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Review Article

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Arenavirus in America: a Mini-Review

Mello Aline Hevelin Walder de[1*], Dezorze, Luiz Fernando[2] and Souza, Michele de[3]

[1]*Infectious Diseases Specialist – Universidade Federal de São Paulo – UNIFESP – Brazil*

[2]*Master in Gastroenterology - Universidade Federal de São Paulo – UNIFESP – Brazil*

[3]*Degree in Administration – Universidade Nove de Julho – UNINOVE – Brazil*

[*]Corresponding Author
Mello, Aline Hevelin Walder de, Infectious Diseases Specialist – Universidade Federal de São Paulo – UNIFESP – Brazil.

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Abstract

Arenaviral infections are frequent causes of acute diseases in humans.Some Arenavirus that can cause hemorrhagic fever in America are Junín virus in Argentina, Chapare Virus in Bolívia and Sabiá Vírus in Brazil [1]. Although each one has a distinct geographic distribution, they have common modes of transmission and the diseases associated with them [2]. A problem to prevention and control of Arenavirus is the poor knowledge about the viruses, about the diseases, the ecology and biolgy due the little number of cases until the moment [3].There is a great difficulty in diagnosing Arenavirus infections. These illnesses are insidious and intially indistinguishable from various other common non-specific viral infections [4].

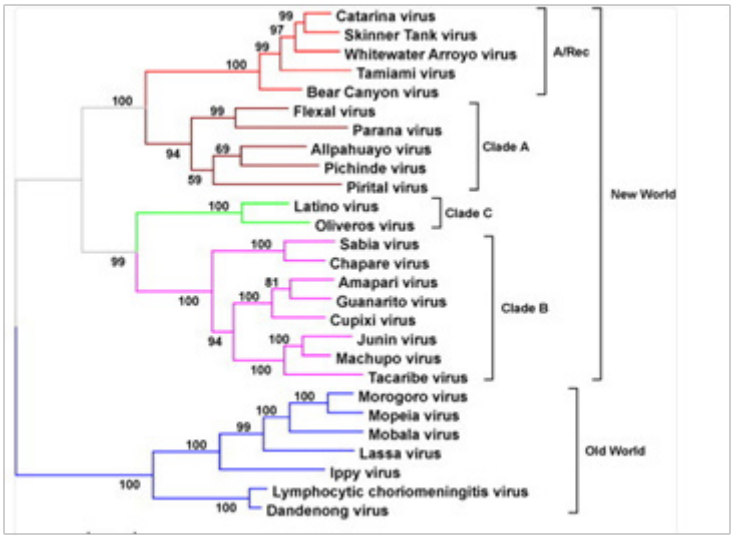
Keywords: Arenavírus, American, Etiology, Patogenesis, Treatment

1. Introduction

The *Arenaviridae* family is classified into three genera: *Mammarenavirus*, *Reptarenavirus* and *Hartmanivirus* [5]. The members of the family Arenaviridae have a single stranded RNA genome composed of two segments and 3400 nucleotides [6]. Most *Mammarenaviruses* have as natural reservoirs rodents and are historically classified into two groups according to their genomic features and antigenic properties [7].

Picture 1: Geographic Distribution of Arenaviruses in the Americas and their Reservoirs.

Source: Vásquez, C et al.



Picture 2: Etiology of Arenavirus.

Source: Emonet et al [6]

2. Metodology

A mini-reviwed was carried out through analysis thirty-eight articles dated 1969 – 2025. Among the reviewed articles, 08 addressed the epidemiology of hemorragic fevers, 05 the pathophysiology/ clinical manifestation, 4 hosts/reservoirs, 01 prophylaxis with immunobiologicals 17 treatment and 03 the histological study through necropsies.

3. Sabiá Virus – Brazil (BzHF)

The idetification of the pathogens, their hosts, and their potential to infect different species, enabling the prevention and tratment of this disease. Sabiá Virus belongs to the family *Arenaviridae* and cause the Brazylian Hemorragic Fever (BzHF) [8].

4. Epidemiology /Historic Context

Until now only six cases was have been recorded in the world. The virus was first isolated in 1990 from a fatal case of hemorrhagic fever in São Paulo State (Southeastern Brazil), Cotia, Jardim Sabia neighboorhood. Five cases ocurred at Brazil and one case at EUA [8].

