



LETTER TO THE EDITOR

Acute Thyroiditis Complicating a Systemic Parvovirus B-19 Infection: A Unique Adult Case Report in Italy and Literature Review

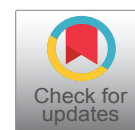
Antonio Mastroianni, MD^{1*}, Sonia Greco, MD¹, Valeria Vangeli, MD¹, Maria Vittoria Mauro, MD² and Roberto Manfredi, MD³

¹Infectious & Tropical Diseases Unit, "Annunziata" Hub Hospital, Cosenza, Italy

²Microbiology & Virology Unit, Annunziata Hospital, Cosenza, Italy

³Infectious Diseases Institute, Alma Mater Studiorum University of Bologna, Bologna, Italy

*Corresponding author: Dr. Antonio Mastroianni, MD, Infectious & Tropical Diseases Unit, "Annunziata" Hub Hospital, Azienda Ospedaliera di Cosenza, Vialledella Republicas n.c, I-87100, Cosenza, Italy, Tel: +39 0984-68-18-33; +39-349-54-44-330



Keywords

Acute thyroiditis, Adult, Parvovirus B 19 infection, Virus-induced autoimmune disorders, Graves-Basedow disease

Introduction

The etiopathogenetic link between Graves' disease, COVID-19 and vaccination is of current and intriguing importance [1]. A very rare case of acute Graves-Basedow adult disease prompted by a systemic *Parvovirus B-19* infection is presented and discussed, on the basis of the most recent literature evidences in this field. *Parvovirus B19* is a small virus parasiting erythrocytes frequently underestimated in clinical practice, when excluding patients with severe hematological diseases [1]. A cutaneous rash involving upper extremities, polyarthralgia, and the association with the chronic fatigue syndrome have been described in otherwise immunocompetent subjects.

A 40-year-old previously healthy male was hospitalized through the Emergency Room of our Hospital because of hyperpyrexia, chills, tachycardia, a diffuse significant polyarthralgia of small joints, and the subsequent appearance of a cutaneous rash involving upper limbs. The patient also complained of diarrhoea, restlessness, especially during the night

with sleep disturbances and tremors. Concurrently, an acute severe thyroiditis appeared (Graves-Basedow disease), treated with corticosteroids, thiamazole and a beta-blocker. A frankly positive anti-Parvovirus IgM and IgG serology accompanied a very elevated Parvovirus B19 viremia (139,000 copies/mL), thus confirming the systemic Parvoviral infection. As a part of a periodic laboratory screening, thyrotropin and thyroideal hormone levels were performed. Moreover, a few months before hospitalization our patient performed an endocrinological assessment at a public endocrinological service of our city due to a persistent infertility issue, which excluded any endocrinological-thyroideal problem. Upon admission, very high free thyroxine levels (77.7 ng/L), completely suppressed TSH levels, elevated anti-thyroid peroxidase antibodies (342 KUI/L), intensely positive anti-TSH receptor antibodies (> 40 U/L, together with positive serum anti-thyroglobulin antibodies (1,232 KUI/L), confirmed the diagnosis of autoimmune thyroiditis. All other microbiological assays tested negative for EBV, CMV, HBV, HCV, HIV, HTLV-I, SARS-CoV-2 and other pathogens, while a full laboratory workup for all searchable autoimmune diseases also proved negative. A neck ultrasonography showed a thyroideal gland of increased volume with dyshomogeneous structure and a diffusely increased

vascularization, as for an acute thyroiditis, in the absence of neck adenopathies and thyroid nodules. Parvoviral detection in the gland tissue was not available at our teaching Hospital, while this invasive procedure was deemed unreasonable in a real life context, where an etiologic treatment for *Parvovirus B19* is not available. The subsequent monthly follow-up did not disclose complications, save a mild and self-limiting skin rash, probably due to thiamazole. Residual hypothyroidism was ruled out after a prolonged endocrinological monitoring. We hypothesized that parvovirus infection triggered a thyroid autoimmune response that caused hyperthyroidism, similarly to what was reported for EBV infection as well as HIV and HTLV-I.

Prolonged viral infections represent a known trigger for autoimmune-related disorders, with HCV, HBV and EBV as the major determinants of *Hashimoto* thyroiditis [2], apart from recent observations regarding the potential role of SARS-CoV-2, too [3]. Several reports discussed about the emerging role of *Parvovirus B19* in the setting of acute thyroiditis especially in children, while adult cases are not represented until now, as to the best of our knowledge. A long-term persistence of *Parvovirus B19* together with the continued expression of *Parvovirus* antigens in non-erythroid tissues may play a key role in triggering autoimmune diseases like thyroiditis. In parallel, the progress of all diagnostic virological testing potentially allow a direct *Parvovirus* detection in tissues, thus improving current diagnostic tools available on thyroid specimens, too. The wise observation regarding the questioned role of viral infection in autoimmunity remains extremely up-to-date: the pathogenetic association between autoimmune disorders and a prolonged *Parvovirus B19* infection deserves in -depth investigation, and further clinical description. In conclusion, our report of an acute

thyroiditis during a documented, systemic *Parvovirus B19* infection in adulthood adds significantly to the present knowledge. We suggest it is reasonable to routinely assess the thyroid functions both in the acute phase and during the convalescence of *Parvovirus B19* infection. Human *parvovirus B19* may be a candidate virus for environmental factors determinant for the appearance of autoimmune thyroid diseases (AITD) in susceptible subject and the exact relationship between *Parvovirus B19* and thyroid disorders needs to be more extensively investigated.

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None.

Conflicts of Interest

None.

Patient's Consent

A signed informed consent for publication was obtained, and the manuscript is in accordance with the institution's ethics committee.

References

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