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REVIEW ARTICLE

Clinical features of Poncet's disease. From the description of 198 cases found in the literature

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Abstract Poncet's disease (PD) is an entity described as a reactive arthritis due to tuberculous infection elsewhere from the joints. PD existence has been questioned; however, more cases have been reported over the years. Due to its rare nature, little is known about the clinical picture of this disease and no prospective studies had been made to address this issue. We performed a systematic review of the written literature on PD in different databases using the key words "Poncet's disease," "tuberculous rheumatism," and "tuberculous reactive arthritis." Out of 78 articles, 198 patients were included in the analysis, plus our patient. Several characteristic patterns were found. Also, a review of the pathogenesis and some hypotheses are made. PD is a well-defined entity, which should be taken as a reactive arthritis for future studies given the increase in TB incidence and prevalence around the world, especially in high-burden countries.

Keywords Clinical patterns · Diagnosis · Pathogenesis · Poncet's disease · Tuberculous rheumatism

Introduction

Throughout the years, the incidence of tuberculosis (TB) has increased exponentially. According to the World Health Organization, in 2007, the incidence of new tuberculosis cases was 9.27 million (139 new cases per 100,000 populations) [1].

This worldwide resurgence of TB raised interest on the disease including extrapulmonary TB. Approximately 10–11 % of extrapulmonary TB involves joints and bones (about 1–3 % of all cases of TB) [2]. Almost half of these cases are spinal TB, followed by TB arthritis, TB osteomyelitis, TB dactylitis, and reactive arthritis [2]. The latter reactive arthritis can be divided in arthritis associated to adjuvant mycobacterial treatment for bladder carcinoma and Poncet's disease (PD).

Antonin Poncet first introduced PD in 1897 when he described a polyarthritis in an acute stage of TB, which resolved without joint damage [3]. His broad concept was based on the association of polyarthritis with active or inactive visceral TB or a family history of TB or the presence of true TB arthritis in a patient before, coincident with, or following a polyarthritis of any type [4]. This definition lacked diagnostic precision and was rapidly rejected by fellow colleges [5]. Continuous reports on patients with similar characteristics led authors to improve the definition and, in 1978, Bloxham and Addy defined PD as a para-infective arthritis [6]. To the authors, PD was similar to a reactive arthritis, an aseptic arthritis triggered by an infection outside the joint, in this case TB, but with complete resolution of the arthritis and no joint damage or chronicity once the infection was treated.

Nevertheless, PD existence continued to be questioned by some authors. Summers and Jayson found no evidence of PD in 50 cases of TB. Later on, Holoshitz et al. proposed that PD should be considered only in the presence of synovial biopsy, after finding a patient with clinical features of PD and true TB joint infection, which was confirmed by biopsy [7, 8]. To this date, the disease is not completely accepted.

We took interest in the disease after treating a patient with, what we believe is PD. After reviewing the literature, we came upon the contradictory statements mentioned before and found no complete definition, classification, and characteristics of the disease. We decided to gather an important amount of cases, by reviewing different databases

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around the world, in order to characterize, define, explain, and implement PD as a real well-defined disease.

Case report

A case of PD was identified together with the Rheumatology and Infectious Diseases departments at the Clínica Universitaria Colombia in Bogotá. A 36-year-old male without relevant medical history except as smoker of 10 cigarettes per day since youth, was admitted with a 34-day history of chills, fever, bilateral flank pain, cervical pain, and widespread myalgias. Physical examination revealed tachycardia, temperature of 38 °C, cervical spasm, and several ender points. Initial laboratory testing showed increased C-reactive protein (40 mg/dL), increased erythrocyte sedimentation rate (80 mm/h), and leucocytes of 11.5×10^9 E/l.

The patient was hospitalized, and additional laboratory testing was performed, which resulted negative for mononucleosis, toxoplasmosis, cytomegalovirus, HIV, and syphilis. Later on, the patient complained of pain and swelling of elbows, right knee, and ankles. A synovial fluid analysis was made of the right knee revealing no crystals. Standard cultures and cultures for TB of synovial fluid, blood, and sputum were negative. X-rays of the knee, elbows, and ankles showed no abnormalities. Autoimmune laboratory tests including anti-cyclic citrullinated peptide and antinuclear antibodies were negative. A chest CT scan was performed showing a dense nodule of 12 mm of diameter in the lower lobe of the left lung. Fine needle aspiration cytology resulted in an insufficient specimen and an open lung biopsy with partial pleurectomy was carried out. Results from the biopsy revealed caseating necrosis. A diagnosis of pulmonary TB and PD was made and isoniazid, rifampicin, pyrazinamide, and ethambutol were started with complete resolution of all symptoms including joint pain and swelling within the following 7 days.

