ISSN: 2993-6594

Research Article

Open Access Journal of Disease and Global Health

Chronic Cardiovascular and Autonomic Sequelae following Chikungunya Virus Infection: an 18-year Longitudinal Patient Narrative with Clinical Correlation

Bruce H Knox*

Independent Scholar, Auckland, New Zealand

***Corresponding Author**
Bruce H Knox, Independent Scholar, Auckland, New Zealand.

Submitted: 2026, Feb 23; **Accepted:** 2026, Apr 13; **Published:** 2026, Apr 27

Citation: Knox, B. H. (2026). Chronic Cardiovascular and Autonomic Sequelae following Chikungunya Virus Infection: an 18-year Longitudinal Patient Narrative with Clinical Correlation. *Ope Acce Jou Dis Glo Heal, 4*(1), 01-06.

Abstract

Chikungunya virus (CHIKV) infection is commonly described as an acute mosquito-borne febrile illness characterised by severe arthralgia, rash, and fatigue; however, a substantial minority of patients experience prolonged or chronic sequelae extending well beyond the acute infectious phase[1-4]. This paper presents an 18-year longitudinal patient narrative of cardiovascular and autonomic instability following CHIKV infection acquired during an outbreak in Indonesia in 2008. Within six months of apparent recovery, electrocardiographically confirmed atrial fibrillation emerged and later required catheter ablation. Over subsequent years, blood pressure lability, ventricular ectopy, orthostatic intolerance, and multisystem autonomic symptoms evolved despite structurally normal cardiac findings. Later specialist investigations supported dysautonomia and small-fibre neuropathic involvement. Existing literature recognises chronic inflammatory, neurological, and occasional cardiovascular sequelae after chikungunya, but long-term autonomic dysregulation remains comparatively under-described[2-7]. By integrating lived experience with longitudinal self-observation and medical literature, this paper argues that post-viral recovery may restore rhythm without restoring regulation. The case underscores the importance of long-term functional follow-up, multidisciplinary interpretation, and greater recognition of persistent autonomic manifestations within post-chikungunya care.

Keywords: Chikungunya Virus, Autonomic Dysfunction,Post-Viral Syndrome,Orthostatic Hypotension,Atrial Fibrillation,Small-Fibre Neuropathy

A Patient Perspective

1. Introduction

when the acute illness ends but the physiological story does notIn 2008, during a chikungunya outbreak in Indonesia, I developed what at the time appeared to be a classical acute CHIKV illness: high fever, rash, severe polyarthralgia, and profound fatigue. That pattern is consistent with the standard clinical descriptions provided by the CDC and WHO, both of which characterise chikungunya as an acute viral illness most often marked by fever and severe joint pain, sometimes accompanied by rash, fatigue, and prolonged post-acute symptoms[1,2].

