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International Journal of Current Research
Vol. 10, Issue, 07, pp.71220-71221, July, 2018

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

ISSN: 0975-833X

RESEARCH ARTICLE

HAEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS AFTER RUBELLA-MEASLES VACCINATION IN A 9-MONTH-OLD TODDLER

***Nassih, H.**

Pediatrician at Tinghir Provincial Hospital, Morocco

ARTICLE INFO

Article History:
Received 26th April, 2018
Received in revised form
14th May, 2018
Accepted 09th June, 2018
Published online 30th July, 2018

Key words:

Haemophagocytic lymphohistiocytosis,
Measles vaccination,
Toddler.

Abbreviations:

HLH: Haemophagocyticlymphohistiocytosis
HIV: Human immunodeficiency virus
CMV: Cytomegalovirus
CRP: C-reactive protein
LDH: lactate dehydrogenase
ALAT : Alanine Amino Transferase
ASAT: Aspartate aminotransferase
CPK: Creatine phosphokinase

ABSTRACT

Haemophagocytic lymphohistiocytosis (HLH) is a life-threatening disease resulting from dysregulated activation and proliferation of lymphocytes. Its occurrence after vaccination is possible but exceptional in children. All cases reported in the literature are secondary to measles vaccination. We report the case of a nine-month-old toddler, not consanguineous, with no particular medical history. Having received his first dose of the measles-rubella vaccine. Who presented 7 days after an alteration of the general condition, a generalized papular rash, and an extensive petechial and ecchymotic purpura. A fever of 39.5°c, complicated by a state of convulsive illness, acute diarrhea, and an acute edema of the lungs were noted. The patient admitted to pediatric intensive care unit was intubated-ventilated-sedated and placed on broad spectrum antibiotic therapy. The brain scan followed by lumbar puncture was normal. CRP was slightly increased to 32 mg / l. The remainder of the biological assessment showed bicytopenia (hypochromic microcytic anemia, thrombocytopenia), and predominantly neutrophilic leukocytosis. Acute renal failure, significant hepatic cytolysis (transaminases greater than 10 times normal), and persistent hyponatremia at 129 mmol / l were noted. Feritinemia and LDH were very high. The serologies of hepatitis A, B, C, HIV, and CMV were negative. A realized myelogram showed a histological appearance of a macrophage activation syndrome. The evolution was marked by hemodynamic instability refractory to adapted resuscitation measures. The patient had a multi-visceral failure and died 48 hours after admission. HLH after vaccination against measles remains a rare but formidable complication. Favoring factors, clinical and biological characteristics, as well as the possibility of a genetic predisposition must be specified.

Citation: Nassih, H., 2018. "*Haemophagocytic lymphohistiocytosis* after rubella-measles vaccination in a 9-month-old toddler", *International Journal of Current Research*, 10, (7), 71220-71221.

INTRODUCTION

Haemophagocytic lymphohistiocytosis (HLH) is a life-threatening disease resulting from dysregulated activation and proliferation of lymphocytes. It can be hereditary, or secondary to infection, malignancy, or rheumatologic disease. Its occurrence after vaccination against measles is possible but exceptional in children.

Observation

We report the case of a nine-month-old non-consanguineous infant with no particular medical history, who received his first dose of measles-rubella vaccine, and presented 7 days after an impairment of general condition, a generalized papular rash, and extensive petechial and ecchymotic purpura (Images 1, 2). A fever at 39.5 ° C, complicated by generalized tonic-clonic malaise, acute greenish fluid diarrhea with no abnormal emissions, and acute pulmonary edema were noted. Because of the state of convulsive illness and the acute installation of disturbances of consciousness (confusional state then coma, Glasgow score= 8/15), the patient was admitted to pediatric intensive care unit, where he was intubated-ventilated-sedated, and put under Broad-spectrum intravenous antibiotic therapy (ceftriaxone: 100 mg / kg / day + gentamycin: 3 mg / kg / day). The brain scan followed by lumbar puncture was normal. CRP was slightly increased to 32 mg / l. The remainder of the biological assessment showed bicytopenia (microcytic hypochromic anemia at 8.6 g / dl, thrombocytopenia at 21,000 / mm3), and predominantly neutrophilic leukocytosis (white cell = 29280 / mm3, neutrophils = 20740 / mm3). The blood cultures were sterile. Acute renal failure (urea = 1.22 g / L, creatinine = 18 mg / L), significant hepatic cytolysis (ALAT = 167 Ui / L, ASAT = 525 Ui / L), and hyponatremia at 120 mmol / l (persistent despite intravenous correction), were noted. Feritinemia, CPK and LDH were very high (ferritinemia = 1572.5 ng / mL, CPK = 921.2 IU / L, LDH = 1186 IU / L). Viral serologies (hepatitis A-B-C, HIV, and CMV) were negative.

