

The mitochondria that weren't

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In 2003, a region in northwestern Mexico faced a major dengue outbreak. At that time, the disease was formally classified as classic dengue and dengue hemorrhagic fever. Amid widespread alarm, virtually any fever was provisionally diagnosed as dengue; however, a serious dilemma arose when laboratory results began to systematically come back negative.

Forced to consider other diagnoses, physicians began to suspect bacteria such as *Leptospira* and *Salmonella*. However, there was a larger and even more aggressive group: *rickettsiae*, a group of intracellular bacteria capable of presenting diverse and complex clinical manifestations.

By that time, local physicians' experience with these types of infectious agents was limited. They only had a rudimentary *Proteus* OX19 test to guide diagnosis and chloramphenicol as their primary therapeutic option. Later, the use of *Rickettsia* antibody tests was established, but with a critical drawback: they only showed positive results up to 22 days after the onset of symptoms, while the window of opportunity for effective treatment had to be less than five days from the start of symptoms, given the high risk of complications and death. Adding to this was the shortage of doxycycline, the first-line drug, in pharmacies, a drug whose use is essential even in children under eight and pregnant women.

A first attempt to improve diagnostic timeliness was the implementation of immunohistochemistry, a process that required samples of the characteristic skin lesions of rickettsiosis, but for which the target antibody was lacking. At that time, Dr. David H. Walker, from the University of Texas and a leading authority on the subject, kindly agreed to provide the missing component. Unfortunately, bureaucratic obstacles in Mexico prevented the receipt of the material, thus losing a valuable scientific and medical opportunity.

In the absence of immediate diagnostic tests, patients' health depended entirely on the clinical expertise of physicians and the empirical initiation of doxycycline in any suspected case. Later, faced with the increase in cases of rickettsiosis, Dr. Gerardo Álvarez, from the University of Sonora, promoted

the use of the Polymerase Chain Reaction (PCR), which represented a giant leap toward timely diagnosis.

Despite these advances in diagnosis and treatment in the region, precarious housing conditions and constant exposure to the transmitting vector (the brown dog tick) keep the disease active.

The genus *Rickettsia* is extremely abundant and cosmopolitan. Among its most feared variants are epidemic typhus, caused by *Rickettsia prowazekii*, and murine or endemic typhus, caused by *Rickettsia typhi*. This bacterium has the peculiarity of being strictly intracellular, and a strong evolutionary hypothesis proposes that present-day mitochondria—organelles essential for the survival of eukaryotic cells—share a common ancestor with them or evolved from a similar lineage. It is paradoxical that an element so closely linked to the essence of cellular life can, in its pathogenic form, turn against the organism itself.

Unfortunately, there is currently no specific protection such as a vaccine. Rapid laboratory diagnosis still depends on PCR testing, since detectable antibody levels can persist for up to two months.

Because doxycycline is the treatment of choice—even in young children and pregnant women—and given the frequent lack of immediate laboratory confirmation, it is essential that healthcare personnel initiate antibiotic therapy within five days of the onset of clinical manifestations, guided solely by clinical and epidemiological criteria.

Thus, we can see how a disease that has accompanied humanity since time immemorial continues to be a burden, especially for populations living in the most precarious conditions.

You can consult:

1. Walker DH. *Rickettsiae*. In: Baron S, editor. *Medical Microbiology*. 4th ed. Galveston (TX): University of Texas Medical Branch at Galveston; 1996. Chapter 38. PMID: 21413251.
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