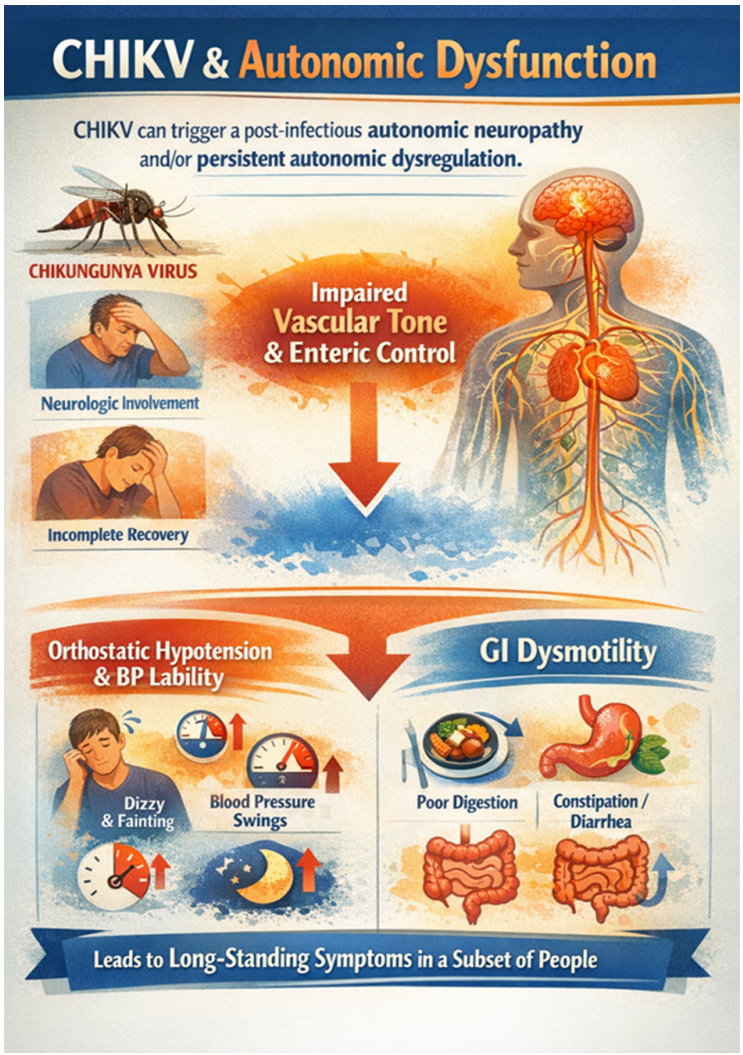
The acute illness settled within approximately two weeks. The fever passed. The rash faded. The joint pain eased. On the surface, recovery appeared complete. Yet the literature makes clear that chikungunya is not always confined to a brief acute phase. Chronic manifestations, especially musculoskeletal and neurological ones, are well recognised, and a proportion of patients continue to experience significant morbidity months or years after infection[2-5]. What I did not understand then was that the apparent end of infection might prove to be the beginning of a longer disturbance in physiological regulation.

1.1. Early Post-Viral Change: from Recovery to Atrial Fibrillation

Within six months of apparent recovery, I developed symptomatic atrial fibrillation. This was confirmed on electrocardiography and later treated with catheter ablation. From a cardiology perspective, the arrhythmia had a name, a traceable rhythm abnormality, and a procedure designed to correct it.

Although cardiovascular involvement in chikungunya is considered less common than articular disease, it has been reported in the literature. Systematic reviews describe myocarditis, arrhythmias, heart failure, and other cardiac manifestations as recognized, though relatively uncommon, complications of systemic CHIKV infection[6]. In my case, the emergence of atrial fibrillation so soon after infection formed the first major sign that the illness may have had consequences beyond the expected acute phase.

The ablation restored sinus rhythm. In one sense, that was a success. But with time it became clear that restoration of rhythm was not the same thing as restoration of stability.



1.2. Blood Pressure Variability and the Beginning of Autonomic Instability

After the arrhythmia was treated, I did not return to a pre-illness baseline. Instead, blood pressure variability began to define the next phase of the illness. At first it was subtle: light-headedness on standing, a sense of disequilibrium, reduced tolerance for exertion, and an increasing need to be careful with posture, heat, and hydration. Over time, these features became more pronounced.

The broader chikungunya literature supports the plausibility of persistent neurological and neuroimmune sequelae. Neurological disease has been documented in chikungunya, including both central and peripheral manifestations, and modern reviews increasingly place CHIKV within the wider category of infections capable of producing prolonged neurological dysfunction[5,7]. That does not prove causation in any one case, but it does provide medical context for symptoms that extend beyond joints and fever.

As I came to understand my condition over time, the distinction between electrical cardiac correction and autonomic regulation became crucial. The heart could beat regularly, yet the body could still fail to regulate blood pressure and postural adaptation reliably.

1.3. Orthostatic Intolerance becomes Clinically Significant

What had begun as intermittent light-headedness progressed into more definite orthostatic symptoms. Standing was no longer simple. There were episodes of visual dimming, cognitive slowing, internal instability, and a sense that the body was not adjusting properly to gravity. These are features compatible with orthostatic intolerance and autonomic dysfunction.

