Systemic Brucellosis with Arrhythmogenic Cardiac Inflammatory Pseudotumor

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Patient: Female, 30-year-old
Final Diagnosis: Brucellosis • cardiac inflammatory pseudotumor
Symptoms: Palpitations • syncope
Medication: —
Clinical Procedure: Ambulatory ECG monitoring • biopsy • brain magnetic resonance venography without contrast • cardiac magnetic resonance imaging • echocardiography • electrocardiogram • electrophysiological study • implantable cardioverter defibrillator placement • laboratory checkup • PET-CT • subcutaneous cardioverter defibrillator placement
Specialty: Cardiology • Infectious Diseases

Objective: Rare coexistence of disease or pathology
Background: Cardiac inflammatory pseudotumors are rarely observed. Their etiology might include immunologic abnormalities, fibrogenetic disorders, specific reactions to infections or abnormalities related to trauma, necrosis, or neoplasm. Life-threatening ventricular tachycardia and cases of sudden death related to cardiac tumors have been reported. The present report describes and discusses diagnostic and therapeutic solutions for the treatment of nonsarcoïd multiorgan pseudotumors with cardiac involvement.

Case Report: A 38-year-old woman presented to the clinic with symptomatic ventricular tachycardia. As coronary artery disease, cardiomyopathy, and channelopathy were ruled out, and electrocardiograms were not typical of idiopathic arrhythmia, the patient underwent detailed diagnostics which included targeted endomyocardial biopsy, which revealed a cardiac inflammatory pseudotumor. Laborious testing (and eventually, antibiotic therapy) led to ex juvantibus diagnosis of multiorgan disseminated brucellosis with cardiac involvement. Treatment with ceftriaxone, doxycycline, and rifampicin resulted in a complete resolution of all lesions after 3 months, and sustained recovery was observed during a 5-year follow-up. As the risk of ventricular tachycardia could not be reliably predicted, the patient had a subcutaneous implantable cardioverter-defibrillator implanted.

Conclusions: A vast diagnostic armamentarium of modern medicine allowed us to diagnose an unsuspected and rare cardiac inflammatory pseudotumor. In the case of travelers, the possibility of regionally specific illnesses, especially infections, must be taken into consideration as possible causes of arrhythmias. Cardiac magnetic resonance imaging may be useful in patients with ‘idiopathic ventricular tachycardias’ to detect non-apparent myocardial lesions which may result from the underlying cause of the arrhythmia.

Keywords: Brucellosis • Case Reports • Tachycardia, Ventricular

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Background

The initial assessment of an episode of sustained ventricular tachycardia (VT) typically includes 12-lead ECG, transthoracic echocardiography, and functional testing and/or imaging for coronary artery abnormalities [1]. If structural or electrical heart disease is not excluded, a patient is deemed to have idiopathic VT, and further treatment, either pharmacological or invasive, must be planned, with consideration for the patient’s preference [2]. However, in rare cases, a thorough diagnostic process may lead to unsuspected findings. Infections should also be taken into consideration as possible causes of arrhythmias. Isolated cases of arrhythmia-triggering cardiac pseudotumors have been reported. Their etiology might include immunologic abnormalities, fibrogenetic disorders, specific reactions to infections, or abnormalities related to trauma, necrosis, or neoplasm. In the case of travelers, the possibility of contracting regionally specific illnesses, especially infections (including zoonoses), must be taken into consideration as possible causes of arrhythmias.

Case Report

A 38-year-old woman, with no previous medical history, presented in our hospital with palpitations which had lasted a few hours and were associated with 2 episodes of presyncope. Ventricular tachycardia of approximately 180 bpm (Figure 1) was diagnosed and sinus rhythm was restored with direct-current cardioversion 200J (Schiller DEFIGARD 5000).

Ventricular tachycardia recurred after several days and required another cardioversion. Initial evaluation included basic laboratory blood tests, the results of which were found to be normal, and transthoracic echocardiography that disclosed no abnormality. ECG revealed sinus bradycardia, lack of R-wave progression V1-4, and non-specific repolarization abnormalities with QTc falling within the normal range (QTc=380 ms) (Figure 2). The patient presented adequate exercise tolerance, heart rate, and blood pressure reactions, with no ECG changes during the treadmill test. Coronary angiography disclosed no abnormalities. The patient denied having any history of ventricular arrhythmia events, sudden cardiac deaths, or premature deaths.

