

## No Serological Indications That Sjögren's Syndrome Is Linked with Human Parvovirus B19 Infection

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### Abstract

**Background:** The association of Human parvovirus B19 infection with autoimmune disorders has been well documented. Some recent studies have suggested a possible role of B19 in the Sjögren's syndrome pathogenesis.

**Objectives:** To investigate the prevalence of anti-B19 IgM and IgG antibodies and to detect viral DNA in serum samples of patients with Sjögren's syndrome.

**Methods:** We assessed antibodies against Parvovirus B19 in serum samples from two patients with Sjögren's syndrome and from healthy volunteers. Blood samples were examined for B19 serology and genome expression.

**Results:** Positivity for IgM and IgG antibodies to B19 was not detected in any patients. None of these patients showed evidence for B19 viremia.

**Conclusions:** This study showed that none of the patients with Sjögren's syndrome showed serological markers of Human Parvovirus B19 infection. Therefore, we don't support an association between Sjögren's syndrome and B19 infection.

**Keywords:** Sjögren's syndrome, Human Parvovirus B19

### Introduction

The Sjögren's syndrome, also called sicca syndrome, is a chronic disease of autoimmune origin. This syndrome affects the glands that normally produce lubricating fluids. Salivary glands of the mouth and lachrymal glands of the eyes are the most commonly affected: they then cease to function. This results in an abnormal dryness of the eyes and mouth (3). The exact cause of Sjögren's syndrome is unknown. Some evidence suggests that genetic and environmental factors play a role in the onset of the disease. According to other research, bacterial or viral infections are likely to trigger an immune attack on glands that produce wet substances.

Parvovirus B19 is an *Erythrovirus* and is the only known member of the *Parvoviridae* family of viruses causing diseases in Human. B19 is a small, nonenveloped virus with icosahedral capsid containing single-stranded DNA genome, which is approximately 5.5 Kb (7). The B19 was identified as the causative agent of transient aplastic crisis of hemolytic disease (Pattison et al, 1981), the common childhood exanthem called fifth disease (1;8) and polyarthralgia syndrome in normal adults (10).

In addition, B19 parvovirus may be responsible for some cases of hydrops fetalis (11). B19 also associated with several autoimmune diseases (2; 6) The aims of this study were to investigate the prevalence of anti-B19 IgM and IgG antibodies and to detect viral DNA in patients with Sjögren's syndrome and who are seropositive for IgM antibodies.

### Patients and methods

**Patients:** We report on two patients who are diagnosed with Sjögren's syndrome according to the criteria proposed by the European Community for Sjögren's syndrome. Patients selected were in the active phase of disease.

**Controls:** We analyzed sera samples obtained from healthy blood donors.

## Laboratory study

IgM and IgG antibodies to B19 were determined in sera by Enzyme-Linked Immunosorbent Assay (ELISA) (Biopharm, Germany, Ridascreen). Recent B19 infection was defined as the presence of positive IgM (with or without IgG anti-B19). In this case, we proceed by DNA purification using the QIA Amp Kit (Qiagen, Hilden, Germany), then amplification by Nested PCR. However, past B19 infection was defined as the presence of IgG antibodies in serum samples in the absence of IgM antibodies.

## Results

IGM antibodies to B19 were not detected nor in patients with Sjögren's syndrome nor in controls. Additionally, serological evidence of past B19 infection was absent in patients.

## Discussion

Taking into account all results, it appears that, in the situation of Sjögren's syndrome, subjects enrolled in this clinical picture have not contracted the virus. This excludes any possibility of an association between Parvovirus B19 infection and this disease. Therefore, our results seem to correlate with those of the team of De Stefano et al (5) who have shown, thanks to a serological study followed by a molecular study, the presence of a past Parvovirus B19 infection in only one subject with sicca syndrome. This was also observed in one subject in the control group. In addition, and as part of this same study, no patient showed a recent infection with this viral agent. However, the presence of Parvovirus DNA in salivary gland biopsies may be accidental, and therefore, cannot assert a direct association between Parvovirus B19 infection and the pathogenesis of Sjögren's syndrome.

Besides, the team of De Re et al (4), when used PCR to detect viral DNA in tissues damaged during the course of the disease, failed to demonstrate a detectable viremia despite the high sensitivity of PCR. In addition, a study undertaken by Casals et al (9) has highlighted a past Parvovirus B19 infection associated with cytopenia in patients with Sjögren's syndrome. This study also showed that the prevalence of this infection in patients was about 35% and was almost similar to that observed in the control group in which the prevalence was about 37%. At this stage, it would be possible to admit that the cytopenia and B19 infection would likely move patients immunologically and genetically predisposed.

## Conclusion

To date, it has not been possible to demonstrate the exact mechanism by which B19 induce Sjögren's syndrome. The exact correlation between B19 infection and installation of this disease requires further investigation based primarily on further in the presence of large-scale sampling studies.

## References

- Anderson, MJ, Lewis, E, Kidd, IM, Hall, SM, & Cohen, BJ (1984). An outbreak of erythema infectiosum associated with human parvovirus infection. *J. Hyg. (Lond)*, 93, 85-93.
- Cope, AP, Jones, A, Brozovic, M, Shafi, MS, Maini, RN (1992). Possible induction of systemic lupus erythematosus by human parvovirus. *Ann Rheum Dis*, 51 (6), 803-804.
- Daniels T, Fox PC (1992). Salivary and oral components of Sjögren's syndrome. *Rheum Clin Dis North Am*, 18, 571-589.
- De Re, V, De Vita, S, Battistella, V, Marzotto, A, Libra, M, Ferraccioli, G, Boiocchi, M (2002). Absence of human parvovirus B19 DNA in myoepithelial sialadenitis of primary Sjögren's syndrome. *Ann Rheum Dis* 61 (9), 855-856.
- De Stefano, R, Manganelli, S, Frati, E, Selvi, E, Azzi, A, Zakrzewska, K, Marcolongo, R (2003). No association between human parvovirus B19 infection and Sjögren's syndrome. *Ann Rheum Dis*, 62, 86-87.
- Finkel, TH, Török, TJ, Ferguson, PJ, Durigon, EL, Zaki, SR, Leung, DY, Harbeck RJ, Gelfand, EW, Saulsbury, FT, Hollister, JR (1994). Chronic parvovirus B19 infection and systemic necrotizing vasculitis: opportunistic infection or aetiological agent? *Lancet*, 343 (8908), 1255-1258.
- Heegaard, ED, Brown, KE (2002). Human parvovirus B19. *Clinical Microbiology*; 15: 485-505.
- Pattison, JR, Jones, SE, Hodgson, JS, Davis, LR, White, JM, Stroud, CE, & Murtaza, L (1981). Parvovirus infections and hypoplastic crisis in sickle-cell anaemia. *Lancet*; 1: 664-665.
- Ramos-Casals, M, Cervera, R, Garcia-Carrasco, M, Vidal, J, Trejo, O, Jimenez, S, Costa, J, Font, J, Ingelmo, M (2000). Cytopenia and past human parvovirus B19 infection in patients with primary Sjögren's syndrome. *Semin Arthritis Rheum*, 29 (6), 373-378.
- White, DG, Woolf, AD, Mortimer, PP, Cohen, BJ, Blake, DR, & Bacon, PA (1985). Human parvovirus arthropathy. *Lancet*, 1, 419-421.
- Yaegashi, N (2000). Pathogenesis of non immune hydrops fetalis caused by intrauterine B19 infection. *Tohoku J. Exp. Med*, 190, 65-82.