Consensus criteria define orthostatic hypotension as a sustained fall in systolic blood pressure of at least 20 mmHg or diastolic blood pressure of at least 10 mmHg within 3 minutes of standing or head-up tilt, with a higher systolic threshold sometimes used in the setting of supine hypertension[8]. Serial blood pressure observations over time, together with formal autonomic assessment, placed my experience within that clinical territory. What I was living subjectively was not merely “feeling faint”; it aligned with established autonomic concepts used in the clinical literature[8].

This mattered because brief clinical encounters do not always capture fluctuating regulatory problems. A structurally normal echocardiogram and reassuring imaging can coexist with substantial functional instability. In lived experience, this becomes the difference between “you look well” and “your body does not behave reliably.”

1.4. Ventricular Ectopy and Persistent Regulatory Disturbance

As the years went on, premature ventricular contractions emerged and became increasingly troublesome. These too were documented and eventually treated with a further ablation. Again, the electrical abnormality was visible, measurable, and procedurally actionable. The medical literature on chikungunya-associated cardiac involvement supports the possibility of arrhythmic and inflammatory sequelae, including myocardial involvement and rhythm abnormalities[6]. My own clinical course seemed to reflect that broader pattern: objective rhythm disorders arose and could be addressed, yet they did not explain the full syndrome. Even when rhythm improved, blood pressure lability and orthostatic symptoms persisted.

That was one of the most important lessons of the journey. The medical system is often strongest where pathology is visible and targetable. But regulation can remain impaired even when structure appears normal and rhythm is corrected.

1.5. Multisystem Extension and the Possibility of Small-Fibre Autonomic Involvement

With time, the dysfunction was no longer confined to cardiovascular symptoms. Heat intolerance, thermoregulatory instability, gastrointestinal dysmotility, bladder disturbance, and other multisystem features became part of the picture. Later investigations supported dysautonomia, and skin biopsy confirmed small-fibre neuropathic involvement.

This development also has medical plausibility. Small-fibre neuropathy affects thinly myelinated and unmyelinated fibres, including autonomic fibres, and can produce cardiovascular, gastrointestinal, sudomotor, sensory, and other dysautonomic symptoms[9,10]. Diagnostic reviews note that skin biopsy, autonomic testing, and specialised neurophysiological assessment are among the tools used to support the diagnosis when conventional large-fibre tests are unrevealing[9,10]. In my case, objective confirmation mattered greatly because many of the symptoms had long been real in experience but less visible in routine testing.

I cannot claim that CHIKV alone explains every later diagnosis with certainty. That would go beyond the evidence. But I can say that the chronology, the specialist findings, and the evolving literature on post-viral autonomic and neurological dysfunction together provide a coherent framework in which the lived story is medically credible[5-7,9,10].

1.6. Living with Regulation rather than Cure

The daily adjustments required by this illness have been quiet rather than dramatic. Hydration became structured. Heat required caution. Exertion required pacing. Standing became something to manage rather than something I could take for granted. The head of the bed was raised. Compression garments became useful. Energy had to be allocated rather than assumed.

These are standard non-pharmacological measures in autonomic practice, particularly where orthostatic intolerance and blood pressure instability are present[8]. What is easily summarised in clinical language as “lifestyle adaptation” is, in lived experience, a fundamental change in how one inhabits one’s own body.

That is why the phrase "stable but not restored" best captures the present situation. I am not describing a state of unremitting collapse. I am describing a long-term recalibration in which adaptation has produced livability without returning me to my pre-2008 physiology.

2. Clinical interpretation

From a clinical perspective, this case illustrates several important points. First, chikungunya is well established as a cause of acute febrile illness and chronic rheumatic sequelae, and the literature increasingly recognizes that neurological and occasional cardiac complications also occur[1-7]. Second, post-viral illness may evolve over years rather than resolving neatly within the time frame of the original infection[2-5]. Third, normal structural studies do not exclude persistent autonomic dysfunction[8-10]. Fourth, longitudinal observation, especially when supported by specialist testing, can reveal consistent physiological abnormalities that may be missed in snapshot assessments.