Figure 1. 12-lead electrocardiogram during ventricular tachycardia. A clinical electrophysiologist noticed that the arrhythmia did not have the typical morphology for idiopathic left ventricular tachycardia (more precisely, left posterior fascicular ventricular tachycardia) mainly due to inconsistent R-wave amplitude differences between adjacent leads (ie, amplitudes in leads: V1 – low, V2 – high, V3 – low, V4 – residual, V5- high). Such a pattern might suggest scar-dependent ventricular tachycardia, but this requires confirmation.
in her relatives. As typical pathologies related to VT (coronary artery disease, cardiomyopathies, and channelopathies) were excluded, idiopathic VT was considered as a primary working diagnosis. However, an electrophysiologist opined that our patient’s ECGs did not match a typical picture of idiopathic VT and scar-related arrhythmia was suspected (detailed description in Figure 1 footnote). Therefore, the patient was referred for cMRI, which revealed tumorous contrast enhancement in the middle portion of interventricular septum (1×2.7 cm) and in the subendocardial region, in the basal segment of the right ventricle (Figure 3).

As the nature of the cardiac tumor could not be precisely established based on cMRI, a whole-body positron emission tomography scan (18F-FDG-PET/CT) was performed. The scan revealed several foci of increased metabolism in multiple organs with maximal standardized uptake value (SUVmax) varying from 5.3 to 14.5 (Figure 4).

Additionally, MRI of the brain disclosed a 12×13×10 mm lesion in the central part of the pons (Figure 5). The patient presented no symptoms typical of localized or disseminated infection or myocarditis. The markers for systemic inflammation and myocardial injury were within normal ranges. Disseminated sarcoidosis was considered as the most probable initial diagnosis. Transbronchial biopsy of pulmonary foci, however, appeared to be inconclusive as no abnormal cells were found on histopathology. For the abdominal foci, it was too risky to undergo biopsy, so targeted biopsy of the septal focus in the heart was performed. For this purpose, we used multiple-imaging guidance, which included intraoperative fluoroscopy, transesophageal echocardiography (TEE), and 3D electrophysiological mapping with the CARTO System (Biosense Webster Inc., Irvine, CA, USA) (Figure 6).
The echocardiographer led the biopcone to the middle of the septum and electroanatomical mapping showed areas of sub-tle reduction of voltage which were considered to be connect- ed with the tumor. The subsequent histopathology disclosed an inflammatory pseudotumor (IP) with no signs of sarcoidosis (Figure 7).

Therefore, bearing in mind that the patient had visited 4 different continents during the previous 6 months, stepwise di- agnostics (Table 1) of possible causes of multiorgan IP were undertaken. The following tests gave positive results: (1) IgG antibodies against Toxoplasma gondii, Aspergillus, and Pneumocystis carinii; (2) IgM antibodies against Brucella sp.; (3) antinuclear antibodies (1:320). As the blood count, serum protein electrophoresis, and C-reactive protein were all found to be normal, our consultant specialist in internal medicine found no indication for a bone marrow biopsy.

Multiorgan brucellosis or an autoimmunological disorder did not seem likely, but they were not impossible either. However, having considered a possibility of infection with Brucella mi-croorganisms, we meticulously reassessed the patient’s his- tory of possible risky habits. It was disclosed that during her travels to central and southern Asia, as well as to the Middle East and southeastern Europe, she did consume non-pasteur- ized and non-processed milk, home-made cheese, and oth- er local meals. Additionally, during the 3-month period pre- ceding hospitalization, she had experienced several episodes
of fever and joint pains which were interpreted as seasonal common infections. Therefore, after consultation with a multidisciplinary specialist team, we decided to start antimicrobial treatment against Brucella sp. with ceftriaxone 2 g intravenously, oral doxycycline 100 mg, twice daily, and oral rifampicin, 900 mg daily. Considering the potential for failure, steroid therapy was to be initiated if the antimicrobial treatment failed to resolve her symptoms. At 4 weeks of antibiotic therapy, 18F-FDG-PET/CT revealed a significant regression of the hypermetabolic foci (Figure 8), and cerebral MRI disclosed a lower saturation of the lesion in the pons, interpreted as a post-inflammatory scar. As we could not reliably estimate the risk of VT recurrence, the patient had a subcutaneous implantable cardioverter-defibrillator (S-ICD) inserted (Figure 9). The antibiotic treatment was continued for 12 weeks, when 18F-FDG-PET/CT revealed a full regression of all hypermetabolic foci. Approximately 2 and 4 years after the cessation of antibiotic therapy, the patient underwent additional general evaluations, which confirmed the regression of the disease. During S-ICD check-ups performed every 6 months, no arrhythmic events were detected (Table 2).