4.1. Index Case

This index case was a female, 25-year-old, agricultural engineer that lived in a newgborhood Sabiá, (name given to the disease) in Cotia, São Paulo, Brazil. She worked mailly in a office but it is believed that she entered the forest region to carry out research. There is not evidences of a travel outside São Paulo during this period. She was admitted on Jan 12,1990 in the hospital. In the next 03 days she had fever, headache, myalgia, nausea, vomiting, and weakness. She developing neurological symptons as tremors, difficulty in walking, and generalized clonic tremors, acutely ill, somnolent, and mildly dehydrated woman with a very red oropharynx. Due the hemorragic fever the death occur on the fourth day of hospital admission [8].

4.2. The Second Case

A male laboratory technician, a 39 year-old in Brazil, Pará State was infected with the virus in 1992, probably by aerossol. He developed a prolonged influenza-like illness, fever, 38–40°C, chills, malaise, headache, generalised myalgia, sore throat, conjunctivitis, nausea, vomiting, diarrhoea, epigastric pain, and bleeding gums for 15 days [8].This case was not fatal [8]. The technician received as treatment fluid control soon after the first symptoms appeared [8].

4.3. Third Case

The third case happened in 1994. It was an accidental laboratory exposure. During the routine work research scientist at Yale University (USA), 46 years – old was infected with the virus after an accident involving a centrifuge. This patient survived after being hospitalized and receiving the antiviral drug Ribavirin. It is recommended to handle the SABV in biosafety level 4 laboratories [8].

4.4. Fourth Case

The fourth case of SABV infection was reported in 1999 and was naturally acquired. A 32-year-old male coffee-grain machine operator, resident of a rural area (Espírito Santo do Pinhal) of the São Paulo State, presented with a febrile illness. After hospitalization for seven days, the patient died. It was found that the SABV was responsible for the pathological condition of the patient [8].

4.5. Fifth Case

A 52-year-old-man had been hiking in the forest in the city of Eldorado (170 kilometers of São Paulo) and began experiencing symptons such as muscle pain, and dizziness. One day later he developed conjunctivits. Four days later he was hospitalized due a high fever and drowsiness. Due the critical state of healt was transferred to Hospital das Clínicas. During the hospitalization

was trasferrred to Intensive Care Unit (ICU).
He died two days layer [8].

4.6. Sixth Case

A 63-years-old man, a rural workers from Assis (440 kilometers of São Paulo) presented fever, nausea, prostration and generalized mylagia. Eight days later was admitted to the hospital due depressed level of consciousness and respiratory failure. He had ventricular dysfuntion resulted em refractory shock. He died 11 days after sympton onset [8].

5. Hosts

The source of infection in the index case is unknown, but it seems likely that Sabiá virus exists in a rodent reservoir [9]. A serological investigation among rodents that were captured in the city (Espirito Santo do Pinhal) where this case was reported to identify the host rodent species of the SABV.

A total of 412 rodents of 7 different species.:*Necromys lasiurus Akodon* spp, *Calomys tener;Mus musculus; Oligoryzomys nigripe; Bibimys labiosus*, and *Rattus*, were evaluated. However, none of the rodents showed evidence of SABV infection [10].

6. Incubation Period

07 -14 days [11].

7. Transmission

The transmission of SABV to humans is most likely through ingestion or exposure to aerossol generated from virus-containig feces, urine and salive in peri-urban areas where humans and wild rodents coexist [11].

8. Clinical Manifestation

In general, symptoms caused by the SABV are fever, headache, abdominal and epigastric pain, nausea, vomiting, diarrhea, bleeding gums, conjunctival petechiae, conjunctivitis, cough, diarrhea, difficulty walking, hematemesis, hemorrhage, leukopenia, malaise, myalgia, nausea, somnolence, sore throat, tonic-clonic seizures, tremors, vomiting, weakness, and in severe cases, conjunctival petechia, haematemesis and shock due haemorragic manifestations [8].

9. Vaccine

The hypothesis that the Junín virus vaccine could protect against the Sabiá virus (SABV) needs confirmation [8].

10. Lethality Rate

The letality rate is 33% [8].

11. Necropsy Study

• In the liver: enlarged, hepatites (inflamatory infiltrates, mainly in the portal área) macro/ microvascular steatosis;
• In the lung: pulmonary edema, hemorrages and congestion; Did not find myocarditis, encephalitis, nephritis or pancreatitis [8].

12. Junín virus – Argentina (JUNV)

Junin virus cause Argentina Hemorragic fever (AHF) and is endemic in the humid pampas of Argentina [12]. It is an enveloped virus and has RNA as genetic material [12]. Junín virus hemorrhagic fever was first described in 1953 in the city of Bragado.The doctor Rodolfo Arribalzaga made the first clinical description of the disease he callled nephrotic hyperthermia, also known as epidemic malignant fever, O´ Higgins disease or Junín stubble disease [13]. It was finally called Argentina Hemorragic Fever by the doctor Humberto Rugiero, teacher of Infectious Diseases in The Buenos Aires University. The virus was isolated in 1958, on the city of Junín where the first cases of the disease were reported [13]. In 1963 cases of Argentine haemorragic fever (AHF) were confirmed in the south-east of the Province of Córdoba, and between 1964 and 1967 new áreas were detected in the Province of Buenos Aires.Cases began to appear later in the South of Province of Santa Fé [14]. The epidemics have a seasonal distribution, with a peak incidence in the monthy of May [12]. The disease is more prevalente among rural workers than in the urban population [12].

