all [9].

The rabies virus is a bullet-shaped particle measuring 75–180 nm and causes irreversible damage to the central nervous system of all mammals, including humans, with a case fatality rate approaching 100 % [8,9,10]. Currently available therapeutic approaches for this fatal disease, including the Milwaukee and Recife protocols, have not demonstrated consistent success; according to the literature, only about 20 patients worldwide have survived rabies [8]. Therefore, urgent post-exposure prophylaxis remains the only reliable means of protection. Since its development in 1885 by Louis Pasteur, the rabies vaccine has enabled approximately 15 million people each year to receive life-saving post-exposure prophylaxis [8,10].

Aim

The aim of this study was to conduct a clinical analysis of an atypical course of rabies in a 68-year-old female patient.

Materials and methods

A retrospective analysis of the medical records of the 68-year-old woman who died from the paralytic form of rabies was performed. The patient was hospitalized at the Municipal Non-Profit Enterprise "Regional Infectious Diseases Clinical Hospital" of the Zaporizhzhia Regional Council (MNE "RIDCH" ZRC) from April 19 to April 29, 2024.

The diagnosis of rabies was confirmed post-mortem by detection of rabies virus RNA in brain tissue using polymerase chain reaction (PCR; protocol No. 1352 dated May 2, 2024), as well as by morphological verification of Babes–Negri inclusion bodies in brain tissue. The post-mortem examination was carried out by the Municipal Institution "Zaporizhzhia Regional Bureau of Forensic Medical Examination" of the ZRC.

Clinical observation. Patient T., born in 1956 and residing in the Dnipropetrovsk region, was admitted to MNE "RIDCH" ZRC with impaired consciousness, scoring 13–14 points on the Glasgow Coma Scale (GCS). She was partially disoriented in time and space, exhibited delirium, and remained lethargic but calm. Neurological examination revealed tetraparesis with monoplegia of the right upper limb.

According to the medical history, the disease had an acute onset on April 15, 2024, beginning with numbness and pain in the right forearm, accompanied by general weakness and fatigue. The patient attributed these symptoms to excessive physical exertion. On the same day, she was examined by a neurologist and diagnosed with right-sided upper limb neuropathy with pain syndrome, for which symptomatic treatment was prescribed.

However, on April 16, 2024, the patient's condition deteriorated. She developed headache, dizziness, nausea, and vomiting, and her arterial blood pressure increased to 180/100 mmHg. With suspected acute cerebrovascular accident, she was transferred to the Zaporizhzhia Regional Clinical Hospital, where she was examined by a neurologist and a cardiologist. Computed tomography (CT) of the brain was performed. Based on clinical and instrumental findings, the diagnosis of hypertensive disease, grade II, stage 3, risk 4, complicated by hypertensive crisis with cephalgic syndrome, was established. Outpatient treatment at her place of residence was recommended.

On April 17, 2024, due to intensifying pain, progressive weakness of the right upper limb, and recurrent dizziness, the patient sought medical care at the Limited Liability Company "VITACENTER" (Zaporizhzhia). Neurological examination revealed instability in the Romberg position and impaired coordination test performance. Muscle tone and tendon reflexes in the right upper limb were decreased,

Table 1. Dynamics of acute-phase inflammatory markers and body temperature in the 68-year-old female patient T.

Parameter, units	Date					
	19.04	22.04	23.04	25.04	27.04	28.04
Temperature, °C	37.5	38.8	37.5	39.3	39.6	38.1
C-reactive protein, mg/L	1.6	95.4	128.5	158.8	–	–
Procalcitonin, ng/mL	0.11	–	0.53	0.53	–	–
Leukocytes, $\times 10^9$/L	7.0	10.7	11.2	8.9	9.3	14.0
Band neutrophils, %	3	3	5	7	12	13
Segmented neutrophils, %	87	81	83	72	70	78
Lymphocytes, %	8	11	8	16	14	7
Monocytes, %	2	5	4	5	4	2
Erythrocyte sedimentation rate, mm/h	6	8	17	28	62	56

Table 2. Multiple organ failure assessment according to the Sequential Organ Failure Assessment (SOFA) scale

Affected system / Organ	Date / Score				
	19.04	20.04	23.04	27.04	29.04
Respiratory	0	3	2	4	4
Coagulation	0	0	0	0	0
Liver	0	0	0	0	0
Cardiovascular	0	4	4	4	4
Central nervous system	1	3	4	4	4
Renal	0	0	0	1	3
Total SOFA score	1	10	10	13	15

with limited active movements in proximal muscle groups (muscle strength graded 4/5 on the Medical Research Council scale) and severe weakness of the hand (1/5). The right hand was flaccid; wrist dorsiflexion was limited, and opposition of the thumb to the fourth and fifth fingers was absent. No abnormalities in muscle tone or reflexes of the lower limbs were observed.