Literature review

The electronic databases (MEDLINE, PUBMED, PUBMED CENTRAL, SCIELO, BIREME, LILACS, WPRIM, AIM, IMSEAR, and IMEMR) were systematically searched up to March 2013 for all case reports and studies concerning PD. For the search strategy, the following mesh terms and text words were used: "Poncet's disease," "tuberculous rheumatism," and "tuberculous reactive arthritis." No other limits were employed. Articles in all languages were included. The cases that met the following requirements were included: patients with active or latent TB infection in any organ, presence of arthritis (swelling or pain) in any joint, resolution of joint symptoms with anti-TB therapy, and exclusion of other causes of arthritis (autoimmune, infectious, degenerative, deposit, trauma). The following information was abstracted for the analysis: characteristics of the clinical presentation of the disease (number of type of arthritis, site of arthritis, time of resolution of arthritis), demographics (sex, age, origin), site of TB infection, adenopathy and site of adenopathy, and presence of other tuberculous manifestations. After an extensive and complete search of the literature, 78 reports were detected [4, 6, 9–83], from which 198 patients were extracted and 199 patients were used for the analysis (including our own patient).

Clinical characteristics

Of 199 patients, 42.2 % (*n*: 84) were male and 39.2 % (*n*: 78) were female. The remaining patients (18.6 %; *n*: 37) had no gender specification. Of these, 25 were reported by Duggal and Khosla [9], 8 by Garg et al. [79], 3 by Greenwood [10], and one by Kowalzki and Seitz [11]. The mean age was 33.7 years (SD±12.5 years) with a range of 2–78 years.

Of the patients, 35 % were from India (*n*: 70), 13.1 % (*n*: 26) were from Brazil, 10.1 % (*n*: 20) were from Mexico, 6.0 % (*n*: 12) were from England, and 3.0 % (*n*: 6) were from Nigeria. The remaining patients were from: Russia, Spain, USA, and Pakistan with five cases each (2.5 %), Iran with four cases (2.0 %), Japan, Korea, and France with three cases each (1.5 %), Syria, Zambia, China, and Turkey with two cases each (1.0 %), and Ethiopia, Italy, Peru, Hong Kong, Germany, Tanzania, South Africa, Suriname, Switzerland, Sri Lanka, Serbia, Zaire, Chile, Colombia, Finland, and Argentine with one case each (0.5 %). Only two patients (1.0 %) did not have information on origin which corresponded to the paper by Wilkinson and Roy [12].

A site of TB infection was specified in 96.5 % (*n*: 192), being 56.8 % (*n*: 113) extrapulmonary TB and 42 % of patients (*n*: 84) had lymph node involvement. Only in 8.5 % of patients (*n*: 17) had the presence of erythema nodosum reported by the authors.

The most common affected joints were the ankles (*n*: 126, 63.3 %) followed by knees (*n*: 117, 58.8 %), wrists (*n*: 58, 29.1 %), and elbows (*n*: 46, 23.1 %). Forty percent (*n*: 81) of the patients presented with oligoarthritis, 27.6 % (*n*: 55) with polyarthritis, and 24.6 % (*n*: 49) with monoarthritis. Joint arthritis resolved in an average of 51.6 days (SD±37.5 days) after antituberculous therapy. The clinical features of the study population are summarized in Table 1.

PD is characterized by oligoarthritis, mainly affecting large joints like ankles, knees, wrists, and elbows, with no axial involvement. The arthritis tends to resolve weeks after antituberculous therapy with no tendency to chronicity. Also, no extra-articular manifestations were found and only a few patients presented erythema nodosum. Although