****Corresponding author:*** **Nassih, H.,**
Pediatrician at Tinghir provincial hospital, Morocco.
DOI: https://doi.org/10.24941/ijcr.29953.07.2018

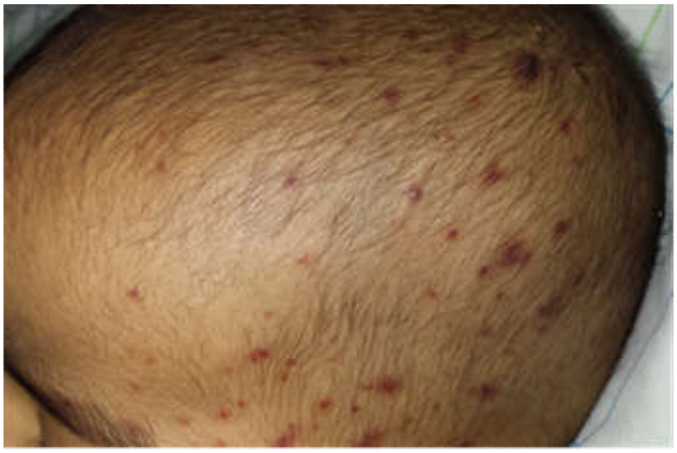

Image 1. Petechial purpura of the scalp

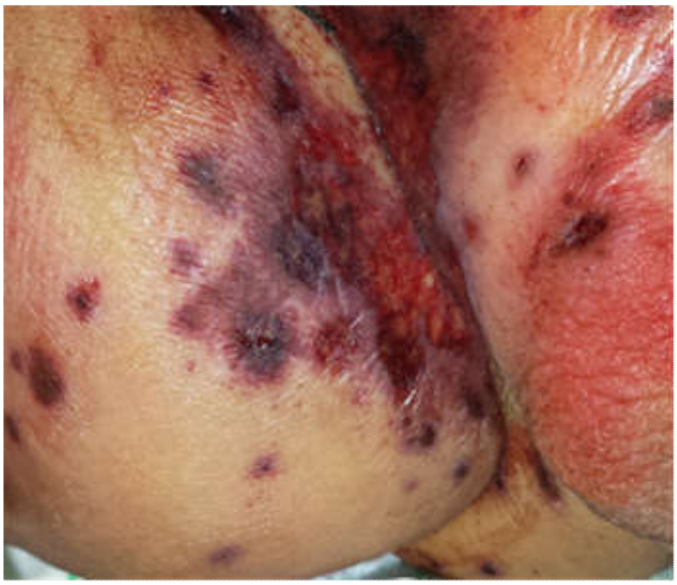

Image 2. Ecchymoticpurpura of the lower limbs

A realized myelogram showed a histological appearance of a macrophage activation syndrome. The evolution was marked by hemodynamic instability refractory to adapted resuscitation measures. The patient died 48 hours after admission to intensive care in a multi-visceral failure state.

DISCUSSION

Secondary HLH is defined by "The Study Group of the Histiocyte Society" as the association of more than 5 of the following criteria: fever, splenomegaly, cytopenia (of more than 2 cell lines), hypertriglyceridemia or hypofibrinogemia, hemophagocytosis (in the bone marrow, spleen or lymphatic ganglia) with weak or absent cytotoxicity of "natural killer" cells, hyperferritinemia; and high levels of soluble CD25 (1). There are few cases of HLH secondary to vaccination reported in the literature (3, 4). The only pediatric case associated with vaccination against measles is published in 2002 in Japan (2). This is the case of a 19-month-old female toddler who developed a persistent fever one week after vaccination, followed by the onset of pancytopenia, hepatic dysfunction, hepatosplenomegaly with marked hemophagocytosis. She was initially put on intravenous immunoglobulin (1 g / kg / day for 2 days) then relayed with oral prednisolone (2 and then 1 mg / kg / day for 3 weeks). But in view of the persistence of recurrent intermittent fever and worsening of pancytopenia, the patient was put on cytotoxic chemotherapy: etoposide, in combination with high-dose dexamethasone according to the HLH94 protocol (5). She responded gradually to treatment and was afebrile at the 5th week of treatment. Treatment was discontinued after 8 weeks, and the patient did not relapse afterwards.

Conclusion

HLH after vaccination against measles remains a rare but formidable complication. Favoring factors, clinical-biological characteristics, as well as the possibility of a genetic predisposition must be specified. Treatment is based on immunosuppressants with high dose of corticosteroids and / or intravenous immunoglobulins.

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