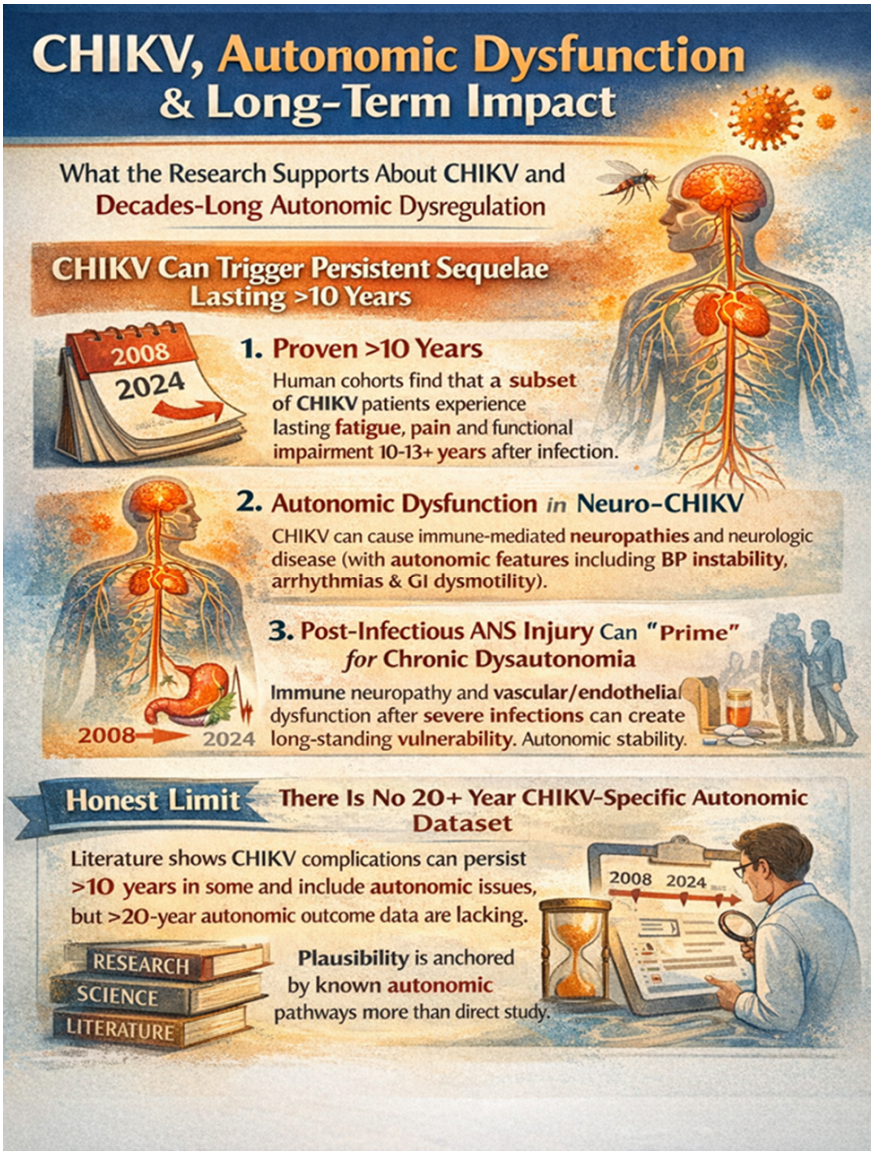
For clinicians, the case is a reminder that persistent post-viral symptoms deserve interpretation rather than dismissal. For patients, the presence of peer-reviewed literature matters because it helps translate isolated experience into medically intelligible experience.

3. Personal Reflection

There is grief in living with what was described as temporary. The outbreak has long passed. The virus no longer circulates in my bloodstream. Yet its consequences remain embedded in autonomic regulation.

The fever resolved in weeks.
The dysregulation has lasted eighteen years.