On an emergency basis, the patient was hospitalized in the Neurology Department with a diagnosis of acute neuropathy of the right radial nerve with paresis of the right hand. Despite ongoing treatment, her condition continued to worsen: the level of consciousness decreased to 13 points on the GCS, neuromuscular conduction impairment progressed to tetraparesis, and hyperthermia developed.

On April 19, 2024, relatives reported that on February 5, 2024, the patient had been bitten by a dog that she had recently taken into her household. The dog inflicted a bite wound to the right forearm. The wound underwent primary surgical debridement, and the first dose of anti-rabies vaccine was administered; however, the patient failed to complete the full vaccination course.

Following receipt of this epidemiological information, the patient was examined by an infectious disease specialist. Based on the combination of clinical presentation and epidemiological history, atypical paralytic rabies was suspected, and the patient was transferred to the MNE "RIDCH" ZRC.

An urgent notification regarding a probable, unconfirmed case of rabies (paralytic form with severe course) was submitted to the regional sanitary-epidemiological service on April 20, 2024.

At the time of admission to the MNE "RIDCH" ZRC, the patient was able to communicate but remained disoriented in time and place and exhibited intermittent delirium. Tetraparesis persisted, with complete absence of voluntary movements in the right upper limb.

Contrast-enhanced brain magnetic resonance imaging (MRI), performed on April 20, 2024, demonstrated signs of cerebral microangiopathy superimposed on moderate cerebral and cerebellar atrophy. Additionally, a porencephalic cyst was identified in the right frontal lobe with associated perifocal gliosis, suggestive of post-ischemic sequelae.

Later the same day, the patient's level of consciousness declined, reaching a score of 9 on the GCS. Her breathing became rapid and shallow. As part of intensive care management, mechanical ventilation was initiated, and vasopressor support with a norepinephrine infusion was commenced.

A complete blood count showed a normal white blood cell count (7.9 × 10^9/L), with the following differential: 3 % band forms, 73 % segmented neutrophils, 19 % lymphocytes, and 5 % monocytes. The erythrocyte sedimentation rate was 6 mm/h.

On April 23, 2024, an ophthalmoscopic examination revealed early signs of retinal angiosclerosis in both eyes. A lumbar puncture was subsequently performed. The cerebrospinal fluid was clear and colorless, with a cell count of 4 cells/μL (100 % lymphocytes). Cerebrospinal fluid chemistry showed protein at 0.165 g/L, glucose at 3 mmol/L, and chlorides at 118 mmol/L. PCR testing of the cerebrospinal fluid for *Mycobacterium tuberculosis*, herpes simplex virus types 1 and 2, cytomegalovirus, and Epstein–Barr virus was negative. Bacteriological cultures yielded no growth.

Subsequently, the patient's condition continued to deteriorate, characterized by febrile hyperthermia *(Table 1)* and progressive multiple organ dysfunction syndrome *(Table 2)*.

On April 29, 2024, the patient experienced cardiac arrest. Despite cardiopulmonary resuscitation efforts, the patient was pronounced dead.

Post-mortem examination revealed advanced cerebral edema with herniation of the brainstem and cerebellar tonsils through the foramen magnum, accompanied by partial brainstem necrosis.

Histopathological analysis identified Babes–Negri bodies within the neurons, and rabies virus RNA was detected in the brain tissue via molecular testing.

Discussion

Annually, approximately 60,000 people die from rabies infection [11]. Lyssaviruses enter the human body through saliva-contaminated wounds caused by contact with infected animals or via direct inoculation into mucous membranes. Potential sources of infection include all warm-blooded animals, including humans [11]. However, the greatest threat to humans is posed by dogs, which account for more than 95 % of all rabies-related fatalities [11,12,13]. Furthermore, with the increasing prevalence of organ transplantation, sporadic cases of rabies transmission from infected donors, whose diagnosis was not established antemortem, have been reported [10,14].

Depending on the risk of exposure, the WHO classifies animal contact into three categories. Category I involves the licking of intact skin, touching, or feeding the animal; these

contacts are considered safe as the rabies virus cannot penetrate intact skin, and post-exposure prophylaxis (PEP) is not indicated [10]. Category II includes minor bites or scratches on exposed skin without bleeding [10]. Category III encompasses single or multiple transdermal bites or scratches, contamination of mucous membranes or broken skin with saliva, and direct contact with bats [10].

Rabies is a disease with a nearly 100 % case fatality rate [15,16]. Once the virus enters the body, it infects the peripheral nervous system and, via retrograde axonal transport, reaches the central nervous system, causing severe encephalomyelitis [10,16]. Death typically occurs within 14 days of clinical onset [17]. The only effective prevention after high-risk exposure is immediate PEP, which involves thorough wound care, rabies vaccination, and, if indicated, rabies immunoglobulin [10,18,19].

