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Pericardial Effusion in a Immunosuppressed Patient, a "Common" Agent

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Abstract

IInfectious processes in transplant patients suppose a diagnostic challenge due to the population's exposure to common microorganisms, making it difficult to differentiate between infectivity and pathogenicity. Pericardial effusion is classified as acute, subacute, or chronic (>3 months) and the hemodynamic impact depends on the speed of onset.

Keywords: Parvovirosis, Pericardial Effusion, Immunosuppression

1. Introduction

In half of patients, the primary cause of pericardial effusion is idiopathic, followed by infectious, neoplastic, and finally iatrogenic causes. In the first month after a solid organ transplant, infections usually originate from the recipient, the donor, or the surgical procedure. From the second to the sixth month, immunosuppression peaks, leading to opportunistic infections, and from the sixth month onward, the risk of infection decreases, with infections similar to those of immunocompetent patients (12).

Parvovirus B19 is a DNA virus belonging to the Parvoviridae family of the Erythroparvovirus genus. Between 50% and 80% of adults have measurable parvovirus B19-specific IgG antibodies [1]. It is transmitted from person to person, primarily via the respiratory route, and can also be transmitted through close person-to-person contact, fomites, vertical transmission, and hematogenous transmission [2]. Infection in immunocompetent patients often goes unnoticed, and 25% may present mild, self-limiting symptoms such as infectious erythema or oligoarthritis [3,4]. However, in immunocompromised patients, parvovirus B19 can destroy erythrocyte progenitor cells, producing anemia or other associated cytopenias with less specific clinical symptoms [4].

2. Case Report

2.1. Personal History

A 66-year-old male kidney transplant recipient (transplanted in 2010 due to polycystic kidney disease) was under immunosuppressive therapy with tacrolimus, mycophenolate, and prednisone. He had a history of hyperuricemia with crystal arthropathy, mild hyperhomocysteinemia, and chronic diarrhea, followed by internal medicine for two years. Surgical history included jejunal resection and gastric polypectomy in 2004.

2.2. Presenting Illness & Physical Examination

The patient was referred to the emergency department after imaging revealed a large pericardial effusion with right atrial compression and suspected tamponade. On arrival, he was hemodynamically stable (BP 156/89 mmHg, HR 99 bpm) and asymptomatic. Physical examination revealed infrapatellar and dependent limb edema, as well as signs of chronic venous insufficiency. Cardiopulmonary auscultation was unremarkable.

2.3. Initial Management & Complication

Pericardiocentesis was performed the following day, draining 900 mL of bloody pericardial fluid. Within 24 hours, the patient developed a complication: colonic perforation secondary to subxiphoid pericardiocentesis. Emergency laparotomy was performed, with removal of the catheter and suturing of the transverse colon and left hepatic lobe, followed by antibiotic therapy. After stabilization, the patient was admitted to the Internal Medicine Department for etiological evaluation.

2.4. Investigations

The emergency department laboratory results revealed a pro-natriuretic peptide of 1126 pg/ml, a hemoglobin of 9 g/dL (with a previous reading of 11 g/dL), with no elevation of acute phase reactants, and a chest X-ray with an elevated cardiothoracic index. The transthoracic echocardiogram showed severe pericardial effusion without right-sided involvement and preserved right ventricular function. The initial pericardial fluid showed biochemical findings without glucose consumption or elevated proteins suggestive of

infection. Microbiological isolation was positive for Klebsiella pneumoniae, Escherichia coli, Streptococcus lutetiensis, and anaerobes. Parvovirus B19 DNA was detected (viral load 134 IU/mL). Mycobacterial culture, PCR for M. tuberculosis (fluid medium), cytology for malignant cells and autoimmunity were negative.

