European Review for Medical and Pharmacological Sciences 2012; 16: 1324-1337

Secondary hemophagocytic lymphohistiocytosis in zoonoses. A systematic review

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Abstract. - BACKGROUND: Hemophagocytic lymphohistiocytosis (HLH) is a rare syndrome that is often fatal despite treatment. It is caused by a dysregulation in natural killer T-cell function, resulting in activation and proliferation of histiocytes with uncontrolled hemophagocytosis and cytokines overproduction. The syndrome is characterized by fever, hepatosplenomegaly, cytopenias, liver dysfunction, and hyperferritinemia. HLH can be either primary, with a genetic aetiology, or secondary, associated with malignancies, autoimmune diseases, or infections.

AIM: To focus on secondary HLH complicating zoonotic diseases.

MATERIALS AND METHODS: PubMed search of human cases of HLH occurring during zoonotic diseases was performed combining the terms (haemophagocytic OR haemophagocytosis OR hemophagocytosis OR hemophagocytic OR erythrophagocytosis OR macrophage activation syndrome) with each one of the etiological agents of zoonoses.

RESULTS: Among bacterial diseases, most papers reported cases occurring during brucellosis, rickettsial diseases and Q fever. Regarding viral diseases, most of the cases were reported in patients with avian influenza A subtype H5N1. Among the protozoan zoonoses, most of the cases were reported in patients with visceral leishmaniasis. Regarding zoonotic fungi, most of the cases were reported in AIDS patient with histoplasmosis. No cases of secondary HLH were reported in patient with zoonotic helminthes.

CONCLUSIONS: Zoonotic diseases are an important cause of HLH. Secondary HLH can delay the correct diagnosis of the zoonotic disease, and can contribute to an adverse outcome.

Key Words:
Hemophagocytic lymphohistiocytosis (HLH), Zoonoses, Developing Countries, Epidemiology, Review.

Introduction

Although zoonotic infections are a major burden worldwide – both in terms of immediate and long-term morbidity and mortality[1,2] and in terms of socioeconomical, ecological, and political impact[3] – scientific and public health interest and funding for these diseases remain relatively minor and inadequate[4]. In the present review we will focus on secondary hemophagocytic lymphohistiocytosis (HLH) complicating zoonotic diseases.

HLH is a potentially fatal hyperinflammatory syndrome that is characterized by histiocyte proliferation and hemophagocytosis. HLH may be inherited (primary, familial) and occurs generally in infants or may be secondary to infection, malignancy or rheumatologic conditions, thereby, occurring at any age. The former is a syndrome associated with autosomal recessive disorders that lead to defects in apoptosis induction of virus-infected cells or tumor cells by cytolytic immune cells, including natural killer (NK) cells or cytotoxic T lymphocytes (CTL). Defects of cytotoxic activities of NK or CTL cells, X-linked lymphoproliferative syndrome type 1 (XLP1) and type 2 (XLP2) can also lead to HLH development[5]. Secondary HLH, called also macrophage activation syndrome (MAS), is a common finding in systemic juvenile idiopathic arthritis (sJIA) in which, an apparent

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hybrid situation is present. In fact, several mutations have been reported recently in sJIA[6]. Thus, as in other infection-associated hyperinflammatory syndromes[7-10], activation of receptors and cells of the innate immunity system is likely to play a major role in HLH.

The most typical presenting signs and symptoms are fever, hepatosplenomegaly, and cytopenias. Less frequently observed clinical findings are neurological symptoms, lymphadenopathy, edema, skin rash, and jaundice[11,12]. Common laboratory findings include hypertriglyceridemia, hyperferritinemia, a coagulopathy with hypofibrinogemia, and elevated aminotransferases[11,12]. However, HLH is diagnosed using clinical criteria developed by the HLH Study Group of the Histiocyte Society[13,14] (Table I).

Literature Review

PubMed search of human cases of HLH occurring during zoonotic diseases was performed combining the terms (haemophagocytic, or haemophagocytosis, or hemophagocytosis, or hemophagocytic, or erythrophagocytosis, or macrophage activation syndrome) with each one of the etiological agents of zoonoses and/or one of the diseases indicated in Tables II and III for the period January 1950 to August 2012. A study was considered eligible for inclusion in the systematic review if it reported data on patients with zoonotic diseases who had microscopic signs of hemophagocytosis and/or fulfilled the diagnostic criteria of the HLH Study Group of the Histiocyte Society.