Table 1 Demographics of the study population

Characteristic	Number	Percent
Gender		
•Male	84	42.2
•Female	78	39.2
•Not reported	37	18.6
Origin		
•Asia	104	52.3
•South America	31	15.6
•Europe	25	12.6
•Central America	20	10.1
•Africa	12	6.0
•North America	5	2.5
•Not reported	2	1.0
TB infection		
•Lung	86	43.2
•Lymph nodes	69	34.7
•Urine	20	10.1
•Bone	9	4.5
•Skin	4	2.0
•Joint	3	1.5
•Finger	2	1.0
•Genital	2	1.0
•Gut	2	1.0
•Kidney	2	1.0
•Eye	1	0.5
•Meninges	1	0.5
•Brain	1	0.5
•Pancreas	1	0.5
•Pleural	1	0.5
•Disseminated	1	0.5
•Not reported	7	3.5
Adenopathy		
•Hilar	37	18.6
•Mediastinal	28	14.1
•Cervical	18	9.0
•Axillar	13	6.5
•Supraclavicular	9	4.5
•Inguinal	2	1.0
•Abdominal	2	1.0
•Auricular	1	0.5
•Submandibular	1	0.5
•Mesenteric	1	0.5
•Not reported	71	35.7
Affected joint		
•Ankles	126	63.3
•Knees	117	58.8
•Wrists	58	29.1
•Elbows	46	23.1
•IP	42	21.1
•MCP	34	17.1
•Shoulders	28	14.1
•MTP	25	12.6
•Not reported	18	9.0
Type of arthritis		
•Oligoarthritis	81	40.7
•Polyarthritis	55	27.6
•Monoarthritis	49	24.6
•Not reported	14	7.0

References: [4, 6, 9–83]

IP interphalangeal, *MCP* metacarpophalangeal, *MTP* metatarsophalangeal

extrapulmonary TB was the most frequent site of infection, our study showed that the lung is the most affected organ associated to PD, and lymph nodes are frequently affected.

Our findings add even more strength to the diagnostic criteria proposed by Novaes et al. (Table 2) [70]. As expected, Asian countries reported most of the cases of PD; however, one should expect more cases from Africa. This could be explained by the lack of research publication from African countries.

Pathogenesis

Although little is known about PD, some hypotheses have risen throughout the years. It is well known that tuberculosis is arthritogenic and several findings have proved this statement. Holoshitz et al. demonstrated antigenic similarity between a fraction of *Mycobacterium tuberculosis* and human cartilage [8]. They also induced arthritis in rats, using a T lymphocyte clone that recognized both *M. tuberculosis* antigens and antigens in human synovial fluid cartilage [84]. Bhattacharya et al. demonstrated circulating immune complexes that could be trapped in the synovium of patients with active tuberculosis [85]. Southwood et al. found in a patient with PD exaggerated reaction of synovial fluid lymphocytes to purified protein derivative compared to peripheral blood lymphocytes [86]. The use of immunotherapy with bacillus Calmette–Guérin as an adjuvant for the treatment of bladder carcinoma may be followed by mono or polyarthritis, which has proven to enhance Th1 cell-mediated responses and suppression of Th2-cell activity [87]. Finally, the animal model of adjuvant arthritis in which injection of heat-killed desiccated *M. tuberculosis* (complete Freund′s adjuvant) results in chronic arthritis resembling rheumatoid arthritis, due to a T cell-mediated cross-reactivity between mycobacterial antigens and human cartilage [88].

Molecular mimicry is thought to be the explanation to all these events. Some epitopes of microbe heat-shock proteins

Table 2 Modified diagnostic criteria of PD

•Evidence of active extra-articular tuberculosis
•Rheumatic manifestation in more than one joint (oligoarthritis, preferably knees and ankles)
•Lack of axial, vertebral column, and sacroiliac impairment
•Unspecific laboratory findings
•Complete remission after antituberculous chemotherapy
•No chronicity with no articular sequelae
•Exclusion of other rheumatologic diseases

(HSP) have shown a high degree of sequence homology with certain host's normal proteins or with the host's HSP, which are produced at the site of inflammation [89]. In TB infection, a cross-reactivity between the epitope of mycobacterial 65 kDa HSP and the human cartilage proteoglycans is the main cause for molecular mimicry [90].

It is believed that all these findings are the key to the pathogenesis of PD, if they are present in a genetically predisposed patient. Those who are HLA-DR3 and/or HLA-DR4, show T cell hyper responsiveness to mycobacterial antigens [89]. Also, several authors have demonstrated the presence of these HLA alleles in patients with PD [26, 30, 72, 77]. In a recent study, however, HLA typing was performed to 16 Mexican mestizo patients with PD, finding a significantly increased frequency of HLA-B27 and DQB1*0301 alleles when compared with healthy controls [77]. In fact, our study shows that of the 15 cases of PD found in the literature that were tested for HLA-B27, 4 (26.6 %) were positive.