2.5. Clinical Course

Despite initial drainage, pericardial effusion persisted and increased. A pleuropericardial window was performed, revealing a thickened, fibrotic pericardium and draining 700 mL of serous fluid. This second sample had similar biochemistry but 10 times higher leukocyte count. The patient was later transferred to the nephrology service. Three months later, he developed pain and swelling in the right knee. Arthrocentesis yielded yellow, cloudy synovial fluid with:

- 26,070 leukocytes/µL (89% polymorphonuclear)
- 1,000 red blood cells/µL
- Glucose: 168 mg/dL
- Protein: 3.10 g/dL
- Cultures: Negative
- PCR: Parvovirus B19 viral load: 11,843 IU/mL

Blood PCR showed Parvovirus B19 viral load of 4,142 IU/mL. The patient also presented with worsening renal function and anemia. He was treated with five doses of non-specific intravenous immunoglobulin (IVIG), resulting in clinical improvement, as well as recovery of renal function and hemoglobin levels.

2.6. Diagnosis

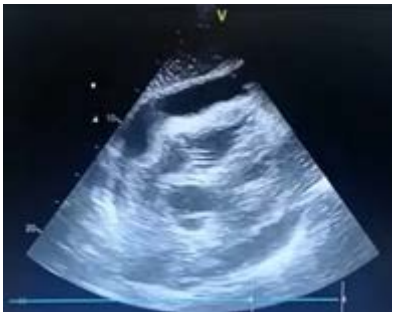
Serositis, monoarthritis, and anemia secondary to infection by parvovirus B19 in an immunosuppressed patient.

3. Discussion

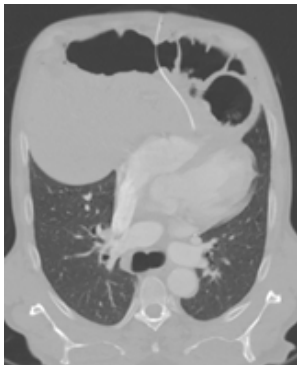
Parvovirus B19 infection in immunosuppressed patients is most common in the second post-transplant period. In a review of 98 recipients, more than half of whom received kidney transplants, the median time to symptom onset was 7 weeks. The most common clinical manifestation was anemia, in 98% of cases, while organ invasion was seen in 11% of patients, of whom 2% were probable cases of myocarditis [5]. In some immunocompromised patients, parvovirus B19-specific DNA has been reported to circulate for months or even years after infection. Therefore, the detection of parvovirus B19 DNA, especially at very low levels, does not necessarily indicate an acute or recent infection [6]. This is the main reason why the possibility that the finding of parvovirus B19 in the pericardial fluid was the cause of myocarditis and the resulting pleural effusion was dismissed.

The lesson learned from this case is that we should not underestimate the value of complementary testing before assuming an idiopathic cause. Although studies and clinical experience guide us in another direction, we must individualize each patient. In cases where we do not have an accurate diagnosis, we must at least assess the benefit-risk of applying empirical treatment and thereby confirm or rule out our suspicions. In this case, the patient progressed favorably with the five doses of immunoglobulins. We should suspect the possibility of parvovirus B19 infection in patients who present with symptoms of chronic reticulocytopenic anemia in the context of immunosuppression. The diagnostic approach depends on the host and the clinical presentation.

3.1. Anex Doccument



1. Echocardiography: Subxiphoid View Showing Diastolic Compression of Right Chambers Due to Severe Pericardial Effusion



2. CT Scan: Hyperintense Image Corresponding to the Pericardial Drainage Catheter

3.2. Pericardial Fluid

Biochemistry

- Cellularity: 5000 red blood cells; 95 leukocytes (cel/mcl)
- Glucose: 109 mg/dL.
- Proteins: 3,72g/dL
- ADA: 13,2.

Microbiology

1. Positive for:
2. K. pneumoniae, E. coli,
3. S. lutetiensis. y anaerobios
4. Positive viral load for parvovirus B19 134 copies/mL

Autoimmunity ANA, ENA, ANCA, ECA negative. Normal proteinogram.	**Serology** VIH, CMV, syphilis, and VHC negative. IgG parvovirus B19 positive and IgM negative.
Metabolic T3, T4, TSH/ Normal urea.	**Neoplastic** Citology negative. Negative tumor markers.

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