Recovery, I have learned, is not always restoration. Sometimes it is endurance under a diagnosis that the world assumes has already disappeared.



4. Conclusion

Eighteen years after chikungunya infection, I continue to live with the cardiovascular and autonomic legacy of that illness. The fever resolved in weeks, but the regulatory consequences did not. Atrial fibrillation emerged within months and was successfully ablated. Ventricular ectopy later required further intervention. Yet even when rhythm returned, blood pressure variability, orthostatic intolerance, and multisystem dysautonomia remained.

The core clinical claims above are supported by current CDC and WHO guidance on chikungunya, peer-reviewed reviews on chronic chikungunya and cardiovascular involvement, and standard autonomic and neuropathy references for orthostatic hypotension and small-fibre/autonomic dysfunction.

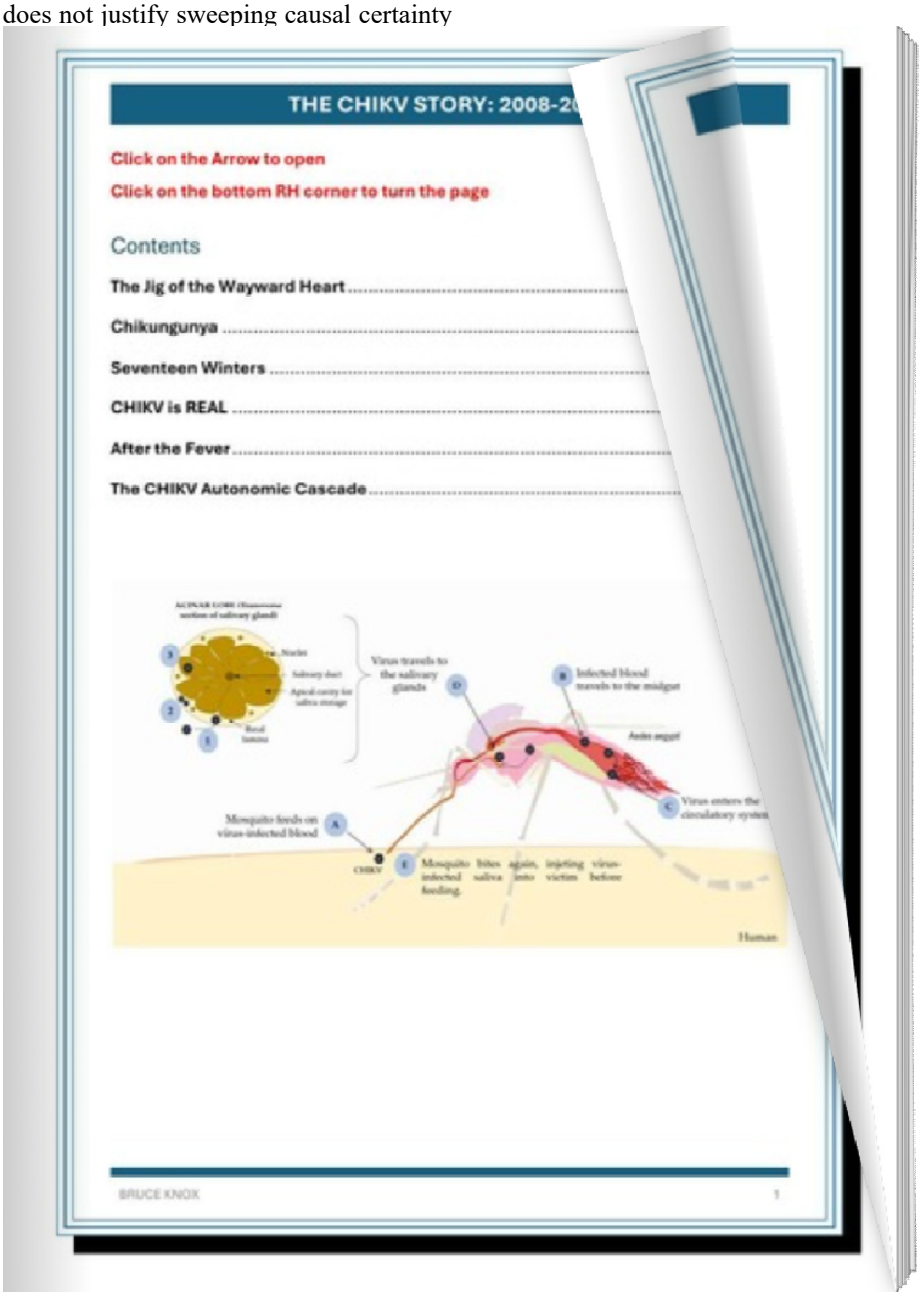
However, the literature does not justify sweeping causal certainty from a single case. But it does support the central credibility of this story: chikungunya can be followed by chronic sequelae; neurological and cardiovascular complications are documented; autonomic and small-fibre mechanisms are biologically plausible; and functional instability can persist even when conventional structural findings are reassuring[2-10].

