DIAGNOSTIC DILEMMA IN LARGE CAVITY PLEURISY

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Abstract

A variety of conditions can cause pleurisy in children. The most frequent is a results of infection, secondary to a pneumonia or a tuberculosis. In most of the cases the diagnostic is easy, by biochimical and bacteriological examination of the pleural fluid. We know from the literature that empyema is a sign of bacterial pleurisy, but in some cases there is dificult to obtaining the diagnosis of certainty. We present a case of pleural effusion whose unexpected diagnosis followed a long line of intermediate diagnoses. This all have been suggested by clinical evolution, but also by laboratory data. The final diagnosis resulted after the occurrence of laterocervical lymph mass and was confirmed by immunohistochemical studies on ganglion biopsy.

Keywords: pleural effusion, empyema, tuberculosis, Non Hodgkin Lymphoma, children

Introduction

Large cavity pleurisy appers in children in a various causes. Most commonly results secondary to an infection, but may be a primary or a secondary manifestation of many disorders. Pleuresy develops because of excessive filtration or defective absorption of fluid (1). Tiukhtin shows a dominance of tuberculous etiology in over 85% of cases, follow by parapneumonic pleurisy in 10%, tumoral pleurisy in 1%, posttraumatic pleurisy in 0,5% cases (2). Other possible aetiologies are nephrotic syndrome, heart failure, metabolic diseases. The most common form of extra pulmonary tuberculosis is, in children, massive pleural effusion, about Merino and Kim (3). This form of Mycobacterium tuberculosis infection appear most commonly in adolescents and young adults (following a recent infection), rarely in the elderly (by reactivation). Not all the time the diagnosis is very easy in a case of pleural effusion. We may encounter difficulties in obtaining the diagnosis of certainty, because the cultures or the serological diagnosis for mycobacterium, but also for the other infectious etiologies (adenovirus, Mycoplasma) being laborious.

Case presentation

We present a case of 6 years old boy, from rural area, who came in our clinic in october 2016, presenting dyspnoea, fever, asthenia, sweating, dry cough. The boy has a rich history of respiratory infections, and no BCG scars. Clinical presentation showed a overweight boy (W 34kg, T 124cm, BMI more then percentil 97% for age and gender), without palpable lymf nods. Respiratory system showed maquity at percussion to inferior two thirds of the right side, mixed dyspnea, with orthopnee and polypnea (50 breath/min), SaO2 87%. Vesicular breath sound was abolished on the right and we found pleural rub present in both phases of respiration pleural.

After one week of antibiotherapy the child was admitted in the Pediatric Intensive Care Unit (PICU) due to respiratory distress (Fig. 1). Chest X-ray revealed a opacity of medium intensity that occupies the right pleural space and is projected, on profile, on the shadow of the heart (Fig. 2).

Because of respiratory distress pleural drainage has been established an emergency, (drain approximately 200-300ml purulent liquid, daily). Biological dates suggested a bacterial infection, but all the cultures from the blood and pleural efussion was negative.

Biological data: HBG = 12,9 g/dl, HTC = 38,7%, WBC = 20 000/mm3, NEU = 65,1%, L = 22,5%, M = 10,8%, E = 1,9%, B = 1%, TGP=19 UI/l, TGO=33 UI/l, LDH=1210 UI/l, urea=0,13 g/l, creatinine=0,46 mg/l, uric acid=1 mg%, TOTAL PROTEIN=55,3 g/l , albumin=25,8 g/l, alfa 1=5 g/l, alfa 2=11 g/l3, beta=5,8 g/l, gamma=7,4 g/l, A/G=0,88. An important inflammatory syndrome was determined: Fibrinogen = 9,64 g/l , ERS = 65 mm/h, PCR 124mg/dl. We start treatment for 10 days with: Lynezolid (Zyvox) and Ciprinol, for purulent pleurisy.

The unfavorable progression of disease (continued to fever, dyspnea, respiratory distress, with continous oxigenotherapy, pleural fluid quickly recovered) forced reconsideration of the diagnosis. The new exam of pleural fluid showed: ph = 7,5; RIVALTA +; protein = 4,5 g/l; glucose = 0,37 g/dl; cellularity: lymphocites 98%; LDH = 2567 UI/l. Chest X-ray: Radiological evaluation showed the same opacity, without mediastinal adenopathy (Fig. 3).