WHO experts recommend immediate and thorough wound washing for at least 15 minutes with soap and water, followed by disinfection with substances proven to inactivate the virus, such as alcohol, iodine, or hydrogen peroxide [10]. Proper initial wound care can prevent the disease in approximately one-third of cases [18]. Since specific neutralizing antibodies typically develop 7–14 days post-vaccination, immediate protection requires combining vaccination with local infiltration of rabies immunoglobulin at the wound site to neutralize the virus and prevent further dissemination [10,19].

In the case presented, rabies developed following traumatic contact with a stray dog, resulting in multiple bites on the patient's right forearm. According to WHO guidelines, these injuries were Category III. Recommended management included thorough surgical debridement, extensive irrigation with soap and water, infiltration of rabies immunoglobulin in and around the wound, and a complete rabies vaccination schedule [10]. It is unknown whether the patient performed initial wound washing; she received only basic surgical care and the first vaccine dose. Subsequently, the mandatory 10-day animal observation was not organized, and the patient failed to present for further PEP. Per WHO recommendations, if an animal dies during the observation period, biological materials must be tested for rabies, and exposed individuals must complete a five-dose intramuscular vaccination schedule [10].

The incubation period depends on the viral inoculum, replication activity, and proximity of the wound to the central nervous system, ranging from five days to several years (typically 2–3 months; exceeding one year in 2–3 % of cases) [10,20]. In this clinical case, the incubation period ranged from 2 to 3 months.

Typically, the first specific clinical symptom is neuropathic pain or paresthesia at the exposure site, caused by the inflammatory response to viral replication in the dorsal root ganglia [10]. The full clinical picture usually develops within 2–3 days and follows either the classical (furious) form, progressing through stages of excitement and paralysis, or an atypical (paralytic) form [21,22]. In the classical form, 50–80 % of patients exhibit hallmark symptoms such as hydrophobia and aerophobia, facilitating early diagnosis [10,21].

However, when the disease manifests as flaccid paralysis mimicking Guillain–Barré syndrome or acute motor-sensory axonal neuropathy, and the exposure history is unknown, diagnosis is extremely challenging [9,10,22]. Therefore, the WHO recommends including rabies in the differential diagnosis of any acute progressive encephalitis of unknown etiology [10].

The atypical form occurs in 20–30 % of cases and is more frequent in individuals who received incomplete PEP. In the case described, the patient received only a single vaccine dose. Approximately 2.5 months later, she developed pain, numbness, and weakness at the bite site. Over the following days, neurological deficits progressed to tetraparesis and generalized weakness, accompanied by signs of encephalitis: hyperthermia, postural instability (positive Romberg test), impaired coordination, and a decreased level of consciousness (scoring 13 on the GCS).

Neuroimaging (CT and MRI) performed within the first five days of illness revealed no focal pathological lesions despite clinical encephalitis. Literature suggests that rabies virus infection induces non-specific moderate contrast enhancement, which typically becomes apparent only in the late stages when the patient is comatose [10]. In this instance, the lack of specific neuroimaging features and the initial absence of an epidemiological history hindered early definitive diagnosis. Suspicion of paralytic rabies was only established after identifying the history of a dog bite 2–3 months prior and the subsequent death of the animal.

Conclusions

1. This clinical case highlights an atypical presentation of rabies, which initially manifested as isolated paralytic involvement of the right upper limb.

2. The diagnostic process was significantly obscured by the dominance of progressive flaccid paralysis and signs of encephalitic brain involvement in the absence of pathognomonic clinical features (such as hydrophobia or aerophobia). Consequently, during the early stages of the disease, only a non-specific symptomatic diagnosis could be established. Clinical suspicion of rabies was only raised following a meticulous clarification of the patient's epidemiological history.

3. We hypothesize that the atypical, protracted course of the disease was associated with the incomplete administration of the post-exposure prophylaxis regimen.

Prospects for further research. Future research should focus on an in-depth investigation of the molecular pathogenesis underlying fatal neurological complications in rabies. Furthermore, there is a critical need to evaluate various prevention strategies and therapeutic interventions for cases where PEP is delayed or partially administered.

Ethical approval

The material presented in the article is an original research work of the Department of Infectious Diseases of Zaporizhzhia State Medical and Pharmaceutical University and is a retrospective analysis of the medical history of the 68-year-old woman who died from paralytic rabies, who was treated at the MNE "RIDCH" ZRC from 19.04.2024 to 29.04.2024. Excerpt from minutes No. 10 of the meeting of the bioethics commission of Zaporizhzhia State Medical and Pharmaceutical University dated 18.09.2025.

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