Results

The PubMed search identified 1157 papers. Duplicate publications or papers not reporting clinical cases were excluded. After a scrupulous analysis, 153 papers were further evaluated. In the Table

Table I. HLH 2004 Diagnostic criteria (modified from ref.[13,14]).

The diagnosis of HLH can be established if one of either 1 or 2 below is fulfilled: 1. A molecular diagnosis consistent with HLH 2. Diagnostic criteria for HLH are fulfilled (five out of the eight criteria below):
• Fever • Splenomegaly • Cytopenias (affecting ≥ 2 lineages in the peripheral blood): Hemoglobin < 90 g/l (in infants < 4 weeks: hemoglobin < 100 g/l Platelets < 100.000/ml Neutrophils < 1000/ml • Hypertriglyceridemia and/or hypofibrinogenemia: Fasting triglycerides ≥ 265 mg/dl Fibrinogen ≤ 1.5 g/L • Hemophagocytosis in bone marrow or spleen or lymphnodes • Low or absent NK-cell activity • Ferritin ≥ 500 μg/l • Soluble CD25 ≥ 2400 U/L
Comments: (1) If hemophagocytic activity is not proven at the time of presentation, further search for hemophagocytic activity is encouraged. If the bone marrow specimen is not conclusive, material may be obtained from other organs. Serial marrow aspirates over time may also be helpful. (2) The following findings may provide strong supportive evidence for the diagnosis: (a) spinal fluid pleocytosis (mononuclear cells) and/or elevated spinal fluid protein, (b) histological picture in the liver resembling chronic persistent hepatitis (biopsy). (1) Other abnormal clinical and laboratory findings consistent with the diagnosis are: cerebromeningeal symptoms, lymph node enlargement, jaundice, edema, skin rash. Hepatic enzyme abnormalities, hypoproteinemia, hyponatremia, VLDL ↑, HDL ↓.

Table II. Clinical significant agents of zoonoses found associated with secondary HLH.

Bacteria	References and notes
Anaplasma phagocytophilum	[15]Review
Bartonella sp.	[16]Renal transplant recipients
Borrelia sp.	[17]Lyme disease
Brucella sp.	[18]Analysis of children with brucellosis associated with pancytopenia, Turkey; [19]8 year-old male, Turkey; [20]84 year-old female, antilymphoma chemotherapy; [21]Multicenter retrospective study, Turkey; [22]Retrospective study, 3 patients, Turkey; [23]11 year-old boy, Turkey; [24]5 patients, Spain; [25,26]disseminated intravascular coagulation, Spain; [27,28]; Retrospective study, Saudi Arabia; [29,30]Two and half years old female, India; [31]Pulmonary involvement, Iran; [32]Bone marrow biopsy findings in brucellosis patients with hematologic abnormalities, China;
Campylobacter sp.	[33]*Campylobacter fetus*, AIDS, USA
Capnocytophaga sp	[34]Sudden Sensorineural Hearing Loss, Japan
Clostridium sp.	[35]AIDS; [36]Pancreatic carcinoma
Coxiella burnetii	[37,38,39,37,38,40]
Ehrlichia chaffeensis and *Ehrlichia ewingii*	[41]Two children, USA; [42]Case report, USA; [43]Fatal case, USA; [44]67 year-old white man, disseminated intravascular coagulopathy, USA
Leptospira sp.	India [45,46]Fatal case, Taiwan:
Listeria sp.	[47]*L. monocytogenes*, bone marrow transplant recipient, France
Mycobacterium avium	[48]*M. avium*, AIDS; [49]*M. avium*, Lupus erythematosus
Orientia tsutsugamushi	[50-53]
Rickettsia spp	[54]Murine typhus in returned travelers; [55]MSF, Italy; [52,56]Rickettsia conorii; [57]MSF; [58]MSF, Israel; [59]Fulminant Rocky Mountain spotted fever
Salmonella sp. (excluding S. typhi)	[60]Child suffering from chronic granulomatous disease, associated with septicemia due to *Salmonella* typhi murium;[61]
Viruses	
Crimean-Congo haemorrhagic fever virus	[62-68]Turkey
Hantaviruses	[69]Hemorrhagic fever with renal syndrome, South Korea. [70]Hemorrhagic fever with renal syndrome
Hepatitis E virus	[71]Japan.
Influenza viruses	[72-77]influenza A virus H5N1 subtype; [78]Fatal case of swine influenza virus in an immunocompetent host, USA
SARS coronavirus	[79-82]China, Taiwan
Protozoa	
Babesia sp.	[83-85]Splenectomized renal allograft recipient, USA
Leishmania spp.	[86]Children with HLH treated at the University Children's Hospital in Belgrade; [87-89]Chronic granulomatous disease; [90]Four childhood cases; China; [91]immunocompetent adult-case report and review; [92]A review of situation in Thailand; [93]Retrospective study Clinical analysis on 28 patients with hemophagocytic lymphohistiocytosis syndrome, China; [94] Two cases, India; [95]AIDS, India; [96]28 years man, India; [97]9 cases, India; [98]Illustrative case and review, India; [99]Fatal case, India; [100]Retrospective study, India; [101]Cerebrospinal fluid involvement, Oman; [102]Nine cases, Saudi Arabia. [103]4.5 month-old infant associated with H1N1 virus infection, Turkey; [104]Adolescent, Turkey; [105] Child, Turkey; [106]Child; Turkey; [107]4 year-old boy travel history, Turkey; [108]5 year-old boy, Turkey; [109]18 Turkish children (2 weeks-72 months); [110]Child, pseudomonas septicemia, myelodysplasia, Turkey [111]Greece; [112] Epstein Barr, Cyprus; [113]2 year-old child, Israel; [114]46 year-old woman, Israel;