Discussion

We describe here the case of a patient in whom multiorgan brucellosis with cardiac involvement in the form of intraventricular tumor provoking symptomatic VT was considered the most probable diagnosis.

The possibility that the lesion was a neoplastic tumor was the keynote of our decisions; therefore, a whole-body PET/CT was
**Microbial and helminthic diagnostics**

**Bacteria:**
- Blood culture – negative (both aerobic and non-aerobic)
  - Francisella tularensis [IgM <1: 20 (N <1: 20); IgG <1: 40 (N <1: 20)]
  - Brucella sp. 1st [EIA: IgM 16.3U; IgG 6.0U; (N <8.5U)] 2nd, (after 12 weeks) [EIA: IgM 8.7U; IgG 28.3U (N <8.5U)]
  - Listeria monocytogenes [IFA: IgG <1: 10]
  - Bartonella henselae [IFA: IgA <1: 10; IgM <1: 100 (N <1: 100); IgG <1: 320 (N <1: 320)]
  - Coxiella burnetii [IHC: IgA, IgM, IgG <1: 60 (N <1: 60)]
- Leptospira

**Viruses:**
- HCV [anti-HCV – negative]
- HBV [HBs-antigen – negative]
- HIV [HIV combo – negative]
- EBV [ELISA: IgM – negative; IgG 3.24 RU/ml (N <16); IgA – negative]
- CMV [ELISA: IgM – negative; IgG <2 RU/ml (N <16); IgA – negative]
- Yersinia sp. [ELISA: IgA – negative; IgG 5 RU/ml (N <16)]
- Treponema pallidium [RPR: negative]

**Parasites:**
- Mycobacterium pneumoniae [QuantiFERON test – negative]
- Pneumocystis jiroveci [ELISA: IgM – negative; IgG 3.24 RU/ml (N <16); IgA – negative]
- Aspergillus [IgG, IgA, IgM 1: 320; (N <1: 80)]

**Miscellaneous assays:**
- Blood culture – negative
- Parasites in urine and blood (microscopy) [negative]
- WBC 8900/µl
- CRP [2.6 mg/l (N <5.0)]
- ANA [Sep 2015 1: 320; Dec 2015 1: 80 (N <1: 160)]
- cANCA [1: 20 (N <1: 10)]
- RF [<10 IU/ml (N <14)]
- IgG4 [65.7mg/dl (N: 5-100)]
- IgE [15.5 IU/ml (N <100)]
- Trypanosoma cruzi [IgA: IgG – negative]
- ACE [18U/l (N: 8-52)]
- Serum protein electrophoresis (g/l) [TP 73.5; Alb. 38.0; Globulins: α1 2.0; α2 9.0; β1 5.3; β2 4.2; γ 15.1]
- Table 1. List of laboratory tests focused on the underlying cause of the inflammatory pseudotumor.

HCV – hepatitis C virus; HBV – hepatitis B virus; HIV – human immunodeficiency virus; EBV – Epstein-Barr virus; CMV – cytomegalovirus; CRP – C-reactive protein; ANA – antinuclear antibodies; cANCA – antineutrophil cytoplasmic antibodies targeting proteinase 3; pANCA – antineutrophil cytoplasmic antibodies targeting myeloperoxidase; RF – rheumatic factor; EIA – enzyme immunoassay; EIT – enzyme immunoassay test; MONA – multiple of normal activity; IHA – indirect hemagglutination assay; IFA – indirect immunofluorescence assay; IIA – indirect immunoassay; JHC – immunohistochemical assay; ELISA – enzyme-linked immunosorbent assay; BBU – Biomedica Borrelia units; TP – total proteins; Alb. – albumins; ACE – angiotensin converting enzyme.
SUVmax of the foci fell within the range of overlap between malignant neoplasms and inflammatory lesions [3]. As the biopsy of foci in peripheral organs was either unsuccessful or posed too high a risk to be performed, targeted transvenous biopsy of the interventricular septum was performed. Although a combination of echocardiography and fluoroscopy is usually used to guide biopsies of heart tumors [4], we could not apply this technique because our patient’s tumor was not clearly visible in echocardiography. Therefore, we performed additional 3D electroanatomical reconstruction of the intraventricular septum, which is known to target myocardial scars in cardiomyopathies and cardiac masses [5]. Our approach was unique in such a setting because in the majority of the reported cases of inflammatory cardiac pseudotumors, histopathology was performed either as post-mortem autopsy or after cardiac surgeries that often entailed severe or lethal complications [6].