13. Hosts

It is considered a zoonosis linked to a reservoir in wild rodents of the species Calomys musculinis (*Corn mouse*). The Junín virus of the genus Mus musculus was isolated from 52% of rodents. This rodent suffers from a chronic, asymptomatic infection and is very common in peridomestic environments. Females give birth once or more times a year, but populations are renewed annually because these rodents have a lifespan of 7 to 12 months [12].

14. Incubation Period

The 06-14 days [15].

15. Transmission

Calomys rodents shed JUNV in their urine, saliva, and feces, and the animals develop a persistente lifelong infection. Although *C.musculinus* is the primary reservoir of JUNV, the virus was also occasionaly isolated C. laucha, Akodon azarae and Oryzomys flavescens. Humans are infected trough inhalation of aerossol, mucosal exposure or by directed contact of abroaded skin with infectious materials of rodent as urinas, feces and saliva. Person-to-person transmission is rare and can occur through direct contact with body fluids of the infected person. Junín virus has occasionally been isolated from oral swabs and from the urine of patients [15].

16. Clinical Manifestations

An acute febril period of 8-10 days durantion is followed by a prolonged convalescence with loss of hair. During the acute phase there is progressive leucopenia and trombocytopenia. There is a the reduction of the number of platelets, alterations in the protrombin time and reduction in factors II, VII and X. Other manifestation are malaise, anorexia, chills, retroorbital pain, photophobia, dizziness, nausea, vomiting, epigastric pain, constipation or mild diarrhea. There is an impairment of renal function. In the severes cases,instead of the gradual defervescence the neurological and/ or haemorragic manifestation increase.(hemorrages may or may not presente), convulsion, shock and coma. Neurological signs

and symptoms are very common, indicating changes in the central nervous system Usually there are no meningeal signs [15].

17. Vaccine

There is a vacine produced from live attenuated viruses [16].

18. Lethality Rate

20 – 30 % without early treatment [12].

Necropsy study

- In theBrain: Gliosis;
- In the lung:Hemorrhagic focus;
- In the hearth: pericardial and myocardial lymphocitic infiltrates;
- In the liver: formation of hepatic acidophilic bodies;
- In the kidney: hemorrhage and necrosis of the renal papillae.

19. Chapare Virus – Bolivia

Until now cases only have been recorded in Bolívia. The first outbreak was reported in late 2003. The exact number of cases that ocurred in this outbreak remains unclear, and clinical symptons associated with the course of the infection were not recorded in all individuals. There is a reporto f a 22-year-old farmer in the Province of Chapare, Department of Cochabamba with no travel history and no contact with cases of ilness. Initially presented fever, headache, joint stiffness, muscle pain and vomiting. Hemorrhagic signs appeared, resulting in significant deterioration and death of the patient 14 days after the onset of symptoms [17]. Viral samples from this case were preserved and analyzed, revealing that the infecting agente was a previously unknow arenavírus closely related to Sabiá virus [17]. In 2019 a second outbreak ocurred in La Paz department resulting in 9 cases including 4 deaths (case fatality rate: 60%) [17]. This second outbreak was caused by a different strain of CHAPV than one identied in 2003 [17].

The third outbreak ocuured in 2021 in the department of La paz, with 3 confirmed cases (2 fatal) [17]. Another outbreak ocurred in 2024 with one laboratory-confirmed case, also in the department of La Paz [17]. On January 7, 2025 the National Focal Point (NFP) of the International Health Regulations for the Plurinational State of Bolivia notified the World Health. Organization (WHO) of a laboratory-confirmed human infection with Chapare virus (CHAPV) in one of the municipalities of Department od La Paz. The patient was an adult male farmer, aged 50 to 60- years- old. The patient presented with symptons including fever,headache, muscle pain, joint pain and bleeding gums on December 19,2024. He sought medical attention on December 24. On December 30, he was transferred to the local Health Center in the municipality due worsening symptons Where died in the same day.

20. Hosts

Given the detection of CHAPV RNA in Oligorizomys microtis, it is possible that this rodent is a reservoir of CHAPV.

21. Incubation Period

04 – 21 days.