Table continue

Table II. ***(Continued).*** Clinical significant agents of zoonoses found associated with secondary HLH.

Bacteria	References and notes
Protozoa	
Leishmania spp.	[115]20 month-old boy, Tunisia; [116]2 year-old boy, Tunisia; 117 Tunisia; 118 2 severe cases, Tunisia; [119]15 month-old girl, Travel Spain, Norway; [120]7 year-old previously healthy Czech boy, travel in Italy; [121]16 month-old girl, Spain; [122]Spain; [123]Spain; [124]Spain; [125]Pericardial effusion, Spain; [126]Rheumatoid arthritis, adalimumab, Spain; [127]steroid, bronchial asthma, Spain; [128]France; [129]France; [130]12 month-old girl, France; [131]12 cases, France
Toxoplasma gondii	[132,133]Primary disseminated toxoplasmosis; [134,16,135]renal transplantation; [136]Bone marrow transplantation; [137,138,139]AIDS
Fungi	
Cryptococcus neoformans	[140]A child case, cryptococcal meningoencephalitis, Japan
Histoplasma capsulatum	[139, 141-149]AIDS; [150]AIDS Reconstitution inflammatory syndrome; [151]Pediatric AIDS [152,153]Leukemia; [154]21-year-old man with Still's disease; [155,156]Adult-onset Still's disease, adalimumab, [157]Kidney transplant recipients, USA; [158]Heart transplant recipient, USA; [159]6 year-old boy with chronic mucocutaneous candidiasis, USA; [160]Sarcoidosis on chronic steroid treatment, USA; [161]France; [162]Immunocompetent, India; [163]2 cases, India; [164]Fungal endocarditis, chronic hepatitis C, cryoglobulinemia, renal failure and *Staphylococcus aureus* perinephric abscess and bacteremia
Penicillium marneffei	[165]AIDS, China; [166]AIDS, Thailand; [167]Thailand; [168]China

Table III. Bacterial, viral, fungal and helminthic agents of zoonoses not associated with secondary HLH.