The term “inflammatory pseudotumors” describes various inflammatory masses that are known by different names (most commonly as inflammatory myofibroblastic tumors, but also as plasma cell granuloma, histiocytoma, and others). IP etiology might include immunologic abnormalities, fibrogenetic disorders, specific reactions to an infectious agent, or abnormalities related to trauma, necrosis, or neoplasm. The clinical course of IPs has not been precisely determined and their classification is still uncertain. To date, neither diagnostic schemes nor adequate treatments have been agreed on [6-8]. IP secondary to infections, including brucellosis, have only rarely been described.

As the biopsy revealed neither signs of neoplasm nor of sarcoidosis, a differential diagnosis became even more challenging because the underlying causes of non-sarcoid inflammatory pseudotumors include a variety of pathologies that are not commonly diagnosed by cardiologists. Therefore, after multidisciplinary consultations, diagnostic tests for specific infections (bacterial, viral, fungal, protozoal, and helminthic) as well as rheumatic diseases were performed (Table 1). The obtained results were not straightforward, but did narrow down the probable causes of the underlying pathologies to 2 possibilities: brucellosis or an autoimmune disorder. Anti-inflammatory therapy with steroids could potentially exacerbate the course of infection if the cause were indeed brucellosis, and it was...
Table 2. Timeline of the present case report.

