RELAPSE OF MULTISYSTEM INFLAMMATORY SYNDROME IN CHILDREN (MIS-C) DURING THE CORTICOSTEROID TAPERING, DESCRIPTION OF TWO CASES.

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Introduction

The MIS-C is a syndrome that affects young individuals arising with fever and multisystem organ involvement (cardiac, hematologic, gastrointestinal, renal, respiratory, dermatologic or neurological).
It is caused by a massive inflammatory response to the SARS-CoV-2 infection.
It shares many similarities with Kawasaki syndrome, therefore many cases of relapse after MIS-C were not expected.

Case 1

An 11-years old boy suffered from MIS-C with gastrointestinal and cardiac involvement.

Cardiovascular involvement: pericardial effusion in subacute phase and dilation of both coronaries, with no signs of ventricular dysfunction.

Therapy: intravenous immunoglobulin and methylprednisolone (3 mg/kg/die) two weeks after the start of symptoms.

Relapse: About eight weeks after the diagnosis, when the patient was tapering the prednisone, he presented thoracic pain and fever.
The blood exams showed alteration of C-reactive protein, ferritin, increase of D-dimer and NTproBNP.
The cardiological evaluation revealed clear clinical and instrumental signs of relapsing pericarditis (typical pain, EKG, evident pathological signs in the echocardiography).

Therapy: It was thereby decided to prescribe Anakinra, a biological drug functioning as IL-1 receptor antagonist, with an immediate clinical improvement. The echocardiographic control exposed the progressive resolution of the pericarditis, and a minor myocardial and coronary involvement (slight septal dyskinesia, altered left ventricular function and Global Longitudinal Strain, left coronary ectasia).

Case 2

A 12-years girl was diagnosed with MIS-C, manifested with arthritis, gastroenteric and cardiac involvement.

Cardiovascular involvement: atrioventricular block of first grade (PR 280 ms).

Therapy: intravenous immunoglobulin and prednisone (1 mg/kg), two weeks after the beginning of the symptoms.

Relapse: About three weeks after the first treatments, once the deltacortene was suspended, she developed fever, asthenia, diffused arthralgia.
The blood exams showed a high CRP and ferritin, an increase in hepatocellular enzymes and bilirubin.
The patient was treated with corticosteroid.
A month later, during the tapering down of corticosteroid, she re-presented fever, arthralgia and vomit. The EKG showed an atrioventricular block (PR 250ms). Tapering down the corticosteroids, the clinical condition worsened and the blood labs came back with high inflammatory markers and an alteration of the liver profile. The echocardiography detected a slight septal dyskinesia and a posterior pericardial effusion.

Therapy: The tapering down program of corticosteroid was pursued at a slower pace. During the follow up, the AV block was detected intermittently and the cardiac magnetic resonance resulted normal.

Conclusion

By descripting these two cases, we desire to point out the possibility of relapses. The cause of the incomplete extinction of the inflammatory process might be a non-aggressive therapy at the onset (no prescription of boluses of corticosteroid or Anakinra) or the delay of the diagnosis and hence the treatment at the onset.