Bacteria	*Bacillus antraci, Chlamydophyla psittaci, Corynebacterium ulcerans, Escherichia coli O157H7, Francisella tularensis, Helicobacter* sp*, Mycobacterium bovis, Mycobacterium caprae, Mycobacterium marinum, Mycobacterium microti, Mycobacterium ulcerans, Mycobacterium genavense, Mycobacterium malmoense, and Mycobacterium farcinogenes, Pasteurella* sp*. Shigella* sp*., Staphylococcus aureus* (clearly associated to animal reservoir), *Streptococcus suis, Streptococcus equi, Streptococcus canis, Streptococcus acidominimus, Streptococcus bovis,, Vibrio* sp*. (excluding Vibrio colera), Yersinia* sp*.*
Viruses	Borna disease virus, California serogroup viruses, Chikungunya virus, Cowpox virus, Ebola virus, Hendra virus, Japanese encephalitis virus, Kyasanur forest disease virus, Lassa virus, Lymphocytic choriomeningitis virus, Marburg virus, Monkeypox virus, Nipah virus, Omsk haemorrhagic fever virus, Oropouche virus, Rabies and lyssaviruses, Rift Valley fever virus, Ross River virus, Sindbis virus, Tick-borne encephalitis, Venezuelan equine encephalitis virus, West Nile virus, Yellow fever virus, Zika virus
Protozoa	*Balantidium coli, Blastocystis hominis, Cryptosporidium parvum, Giardia lamblia, Plasmodium knowlesi, Trypanosoma brucei, Trypanosoma cruzi*
Fungi	*Basidiobolus rana rum, Malasezia* spp*., Micro*sp*orum* spp., *Paracoccidioides brasiliensis, Trichophyton* spp.
Helminths	*Ancylostoma* spp., *Angiostrongylus* spp., *Clonorchis sinensis and Opisthorchis* spp., *Diphyllobothrium* spp*, Dirofilaria* spp*., Echinococcus* spp*., Echinococcus* spp*., Fasciola* spp*., Fasciolopsis buski, Gnathostoma* spp., *Paragonimus* spp*., Thelazia* spp., *Toxocara* spp*., Trichinella* spp.

II are reported all the agents of zoonoses associated with secondary HLH, while in the second column of Table III are listed zoonotic agents not associated with secondary HLH.

Among bacterial diseases, 15 papers reported cases occurring during brucellosis, 16 during rickettsial diseases (*Rickettsia* spp, *Orientia* sp, *Erlichia* spp and *Anaplasma* spp), 6 during Q

fever, 2 during leptospirosis. One paper each reported papers during Lyme disease or *Capnocytophaga* sp. infection. Most of the above papers described cases of secondary HLH occurring in immunocompetent patients without important comorbidities. Cases of *Bartonella*, *Clostridium*, *Listeria*, *Mycobacterium*, *Salmonella* spp and *Campylobacter fetus* infections were less reported and most of them occurred in patients with major comorbidities. Among the zoonotic mycobacterial diseases, HLH was reported only in patients with *Mycobacterium avium* infection affected by HIV infection or by systemic lupus erythematosus.

Among viral diseases, cases were reported in patients with avian influenza A subtype H5N1, swine influenza, SARS coronavirus, Crimean–Congo haemorrhagic fever, hepatitis E virus. Most of cases occurred in immunocompetent patients without important comorbidities.

Regarding the protozoan zoonoses, most of the cases were reported in patients with visceral leishmaniasis. Only in few cases of the 46 articles reporting such cases, comorbidities were present. Nine papers reported cases occurring in patients with toxoplasmosis and most of them were immunocompromised. Two cases were reported in patient with babesiosis.

Regarding zoonotic fungi, 23, four and one paper reported cases occurring in patients with *Histoplasma capsulatum*, *Penicillium marneffei* and *Cryptococcus neoformans* infections, respectively. Most of them occurred in immunocompromised patients. No cases of secondary HLH were reported in patient with zoonotic helminths.

In one case each, a double infection with Epstein Barr virus[112], H1N1 virus[103], *Pseudomonas* septicemia,[110] and S*taphylococcus aureus* perinephric abscess[164], in addition to the zoonotic agent, were reported.

Regarding comorbidities, five papers reported cases occurring in kidney transplant recipients[16,134,135,157], one in a heart transplant recipient[158] and two in bone marrow transplant recipients[47,136]. Twenty papers reported cases occurring in HIV-infected patients[33,48,95,137-139,141-151,165,166]; of them, one occurred in a pediatric patient[165], and one in the course of AIDS Reconstitution Inflammatory Syndrome[150]. In two and four papers leukaemia[152,153] and chronic granulomatous disease (CGD)[60,87-89], respectively, were present. Other major comorbidities reported were rheumatologic diseases[49,126,154-156], which in three cases were under treatment with adalimumab[126,155,156]. Other major comorbidities/immunosuppressive conditions were chronic steroid treatment[127,160], chronic mucocutaneous candidiasis[159], antilymphoma chemotherapy[20], and pancreatic carcinoma[36]. Five papers reported cases occurring after travel to endemic zones[54,80,107,119,169].

