Muscle involvement in inflammatory bowel disease is uncommon; in fact, only 7 cases associating PM and UC have been published to date[3–9] (Table 1). Only in the patient we describe was the diagnosis reached because of the increase in muscle enzymes. The response to glucocorticoid treatment was so good, in general, except in an individual who was refractory to several immunosuppressive agents and whose disease entered remission with mycophenolate mofetil.[9] It has been suggested that the basis for the development of PM in UC results from the common immune-mediated mechanism, in which bowel inflammation and the damage to the mucosa would lead to a release of antigens, stimulating the production of antibodies that would damage the muscle. In conclusion, the development of PM in UC should be taken into account, especially when these patients describe symptoms such as myalgia, muscle weakness or present an increase in muscle enzymes. Although there are few cases reported in the literature, it may be underdiagnosed given that the symptoms may be few and not very specific, and can be attributed to other causes.

## Conflict of Interest

The authors declare they have no conflict of interest directly or indirectly related to the contents of this manuscript.

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## Spondylodiscitis Without Endocarditis Caused by *Streptococcus mitis*☆

## *Espondilodiscitis sin endocarditis causada por* Streptococcus mitis

*To the Editor,*

Spondylodiscitis is an infection of an intervertebral disc and the vertebrae. Its incidence is on the rise in recent years. The causative microorganism in most adult patients is *Staphylococcus aureus*. To date, *Streptococcus* species have had little relevance as causative agents of vertebral osteomyelitis. However, the number of cases caused by these microorganisms has increased in recent years.[1] We report the case of a patient with spondylodiscitis caused by *Streptococcus mitis*.

The patient was a 49-year-old man, an ex-smoker, with nothing else remarkable in his clinical history. He presented with a 4-week history of low back pain with inflammatory features and pain that radiated down his left leg. He did not have associated fever or metabolic syndrome. There was no evidence of skin lesions, either, and he did not consume farmhouse dairy products. He had not undergone any dental procedures in the preceding 2 years. Notable findings in the results of laboratory tests were the absence of leukocytosis in the complete blood count and a normal differential leucocyte count, but elevation of acute-phase reactants was detected (erythrocyte sedimentation rate [ESR], 80 mm; C-reactive protein [CRP], 42 mg/L). Serological tests for human immunodeficiency virus and *Brucella* were negative, as was the tuberculin skin test. A radiograph of the lumbar spine revealed a slight irregularity in the vertebral endplate below L5. Magnetic resonance imaging of the lumbosacral spine disclosed signs compatible with spondylodiscitis involving L5–S1. Blood cultures were negative. A transthoracic echocardiogram showed no evidence of endocarditis. Finally, a biopsy and fine-needle aspiration cytology were performed, both under computed tomography (CT) guidance. Bacterial culture yielded colonies of *S. mitis* that were sensitive to all the different classes of antibiotics except penicillin. Four hours after the vertebral biopsy, a blood sample was again obtained for culture, in which *S. mitis* was also isolated. Treatment consisted of intravenous ceftriaxone for 3 weeks, followed by another 3 weeks with oral levofloxacin. The patient responded well to the treatment, with resolution of the clinical signs and normalization of the acute-phase reactants.

To date, 7 cases of spondylodiscitis caused by *S. mitis* have been reported in the English-language literature[1–3] (PubMed: Spondylodiscitis and *S. mitis* 1969–2015). *S. mitis*, which belongs to the group of viridans streptococcal species, forms part of the commensal flora of the mouth and nasal sinuses and is a rare cause of spondylodiscitis. Spondylodiscitis is uncommon in adults. The predisposing factors are diabetes mellitus, malnutrition, intravenous drug use, immunodeficiency, neoplasms, prolonged glucocorticoid therapy, chronic kidney disease and cirrhosis. It usually presents with inflammatory back pain, which can be accompanied by fever and clinical signs of a systemic disorder.[2] In contrast to spondylodiscitis caused by *S. aureus* and by streptococci from a group other than viridans, that attributed to viridans streptococci has a more subacute course, with less systemic involvement. For this reason, the diagnostic delay is usually longer. The majority of the patients with infection caused by this microorganism present with infective endocarditis, as well. Thus, performance of echocar-

☆ Please cite this article as: Prior-Español Á, Mateo L, Martínez-Morillo M, Riveros-Frutos A. Espondilodiscitis sin endocarditis causada por *Streptococcus mitis*. Reumatol Clin. 2016;12:362–363.