The most accurate summary I can offer is this: the infection was acute, but its physiological consequences were not. My story is personal, but it is no longer medically isolated.

The Story and Understanding in Song

could the link for the flip book please be highlighted and made live so that when you click on it it actually works

https://brucehknox.aflip.in/2efa443802.html



Declarations

Competing Interests

None declared.

Funding

None.

Ethics

Not required (patient-author narrative).

Consent

Obtained.

Data Availability

Available upon request.

References

1. Centers for Disease Control and Prevention. Clinical signs and symptoms of chikungunya virus disease. Atlanta: CDC; 2024.
2. World Health Organization. (2025). Chikungunya fact sheet. *World Health Organization, Geneva, Switzerland*.
3. Suhrbier, A., Jaffar-Bandjee, M. C., & Gasque, P. (2012). Arthritogenic alphaviruses—an overview. *Nature Reviews Rheumatology, 8*(7), 420-429.
4. Rodriguez-Morales, A. J., Gil-Restrepo, A. F., Ramírez-Jaramillo, V., Montoya-Arias, C. P., Acevedo-Mendoza, W. F., Bedoya-Arias, J. E., ... & Lagos-Grisales, G. J. (2016). Post-chikungunya chronic inflammatory rheumatism: results from a retrospective follow-up study of 283 adult and child cases in La Virginia, Risaralda, Colombia. *F1000Research, 5*, 360.
5. Brito Ferreira, M. L., Pessoa Militao de Albuquerque, M. D. F., Antunes de Brito, C. A., de Oliveira Franca, R. F., Porto Moreira, A. J., de Morais Machado, M. I., ... & Solomon, T. (2020). Neurological disease in adults with Zika and chikungunya virus infection in Northeast Brazil: a prospective observational study. *Lancet Neurology, 19*(10), 826-839.
6. Alvarez, M. F., Bolívar-Mejía, A., Rodriguez-Morales, A. J., & Ramirez-Vallejo, E. (2017). Cardiovascular involvement and manifestations of systemic Chikungunya virus infection: A systematic review. *F1000Research, 6*, 390.
7. Carod-Artal, F. J. (2015). Neurological complications and long-term sequelae of chikungunya virus infection. *Rev Neurol61*(4), 161-7.
8. Freeman, R., Wieling, W., Axelrod, F. B., Benditt, D. G., Benarroch, E., Biaggioni, I., ... & Van Dijk, J. G. (2011). Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. *Autonomic Neuroscience, 161*(1-2), 46-48.
9. Oaklander, A. L., & Nolano, M. (2019). Scientific advances in and clinical approaches to small-fiber polyneuropathy: a review. *JAMA neurology, 76*(10), 1240-1251.
10. Terkelsen, A. J., Karlsson, P., Lauria, G., Freeman, R., Finnerup, N. B., & Jensen, T. S. (2017). The diagnostic challenge of small fibre neuropathy: clinical presentations, evaluations, and causes. *The lancet neurology, 16*(11), 934-944.