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Effectiveness of the Milwaukee Protocol in Human Rabies: A Review

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Abstract

Introduction

Human rabies remains almost invariably fatal once clinical symptoms appear, despite widespread use of pre- and post-exposure prophylaxis. The so-called “Milwaukee Protocol” — a structured regimen of therapeutic coma, NMDA antagonists, antivirals and supportive care — generated intense interest after a single widely publicized survivor in 2004.

Aim of the study

This review summarizes current evidence on treatment of symptomatic human rabies with emphasis on the Milwaukee Protocol.

Materials and Methods

We synthesized published case reports, experimental studies, and review articles addressing therapies of clinical rabies and the Milwaukee Protocol. Key outcomes were survival, functional recovery, and reported adverse events. Limitations of the evidence base are highlighted.

Results

Documented recoveries prior to 2004 were rare but reported under intensive supportive care. The original Milwaukee Protocol case displayed substantial recovery and prompted iterative protocol versions. Subsequent application of the protocol worldwide has produced numerous reported failures; published, verifiable survivor accounts after 2004 remain scarce. Experimental data provide limited mechanistic support for core elements of the protocol. Mortality has not been convincingly reduced beyond outcomes achievable with modern intensive supportive care alone.

Conclusions

Current evidence does not conclusively support the Milwaukee Protocol as an effective, reproducible curative treatment for clinical rabies. High-quality clinical reporting and standardized case registries are needed before the protocol can be eventually recommended or totally abandoned. Meanwhile, prevention through vaccination of dogs and timely post-explosure prophylaxis remains the cornerstone of rabies control.

Key words: rabies, human rabies, rabies survivors, milwaukee protocol, critical care, therapeutic coma, amanatadine, ketamine, treatment efficacy

Introduction

Even though many rabies control efforts are taken all around the world and both pre-exposure (PrEP) and post-exposure prophylaxis (PEP) is available, the disease still remains deadly – mainly in developing countries of Africa and Asia. The most current estimates suggest around 44 000 human deaths each year in over 150 countries. Furthermore, it is important to acknowledge that human rabies remains substantially underreported in many regions of the world. [1]

Rabies is a zoonotic disease caused by rabies virus (RABV) - a single stranded RNA virus, a part of the Rhabdoviridae family of viruses and genus *Lyssavirus*. It is transmitted through direct contact between human mucosal surfaces (such as open wounds or the eyes) and the saliva, urine, sweat, or neural tissues of an infected animal. Dogs are responsible for the vast majority of human rabies infections, accounting for around 99% of cases; however, transmission can occur from any infected mammal. [2]

The virus attacks central nervous system and causes an acute progressive encephalitis. Firstly it migrates from the site of the entry into the peripheral nerves, eventually reaching the brain, where it replicates and causes cerebral damage. Later it spreads centrifugally along the autonomic and sensory nerves to various organs – for example heart, skin and salivary glands. [3] [4]

The incubation period usually lasts 1–3 months, but it may fluctuate between a few days and even over a year. The disease has 2 main forms - encephalitic (furious or classical) and paralytic (dumb) rabies. The encephalitic form is more common (80% of cases) and is characterized by generalized hyperexcitability with autonomic dysfunction - hypersalivation, sweating, piloerection - and unique symptoms such as hydrophobia and aerophobia. Eventually the disease leads to quadriparesis, coma and death caused by cardiopulmonary complications and multiple organ failure.

Patients with the paralytic rabies live approximately 41% longer than patients with encephalitic form. These patients are initially often alert, the weakness of muscles usually starts from the wound site and progresses gradually to quadriparesis.

Both forms of the disease are almost invariably fatal, with death typically occurring within 14 days of symptom onset. No established treatment exists for rabies once clinical symptoms appear. [3][5]

The aim of this study is to summarize the current knowledge on the treatment of rabies in patients who have already developed clinical symptoms, with particular emphasis on the most structured treatment approach—the Milwaukee protocol. [6]

Materials and Methods

This narrative literature review was conducted using structured searches of PubMed and Google Scholar. Search terms included: "human rabies", "milwaukee protocol","rabies treatment" and "rabies survivors". Peer-reviewed case reports, experimental studies, reviews, and clinical guidelines addressing symptomatic human rabies treatment were included. Studies focusing solely on prophylaxis without treatment of clinical cases were excluded. Data on survival, functional outcomes, and therapeutic interventions were extracted, organized, and synthesized into a narrative overview highlighting current evidence, gaps, and areas of ongoing debate. AI-assisted language tools were used during manuscript preparation to enhance grammatical accuracy, clarity, and coherence. These tools were employed solely for stylistic refinement and did not influence the interpretation of data or the conclusions of the review.

Limitations in Current Rabies Therapeutic Research

There are several reasons why an effective treatment for rabies has not yet been developed.