22. Transmission

The virus are transmited to humans through contact with the saliva, urine and droppings of infected rodents. Human-to-human transmission was observed in 3 of 5 cases through suspected nosocomial infection. As evidenced by RT – q PCR detection of viral RNA in sêmen and whole blood of survivors of CHAPV infection from the 2019 outbreak for up to 170 days after symptom onset.

23. Clinical Manifestation

Fever, headache, joint stiffness, muscle pain and vomiting. Hemorrhagic signs (gum, vaginal) and neurological signs: paraparesis.

24. Vaccine

There is not.

25. Lethality Rate

15% to 30% in patients without supportive care. Necropsy study [18].

Tissue analysis of guinea pigs with the Virus identified:

- In the liver: hepatocelular degeneration and necrosis;
- In the gastrointestinal tract: hemorragic ulcerative enterits;
- In the lung: Intersticial pneumonia;
- In the brain: minimal gliosis;
- In the kidney: inflamatory process [18];
- Tissue analysis of cynomolgans macaques with the Virus identified :
- In the liver: Multifocal necrotising hepatites with necrosis of hepatocytes;
- In the brain: minimal gliosis;
- In the splen: inflamatory rocess;
- In the gastrointestinal tract: gastrites and extensive necrotising enteritis and colitis and typhlitis [18].

26. Treatment Strategies for Arenavirus Infections

Arenavirus infections, including severe hemorrhagic fevers such as Lassa fever (LASV) and Argentine Hemorrhagic Fever (AHF, caused by Junín virus, JUNV), remain challenging to manage [19]. Current treatment relies mainly on supportive care and the use of ribavirin while experimental therapeutics, particularly entry inhibitors and antibody-based therapies, offer emerging hope for broader antiviral coverage [20-23]. Supportive care remains the primary method for treating Lassa fever (LF) and is considered the mainstay of therapy for Arenavirus infections generally, as an effective antiviral agent has not yet been fully developed [24]. LF is a life-threatening infection, and although there are no definitive guidelines for its management, finding a definitive treatment would ideally reduce the mortality rate and shorten the disease course, thereby relieving pressure on isolation units in health facilities [19]. For Arenaviruses, supportive management includes meticulous attention to fluid balance and electrolyte management [25]. Ribavirin is a purine nucleoside analog (or guanosine analog) known for its broad-spectrum antiviral properties, exhibiting a virus-static activity against various RNA viruses, including Lassa fever virus [26].

The mechanism of action against LF is not yet fully identified, but studies suggest it employs multiple pathways [27,28]. These mechanisms include the inhibition of host inosine monophosphate dehydrogenase (IMPDH) and inhibition of viral replication via analog incorporation into viral RNA [29]. Intravenous (IV) ribavirin is the most effective treatment for acute LF, performing best when applied early in the course of the illness [21]. Beyond active treatment, the use of oral ribavirin is recommended for post-exposure prophylaxis because potential prophylactic efficacy was supported by a case where a physician took oral ribavirin after exposure to a confirmed LF patient and developed antibodies without symptoms [20]. Ribavirin is also believed to be effective against other arenaviruses [30]. Despite its accepted role, ribavirin is associated with significant side effects [31]. When administered intravenously, rigors (shaking chills) were reported in 27% of patients in one study, sometimes coupled with headache, lumbosacral pain, vomiting, or mild urticaria [20,32,33].

Besides ribavirin, other valuable therapeutic options include entry inhibitors, which can slow viral spread and help the host mount an effective immune response [34]. These agents work by stabilizing viral proteins and blocking the membrane fusion required for Arenavirus entry into cells. Expanding upon these antiviral strategies, antibody-based therapies have demonstrated considerable potential [35]. These therapies have conferred complete protection against Lassa virus infection in non-human primates and effectively inhibited cell entry of several New World arenaviruses by targeting viral glycoproteins or host entry receptors [22,36]. Arenavirus infections remain difficult to treat, with supportive care and ribavirin forming the current standard therapy. Emerging strategies, including entry inhibitors and antibody-based therapies, show promise by targeting viral entry and enhancing immune protection. Continued research is essential to develop effective and safe treatments for both Old and New World arenaviruses.

27. Conclusions

Despite knowledge of the epidemilogy and fisiopatology/ clinical manifestation of this hemorrhagics fevers the availability immunobiolgicals and drugs to prevention and treatment of this diseases is scarce. Will be of great value since the fatality rate of these diseases is equal too greater than 15%, with multiple organs/ system involvement.the investment in researches for prevention and treatment of hemorrhagic fevers will enable a reduction in morbidity and mortality rates caused by these Arenaviruses [37,38].

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