| Date          | Description                                                                 |
|---------------|-----------------------------------------------------------------------------|
| Sep 4-18, 2015| **First Hospitalization – General Cardiology Unit**                          |
|               | • Symptoms: Palpitations and presyncope                                       |
|               | • ECG (on admission) – Ventricular tachycardia 160-180 bpm                    |
|               | • Direct current cardioversion                                                |
|               | • ECG (resting) – Sinus bradycardia 47 bpm. Left Axis. LAH. PR=0.19 s. QRS=0.09 s. Lack of R-wave progression (V1-V4). Negative T-wave (aVF). ST-segment depression of 1 mm (V5-V6). QT=0.4 s |
|               | • General blood laboratory tests (including troponins, CK-MB, CRP, procalcitonin) – normal |
|               | • TTE – no abnormality; LV-EF=65%                                            |
|               | • ETT (13.5 METs) – appropriate heart rate and blood pressure response; no induced ST-changes |
|               | • Coronary angiography – normal anatomy; no coronary artery stenosis         |
|               | • Cardiac MRI – foci of myocardial late enhancement:                          |
|               | - interventricular septum – 1.0×2.7 cm                                       |
|               | - inferior wall LV – 1.6×2.9 cm                                              |
| Sep 18- Dec 24, 2015 | **Second Hospitalization – Electrocardiology Unit**                     |
|               | • Abdominal USG (Sep 2015) – normal                                           |
|               | • 18F-FDG PET/CT – foci of abnormal hypermetabolism in lungs, liver, spleen and intraventricular septum of the heart |
|               | • Bronchoalveolar lavage (Oct 2015) – histopathology – inconclusive          |
|               | • Targeted EMB-septal cardiac focus (Oct 2015) – histopathology – inflammatory pseudotumor |
|               | • 24hHM-ECG (Oct 2015) – Sinus rhythm av. 55 bpm (40-93). VPBs – 1 bp, 24 h; SVPBs – 160 bp, 24 h |
|               | • Brain MRI (Nov 2015) – lesion (12×13×11 mm) in pons cerebri                |
|               | • 24hHM-ECG (Nov 2015) – Sinus rhythm av. 55 bpm (40-93). VPBs – 463 bp 24 h, nsVT; SVPBs – 70 bp 24 h |
|               | • Laboratory tests (Oct-Nov 2020) (blood, stool, and urine) focused on underlying causes of inflammatory pseudotumor, positive: |
|               | - Anti-Brucella sp. antibodies IgM(+), IgG(-)                                |
|               | - ANA (1: 320), ANCA (1: 30)                                                 |
|               | - Antibodies IgM(-), IgG(+) against: Toxoplasma gondii, Aspergillus sp., Pneumocystis carinii |
|               | • Antibiotic treatment (Nov 2015):                                            |
|               | - Ceftriaxone 2.0 g(iv) once                                                  |
|               | - Rifampicin 300 mg (po) 3x/day                                               |
|               | - Doxycycline 100 mg 2x/day                                                   |
|               | • 18F-FDG PET/CT (Dec 2015; 4 weeks on antibiotics) regression of lesions found in previous PET/CT (Oct 2015): |
|               | - Complete resolution of foci in the liver, spleen, and lymph nodes          |
|               | - Residual foci in lungs and in intraventricular septum of the heart         |
|               | • Brain MRI (Dec 2015; 4 weeks on antibiotics) – regression of lesion in pons cerebri; less prominent after contrast injection |
|               | • Neurologist consultation – no functional abnormality observed             |
|               | • Neurosurgeon consultation – no urgent indication for intervention; wait and watch strategy |
|               | • S-ICD implantation (Dec 2015)                                              |
|               | • Discharge from hospital on antibiotics (Dec 2015)                          |
| Feb-Mar, 2016  | • 18F-FDG PET/CT – full regression in heart, lungs, and abdomen; metabolic activity in small axillary lymph nodes and in anterior mediastinum (non-specific) |
|               | • Brain MRI – lesion unchanged                                               |
|               | • End of antibiotic therapy (after 12 weeks)                                 |
|               | • ECG (resting) – Sinus rhythm 59 bpm. Left axis. LAH. PR=0.19s. QRS=0.09s. Lack of R-wave progression (V1-V4). Negative T-wave aVF. ST-segment depression (1 mm) V5-V6. QT=0.4 s |
| Apr 19, 2016   | • Electromyography of blink reflex – normal                                  |
| May 30, 2016   | • Neurology consultation – no abnormality observed                           |
| Jul-Sep, 2016  | • Asymptomatic; TTE – normal (LV-EF=55%)                                     |
|               | • 48hHM-ECG – Sinus rhythm 71 (40-136) bpm. VPBs – 8327 beats. SVPBs – 33 beats |
|               | • Brain MRI – lesion unchanged                                               |
|               | • Propafenone 150 mg orally 2x/day                                           |
for this reason that we decided to initiate antimicrobial treatment first. As this resulted in full resolution of the inflammatory lesions, the final diagnosis of the underlying cause of the IP was Brucella infection.

To the best of our knowledge, only one case of an inflammatory tumor in the interventricular septum of the heart has been published, and its underlying cause was not disclosed [9]. Cardiac involvement during Brucella infection may have different clinical manifestations: it can be limited to inflammation of the endocardium (usually on an aortic valve), myocardium, or pericardium, but pancarditis has also been reported. On the other hand, infection with the Brucella microorganism can result in tumor-like lesions in different organs (eg, the liver or brain). It is thought that these lesions begin to develop in the acute phase as purulent-like foci that in time transform into pseudotumoral structures that develop into caseous necrotic tumors known as brucellomas [10]. Such unusual inflammatory structures are formed as a result of the suppressive effect Brucella microorganisms produce on the host immune system. Brucellosis presenting as cardiac IP has not been reported so far; thus, we found it baffling to accept such a clinical suspicion. Nevertheless, after stepwise and laborious work-up engaging several specialists of distinct medical disciplines, we deemed such a diagnosis as probable, which resulted in antibiotic treatment initiation. Possible alternatives for therapy of inflammatory pseudotumors include nonsteroidal anti-inflammatory drugs or steroids and chemotherapeutic drugs, both of which are usually applied as adjuvant therapy to surgical resection [11,12]. Of note, although some authors recommend surgical resection as the optimal therapy for inflammatory pseudotumors [6], in our case, this option was considered as the last-line treatment due to multiorgan lesions in unfavorable locations.