Although rabies continues to cause numerous deaths worldwide, the disease is rare in high-income countries due to the effectiveness of pre- and post-exposure prophylaxis. As a result of this rarity, relatively few resources are devoted to research. [7][8]

Therapeutic development is further complicated by the biological diversity of rabies viruses. Numerous RABV variants circulate simultaneously— for example only in the United States: raccoon, skunk, and multiple bat variants. [9]

Progress is also hampered by an incomplete understanding of RABV pathogenesis and mechanisms of neuronal damage. Experimental models often rely on attenuated viruses rather than “street” viruses, and these differ substantially in EEG patterns, REM sleep alterations and

disease course. As a result, laboratory discoveries may not translate effectively to naturally occurring infections.

Furthermore drug delivery to the central nervous system remains difficult due to the blood–brain and spinal cord barriers. Achieving therapeutic concentrations of drugs in the CNS in vivo poses a significant challenge. [8]

The First Documented Survivals

The first well-documented human case of recovery from rabies was published in 1972. A 6-year-old boy was bitten by a bat. A 14-day course of vaccination was initiated 4 days after the bite. Incubation period lasted 20 days. The treatment focused on prevention of all curable rabies complications. Aggressive supportive care included tracheostomy, tracheal suctioning, oxygen therapy (respiratory difficulties, prevention of hypoxia), withdrawal of intraventricular fluid (increase of intracranial pressure) and anticonvulsant therapy (focal seizure). No specific antirabies medication was applied. The patient recovered without long-lasting complications. [10]

From 1972 to 2004, four other case reports [11][12][13][14] documented patients who survived rabies. All of these patients received conventional care and continued to display persistent cerebellar and striatal signs. [15] At least 2 of these patients had received prophylactic rabies vaccine before the onset of illness.

Milwaukee Protocol (MP)

In 2004, in Wisconsin (USA) a 15-year-old girl developed rabies one month after being bitten by a bat. No rabies preexposure or postexposure prophylaxis was given. The patient managed to survive the disease. [16] According to follow-up report, over 2 years after the exposure, the patient had no trouble carrying out complex daily tasks, including driving a car and studying. She experienced only mild neurological deficits such as variable dysarthria and gait problems, along with occasional sensations of cold in her feet. [15]

Her treatment — later called the Milwaukee Protocol — gained widespread attention and was promoted as a novel therapeutic option for symptomatic patients with human rabies.

Therapeutic coma was induced using intravenous midazolam and adjunctive phenobarbital to sustain a burst-suppression pattern on the electroencephalogram. Management included NMDA-receptor antagonists such as a continuous IV ketamine infusion and enteral amantadine. Antiviral treatment with intravenous ribavirin was also provided. [17]

Since 2005, the Milwaukee protocol has been gradually revised based on outcomes from new patients.

The second version of the MP (2007) involved introducing tetrahydrobiopterin (BH4) supplementation into the treatment protocol. BH4 serves as a cofactor for several enzymes, including neuronal nitric oxide synthase (nNOS) and is essential for the synthesis of monoamine neurotransmitters. A deficit of BH4—resulting in a pathological reduction of dopaminergic and serotonergic signaling—was identified and impairment of nNOS function has been shown to cause cerebral arterial constriction. [18][19][20]

In addition, a 7-day sedation protocol was adopted to mitigate dysautonomia, while ribavirin was discontinued because of its immunosuppressive effects, potentially diminishing the antibody-mediated response - critical for recovery from rabies. Barbiturates were also excluded from the treatment protocol due to immunosuppressive properties. [20][7][21]

The next modification of the MP (2009) identified cerebral edema in bat-associated rabies and started the quantification of quinolinate in cerebrospinal fluid using nuclear magnetic resonance. Quinolinate, a potent N-methyl-D-aspartate (NMDA) receptor agonist, is capable of inducing excitotoxic neuronal injury, and its concentrations in rabies patients are higher than those observed in most other neurological infections. According to research [22] in survivors, quinolinate levels decreased over time toward normal ranges, whereas in patients with fatal outcomes, concentrations continued to rise. These observations provided also a mechanistic rationale for including ketamine in the treatment protocol, as it mitigates neuronal injury by inhibiting quinolinate-mediated NMDA receptor activation. [21][22]

Version 4 (2012) reported the occurrence of atrioventricular conduction abnormalities in dog-transmitted rabies and introduced fludrocortisone as prophylaxis against cerebral salt wasting.

In 2017, the protocol was expanded to include bedside diagnostic methods alongside antiviral treatment.

Version 6 (2018) integrated viral lineage, vaccine-related immunologic effects, and patterns of antiviral responsiveness into prognostic modeling.