The sum of the hypothesis explained before and others not yet discovered, could be the basis of the pathogenesis of PD. Therefore, in a genetically predisposed patient with a TB infection, bacterial antigens migrate to the joints where cross-reactivity between mycobacterial antigens and host cartilage occurs, inducing arthritis conducted by T cells. Therefore, by definition, PD should be considered a reactive arthritis (ReA).

Reactive arthritis and Poncet's disease

ReA was originally defined as joint inflammation triggered by an extra-articular bacterial infection, and it was distinguished from post-infectious arthritis by absence of bacterial components in the joint tissue, hence sterile arthritis [91]. Classically, ReA has been related to arthritides triggered by *Chlamydia*, *Yersinia*, *Salmonella*, *Shigella*, *Campylobacter*, and *Clostridium*, which are HLA-B27 related and display similar symptoms to spondyloarthropathies, like axial involvement, tendency to chronicity, and extra-articular manifestations (conjunctivitis and uveitis) [92]. However, other infections have shown to trigger ReA without a clear association to HLA-B27. These include *Borrelia*, *Brucella*, *Haemohilus*, *Hafnia*, *Leptospira*, *Neisseria*, *Staphylococcus*, *Streptococcus*, *Ureaplasma*, *Vibrio*, and *Mycobacterium* [93]. Due to the lack of association to HLA-B27, and maybe because of that, the clinical picture of the "non-classical" ReA has less tendency to chronicity, no axial involvement, and less extra-articular manifestations [93]. Because of this issue, controversy has arisen on the classification of ReA, which has led to several categorizations [94, 95]. Toivanen and Toivanen state that depending on the triggering agent, the arthritides fulfilling the original definition of ReA could be considered to occur in two forms, one, HLA-B27 associated, and another, HLA-B27 non-associated [93]. According to our analysis, PD shares

Table 3 Characteristics of HLA-B27 associated ReA, non-associated ReA, septic tuberculous arthritis and PD

Characteristics	HLA-B27 associated ReA	HLA-B27 non-associated ReA	Septic tuberculous arthritis	Poncet's disease
Triggers	*Chlamydia*, *Campylobacter*, *Clostridium*, *Salmonella*, *Shigella*, *Yersinia*	*Vibrio*, *Borrelia*, *Brucella*, *Haemophilus*, *Hafnia*, *Leptospira*, *Neisseria*, *Staphylococcus*, *Streptococcus*	*Mycobacteria*	*Mycobacteria*
Cultivable microbes present in joint	No	No	Yes	No
Time of onset	Weeks	Weeks	Weeks to months	Weeks
Joints affected	Knee, ankles	Knee	Hip and knee	Knee, ankles
Number of joints affected	Oligoarthritis	Polyarthritis	Mostly monoarthritis	Oligoarthritis
HLA typing	HLA-B27	None	None	HLA-B27, HLA-DQB1
Extra-articular manifestations	Yes	Not frequent	No	No
Tendency to chronicity	Yes	No	Yes	No
Axial involvement	Yes	No	No	No
Improvement with antibiotics	No	Yes	Yes	Yes

both characteristics of HLA-B27 associated and HLA-B27 non-associated ReA, as shown in Table 3. Early hospital-based studies have reported a frequency between 60 and 90 % of HLA-B27 in ReA patients [96]. However, in more recent studies based on outbreaks and surveys at population level, only a slight or no increased frequency of HLA-B27 has been reported [97, 98]. That is why some authors question the utility of HLA-B27 testing in ReA [98].

Once more, controversy arises on this disease. On one hand, the clinical characteristics of PD are more similar to that of an HLA-B27 non-associated ReA, no axial involvement, no tendency to chronicity, and no extra-articular manifestations and improvement with antibiotics. On the other hand, there is an association between PD and HLA-B27 as demonstrated on the study by Lugo-Zamudio et al. [77].

Conclusion

In summary, PD is a ReA, caused by molecular mimicry between TB antigens and host cartilage in a genetically predisposed patient, which is characterized by an oligoarthritis predominantly affecting knees and ankles, with no axial involvement, associated to HLA-B27, which tend to improve weeks after antituberculous therapy with no sequelae. It is a clear entity that should be taken as such for future studies, clinical classification and diagnosis.

Even though PD diagnosis still raises a challenge for the clinician, PD should be suspected in any patient with oligoarthritis and TB infection, especially in countries with high prevalence of TB. We suggest that more studies aiming HLA typing should be undertaken to clarify the involvement of HLA alleles in the pathogenesis of PD.

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