Discussion

Zoonotic infections are defined, in general, as infections transmitted from animal to man (and less frequently vice versa), either directly (through direct contact or contact with animal products) or indirectly (through an intermediate vector as an arthropod or an insect)[170]. The main zoonotic features of influenza are represented by the role of animal hosts as reservoirs and substrates for the development of novel strains, and their role in the introduction of these strains into human pathology. Avian H5N1 influenza is a typical zoonotic infection, requiring close contact with infected animal hosts[1]. The current H1N1 pandemic strain stopped being zoonotic after human-to-human transmission emerged as the cause of the pandemic. The single non-human hosts for each of influenza B and influenza C viruses play a minimal role in human disease[1]. Avian influenza A subtype H5N1 infection and severe acute respiratory syndrome (SARS) due to coronavirus (SARS-CoV) share similar pathologic features. Pneumocytes are the primary target of infection, resulting in diffuse alveolar damage. Systemic cytokine activation results in hemophagocytic syndrome, lymphoid depletion, and skeletal muscle fiber necrosis[171]. However, HLH has also been found in fatal cases of H1N1 infection during the pandemic which emerged in April 2009[172].

HIV infection alone or in the presence of other opportunistic and non-opportunistic infections or malignancies has been associated with HLH, and HLH has also been described in the setting of immune reconstitution inflammatory syndrome[150,173,174].

Also rotavirus infection can cause secondary HLH[175], but this is not a frequent finding[176-180]. Of note, animal rotaviruses might be able to cross species barriers, and lack of systematic surveillance of rotavirus infection in small animals hinders the ability to establish firm epidemiological connections[178,180-182].

Almost all the cases associated with bacterial infections were due intracellular organisms frequently causing epatosplenomegaly and leukope-

nia such as *Brucella* and *Rickettsia* spp. Early treatment of brucellosis with appropriate antibiotics will be life-saving[183-187]. We believe that HLH should always be considered in the severe cases of rickettsial diseases, especially if associated with pancytopenia[186,188-194]. More studies are needed to understand whether immunosuppressive treatments (e.g. with steroids) could be beneficial (as we suspect), especially in those cases not responding promptly to antibiotic therapy[55]. No cases were reported in the course of *Escherichia coli* O157H7 infection, even though in a cirrhosis patient a case caused by *E. coli* infection secondary to bacterial translocation has been described[195-197].

Leishmania donovani and *Leishmania infantum* can cause HLH. Moreover *Leishmania* infection by itself can mimic the syndrome, especially in the presence of organomegaly and cytopenia[198-205]. A bone marrow aspirate determines the correct diagnosis[206-209]. Treatment of leishmaniasis-associated haemophagocytic syndrome with amphotericin B results in cure[108,210-214].

Pulmonary involvement in HLH has been reported in some severe cases[31]. Occurrence of pulmonary involvement is quite frequent especially in HLH triggered by viral infections[72-78,183,215-220].

Being rare itself, HLH is diagnosed almost exclusively in seriously ill, hospitalized patients. Diagnosis of secondary HLH may be laborious, but the primary source must be continually sought, particularly in the case of rare pathogens. Furthermore, it should be stressed that the identification of hemophagocytosis in bone marrow aspirate represents only one of 5-8 criteria needed for the diagnosis of HLH[221]. Treatment of secondary HLH is dependent on its cause. Infectious agents should be eradicated promptly, along with administering supportive care. There are no randomized trials for primary HLH, due to the rarity of this disease. Treatment is based on the combination of immune suppression (such as cyclosporin A) and chemotherapy (such as etoposide)[222,223]. Intravenous immunoglobulins may also be beneficial[114]. Treatment should be started without delay, yet it should be kept in mind that the use of immunosuppression may further delay diagnosis and definitive treatment.

Conclusions

Zoonotic diseases are an important cause of HLH. Secondary HLH can delay the correct diagnosis of the zoonotic disease, and can contribute to an adverse outcome. Further studies are needed to understand whether an immunosuppressive treatment could be beneficial in those cases that do not promptly respond to anti-infective therapy.

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