The most recent iteration of the protocol (2025) targets all seven recognized clinical phenotypes through a combination of antiviral agents and gene-based therapies. These phenotypes, shaped by viral phylogeny, prior exposure to biologic interventions, and the route of infection, exhibit distinct patterns of therapeutic responsiveness. [21]

The current protocol is available online. It starts with a structured decision-making. The decision tree begins by identifying the source of exposure, distinguishing between contact with a dog, a vampire bat, an insectivorous bat, a cat, or a primate such as a marmoset, as well as exposures associated with organ or tissue transplantation. For each exposure category, the next step is to determine whether the patient has previously received rabies vaccination or immunoglobulin. Subsequently, all branches converge on an assessment whether the patient is immunosuppressed. The algorithm directs the clinician toward one of several designated therapeutic pathways within the protocol, labelled from A to K. [6]

Effects of the treatment with the Milwaukee Protocol

Despite the initial widespread hope, the Milwaukee Protocol has been reported to fail in at least 64 documented cases. Meanwhile, at least 34 patients have survived rabies through intensive critical care without the main components of the Milwaukee Protocol. [23]

An online registry of patients treated with the Milwaukee Protocol is publicly available [6]; however, it lacks case-level clinical details, does not clarify the actual number of alleged survivors, and provides no verifiable data in any published form. The protocol's creator has stated that 18 of 117 patients treated by various physicians worldwide reportedly survived, yet almost none [24][25] of these cases has been documented in credible, peer-reviewed publications. Consequently, there is no transparent evidence demonstrating that the protocol is effective. [23][21]

Proponents of the protocol acknowledge that detailed accounts of survivors have not been published. They emphasize that most purported survivors are from low- and middle-income countries (LMICs), where substantial clinical demands, linguistic constraints, and administrative structures frequently impede the preparation of detailed case reports. As a result, they rely on aggregate intention-to-treat data, which they present as evidence of enhanced survival compared with other standard care. [21]

Laboratory Studies on the Milwaukee Protocol

Experimental investigations of the Milwaukee Protocol, both in vitro and in vivo, have provided little support for its efficacy.

Ketamine, a dissociative anesthetic and NMDA receptor antagonist, was initially considered a potential treatment for rabies after early laboratory studies showed that high concentrations inhibited viral replication in vitro and reduced infection in specific brain regions in a rat model. [26] These preliminary findings led to its inclusion in the Milwaukee Protocol. However, later

and more comprehensive in vitro and in vivo investigations failed to confirm any therapeutic benefit. [27]

Amantadine, an NMDA channel blocker, was incorporated into the Milwaukee Protocol following in vitro findings indicating reduced rabies virus production. However, studies with a mouse model did not confirm any therapeutic effect. [28] To date, no antiviral agent has demonstrated clinical efficacy against rabies. [23]

Although therapeutic coma is effective in treating refractory status epilepticus and managing traumatic brain injury by reducing neuronal activity and metabolic demand, its use in rabies lacks a clear scientific rationale. [23] Early hypotheses suggested that excitotoxicity might play a significant role in rabies pathogenesis, leading to the proposal that NMDA receptor antagonists combined with GABAergic agents could provide neuroprotection. However, studies in cell cultures and mouse models have not confirmed this hypothesis. [27]

Conclusions

At present, the medical community remains divided regarding the Milwaukee Protocol. Some advocate for its continued use [21], while others argue that it should be abandoned entirely, as intensive supportive care appears to be the only consistently effective component of the protocol. [23] Although the original 2004 survivor generated global interest, subsequent clinical experience has not confirmed any reproducible benefit of the protocol and no alternative curative therapy for symptomatic human rabies can currently be recommended.

Despite its lack of proven efficacy, the ongoing evolution of the Milwaukee Protocol reflects a meaningful effort to integrate emerging insights into rabies pathogenesis, host immune responses, and viral diversity. Its revisions demonstrate a willingness to adapt therapeutic strategies in response to new experimental findings and clinical observations. Although these adaptations have not yet translated into improved patient outcomes, the protocol's iterative development underscores the broader need for flexible, hypothesis-driven approaches in the pursuit of a future curative therapy.

Progress toward such a therapy will depend on transparent international reporting, standardized registries, collaborative multicenter studies, and improved translational models that more accurately reflect natural infection. Until credible evidence emerges, the Milwaukee Protocol should not be regarded as a proven or recommended therapy.

In parallel, the broader fight against rabies continues. The WHO and its global partners have committed to elimination of dog-mediated human rabies by 2030, reinforcing this goal through

coordinated international action and widespread implementation of both pre- and post-exposure prophylaxis. [1] Nevertheless, the search for a curative treatment must remain a priority, as prevention alone cannot fully address the global burden of this disease.

Disclosure

Author's contribution

Conceptualization: Maciej Kozicki and Karolina Bartkiewicz and Karolina Domosud

Methodology: Julia Kostro and Karolina Bartkiewicz and Karolina Domosud

Investigation: Magdalena Papież and Zofia Jędra and Julia Kostro

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