Life-threatening VTs and cardiac tumor-related sudden death cases have been reported [13]. The future risk of VT recurrence could not be reliably estimated in our patient due to the extremely rare etiology and uncertain dynamics of her inflammatory cardiac lesions. As no stratification method (ie, invasive electrophysiologic study) has been demonstrated in such a clinical scenario, we decided to implant a cardioverter-defibrillator. As the patient did not require permanent pacing and, bearing in mind that over the course of a 3-month in-hospital observation she had no VT recurrence, a subcutaneous device was chosen instead of a transvenous system. Disadvantages of S-ICD systems include the inability to provide antitachycardia pacing in case of monomorphic VT; however, by not implanting the defibrillation lead into the right ventricle, we avoided its possible harmful interaction with the injured myocardium [14]. Other possible options could be a wearable cardioverter-defibrillator but taking into account observations of recurrences of brucellosis, we decided that permanent protection with the implantable device might be a better option.

Table 2 continued. Timeline of the present case report.

| Date       | Examination/Procedure                                      | Results/Comment                                                                 |
|------------|------------------------------------------------------------|---------------------------------------------------------------------------------|
| Nov 12, 2016 | Electromyography of blink reflex – normal                 |                                                                                 |
|            | Neurology consultation – no abnormality disclosed          |                                                                                 |
| Jan-Jul, 2017 | Symptoms: Paroxysmal palpitations                         |                                                                                 |
|            | 48hHM-ECG – Sinus rhythm 70 (39-179) bpm. VPBs – 1834 beats. SVPBs – 11 beats |                                                                                 |
|            | TTE – normal (EF=60%)                                      |                                                                                 |
| Jun-Jul, 2017 | Symptoms: Paroxysmal palpitations                         |                                                                                 |
|            | 48hHM-ECG – Sinus rhythm 63 (39-133) bpm. VPBs – 1143 beats. SVPBs – 17 beats |                                                                                 |
|            | TTE – normal (LV-EF=60%)                                   |                                                                                 |
|            | Electrocardiography – no lesion found, full regression     |                                                                                 |
| Jan-Feb, 2018 | Symptoms: Paroxysmal palpitations                         |                                                                                 |
|            | 48hHM-ECG – Sinus rhythm 63 (39-133) bpm. VPBs – 1143 beats. SVPBs – 17 beats |                                                                                 |
|            | TTE – normal (LV-EF=60%)                                   |                                                                                 |
|            | Electrocardiography – no lesion found, full regression     |                                                                                 |
| Oct 18, 2019 | Symptoms: Paroxysmal palpitations                         |                                                                                 |
|            | 48hHM-ECG – Sinus rhythm 64 (41-111) bpm. VPBs – 142 beats. SVPBs – 9 beats |                                                                                 |
| Feb, 2020 | Symptoms: Paroxysmal palpitations                         |                                                                                 |
|            | 18F-FDG PET/CT – no lesion found, full regression          |                                                                                 |
|            | TTE – normal (LV-EF=60%)                                   |                                                                                 |

ECG – electrocardiogram; LAH – left anterior hemiblock; CRP – C-reactive protein; PCT – procalcitonin; CK – creatine kinase; TTE – transthoracic echocardiography; LV-EF – left ventricular ejection fraction; ETT – exertional treadmill test; MRI – magnetic resonance imaging; LV – left ventricle; USG – ultrasonography; EMB – endomyocardial biopsy; 18F-FDG PET/CT – 18F-fluorodeoxyglucose uptake on positron emission tomography scan; 24hHM-ECG – 24-hour ECG Holter Monitoring; 48hHM-ECG – 48-hour ECG Holter Monitoring; VPBs – Ventricular Premature Beats; SVPBs – Supraventricular Premature Beats; ANA – antinuclear antibodies; ANCA – antineutrophil cytoplasmic antibodies; S-ICD – subcutaneous cardioverter-defibrillator.
Conclusions

A vast diagnostic armamentarium of modern medicine allowed us to diagnose an unsuspected and rare cardiac inflammatory pseudotumor. In the case of travelers, the possibility of regionally specific illnesses, especially infections, must be taken into consideration as possible causes of arrhythmias. Cardiac magnetic resonance imaging may be useful in patients with ‘idiopathic ventricular tachycardias’ to detect non-apparent myocardial lesions, which may prove to be an underlying cause of the arrhythmia. Even though diagnosis *ex juvantibus* exists in medical practices, it is considered an inelegant route to the identification of the underlying cause of a disease. Nevertheless, even in modern medicine, there are clinical situations when such an approach must be considered [15]. Our therapeutic success allowed us to establish a diagnosis of disseminated brucellosis with cardiac involvement with a high degree of certainty.

Declaration of Figures’ Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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