diography is indispensable in these patients.[1–5] Laboratory tests reveal leukocytosis and neutrophilia, as well as elevated acute-phase reactants (ESR, CRP). Blood cultures are essential for the microbiological diagnosis and avoid the need for more invasive procedures. Imaging studies (plain radiography, CT or nuclear magnetic resonance) are performed to exclude other diseases and to identify signs suggestive of spondylodiscitis, as well as to rule out its complications. The definitive diagnosis requires the isolation of the causative pathogen, either in blood cultures or a biopsy. The latter can be percutaneous, and is generally CT-guided or open.[6] Blood cultures performed during the first few hours after the biopsy have been reported to be more sensitive and, thus, are worthwhile as they can confirm that the infection was caused by the microorganism isolated in the biopsy specimen and is not the result of contamination.[7]

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## Periodic Fever, Aphthous Stomatitis, Pharyngitis and Adenopathy Syndrome and Vitamin D: A Possible Treatment Option?☆

### *Síndrome de fiebre periódica, estomatitis aftosa, faringitis y adenopatías y vitamina D. ¿Una posible opción terapéutica?*

*To the Editor,*

PFAPA syndrome (periodic fever, aphthous stomatitis, pharyngitis and adenitis), or Marshall syndrome,[1] is an autoinflammatory disease with no known genetic basis. It predominantly affects patients in the first decade of life. It is characterized by recurrent episodes of high fever of 3–6 days' duration, accompanied by symptoms that define the condition: pharyngitis, cervical adenitis and aphthous stomatitis. The diagnosis is based on the clinical findings, and is reaffirmed by the elevation in acute phase reactants during the attacks, which returns to normal during asymptomatic periods, and negative results on microbiological tests. Although the episodes become increasingly less frequent until complete remission is achieved, the recurrence of the flares has a negative effect on the quality of life of the patient. The therapeutic options may have certain controversial aspects. Oral corticosteroids are the treatment of choice to resolve the attacks. However, after their use, an increase in the frequency of recurrence of the episodes has been reported.[2] Tonsillectomy is performed in cases of refractory disease, but its use is questionable, given that the majority of the patients eventually achieve complete remission. Different drugs, like cimetidine and colchicine, have been employed as prophylaxis in the attempt to reduce the number and severity of the episodes.[3] In recent years, a possible relationship between PFAPA syndrome and low serum vitamin D levels is being studied, and the question has been raised as to whether treatment with this vitamin could modify the course of the disease.[4]

We report the case of a 32-month-old girl with a previous history of multiple visits to the emergency department due to episodes of fever, vomiting and recurrent tonsillitis, which occurred every 6–8 weeks. During these episodes, she had a transient elevation of acute phase reactants (maximum C-reactive protein: 15.39 mg/dL) and of leukocytes of up to 15,000/μL with 85% neutrophils, and negative microbiological tests. As PFAPA syndrome was suspected, in one of the flares, she was given a single dose of oral prednisolone (1 mg/kg/dose) as a diagnostic-therapeutic test, and the symptoms resolved immediately. It was observed that, after several flares in which she was treated with corticosteroids, the episodes became increasingly frequent. The serum 25-OH-vitamin D level was determined, and was found to be deficient (23.7 ng/mL). Treatment was begun with 400 IU of cholecalciferol and was maintained until the patient achieved a normal serum vitamin D level (40 ng/mL). Since the initiation of treatment she has experienced a reduction in the number and severity of the episodes (Fig. 1), and had only 2 mild attacks over the following 12 months.

In recent years, we have begun to realize the importance of the role of vitamin D as an immune modulator.[5] Several epidemiological studies have focused on determining the correlation between vitamin D deficiency and the risk of inflammatory diseases, including allergic and autoimmune disorders,[6,7] and the possibility that vitamin D could be a therapeutic option in some of these diseases is being taken into consideration.[8] Uncontrolled studies have found that, in patients with PFAPA, vitamin D therapy reduces the number, duration and severity of the episodes.[9] However, there are no clinical trials comparing vitamin D with other therapeutic options or placebo. With our current level of knowledge, it is impossible to determine whether the favorable outcome is due to the treatment or to the natural course of the disease. Moreover, the dose of vitamin D and the required duration of treatment have yet to be defined. Therefore, in our patient, we opted for a standard dose until her serum 25-OH-vitamin D level had returned to normal. Despite these limitations, the use this therapy can be considered in patients with PFAPA with vitamin D deficiency, above all, because of its safety profile as compared to other therapeutic alternatives.

☆ Please cite this article as: Rico Rodes A, Sabrido Bermúdez G, Llombart Vidal A, Alcalá Minagorre PJ. Síndrome de fiebre periódica, estomatitis aftosa, faringitis y adenopatías y vitamina D. ¿Una posible opción terapéutica? Reumatol Clin. 2016;12:363–364.