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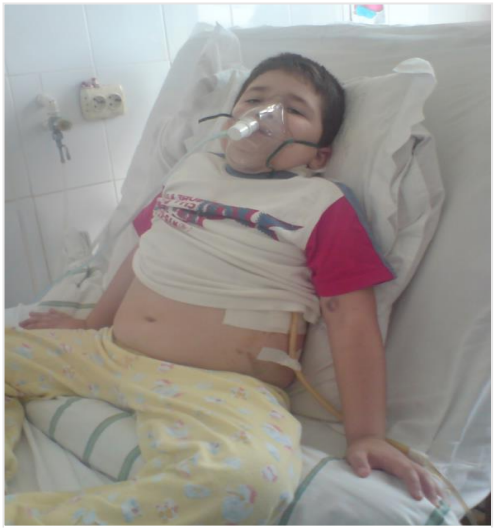

Fig. 1. The child in the Pediatric Intensive Care Unit

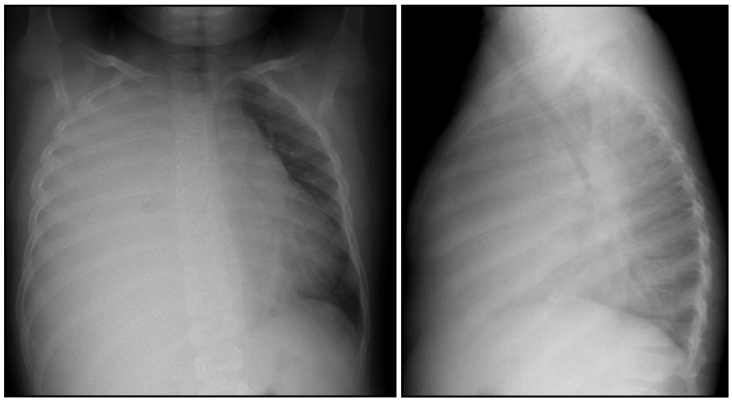

Fig. 2. Chest X-ray

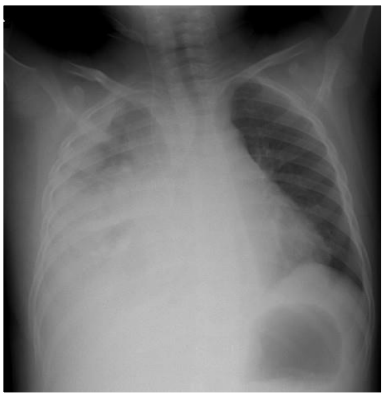

Fig. 3. Second Chest X-ray

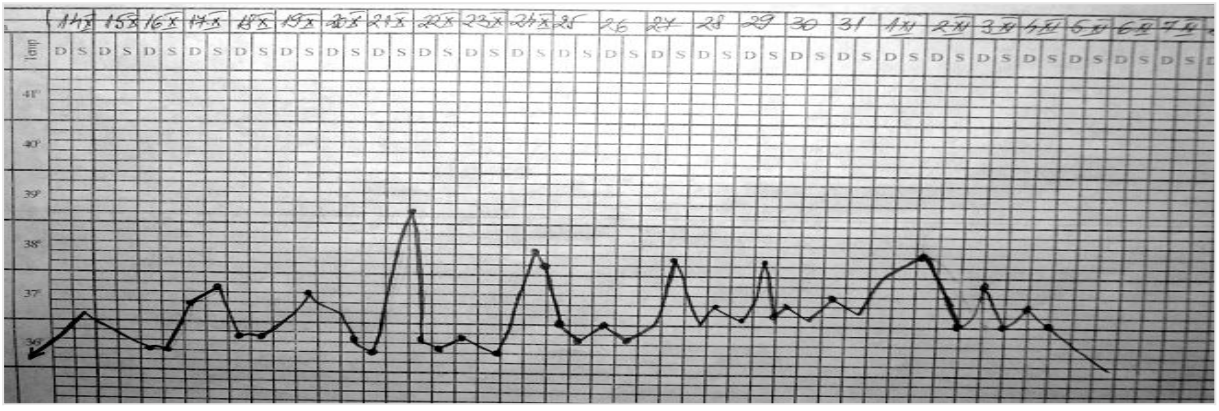

Fig. 4. Fever curve

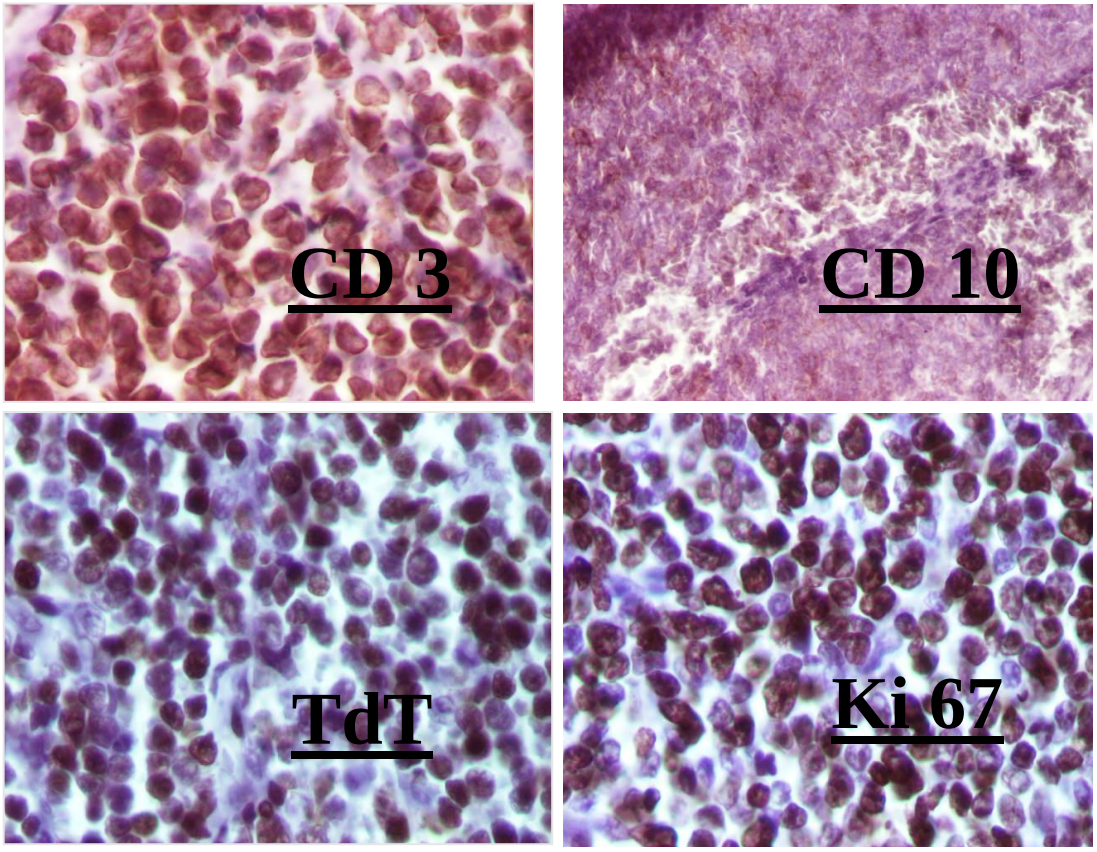

Fig. 6. Immunohistochemical analysis revealed intense positivity of markers CD3, CD 10, TdT

The suspicion of tuberculosis was when the result of test ADA was positive in the pleural fluid (30 U/l), even the IDR 5 u PPD was negative at 72 h. The cultures from Koch bacilli was also collected. The pneumologist considered the diagnosis of Tuberculous pleurisy and has opted for initiating specific therapy with 4 tuberculostatic agents, for 2 months (hydrazide + rifampicin + ethambutol + pyrazinamide), then 2 for another 4 months (hydrazide + rifampicin).

After one week of therapy evolution continued to be unfavorable, the child showed fever (Fig.4), with predominantly in the evening. In the same time appeared a bilateral laterocervical and supraclavicular adenopathic block, with a 2 cm diameter, mobile, painless, local pressing, and oppressive.

On 22 octobre 2016 was performed a ganglionar biopsy. The tuberculostatic treatment was continuing to the 31 X 2016, when the result of biopsy showed non hodgkin lymphoma, atypical mitosis, medium - sized cell proliferation, with homogeneous nuclei, without nucleoli (Fig. 5).

Immunohistochemical analysis was performed with a panel of antibodies effective in paraffin sections, for determining the type of lymphoma. HLA patterns were revealed intense positivity of markers CD3, CD 10, TdT, alog with negative CD20 and Pax 5, suggestive for T-cell phenotype. The antigen Ki 67 was intens positive too, suggesting a lymphoma with a high degree of proliferation (Fig. 6).

To determine the stage of lymphoma we practiced:

- Bone marrow aspiration – no infiltration
- Abdominal ultrasound – no abdominal lymphnods or tumor masses, without hepatic – splenic infiltration
- Lombar puncture – without brain touch

About Lugano classification, which is based on the older Ann Arbor system we diagnosed a III-th stage T – Non Hodgkin Lymphoma (NHL – T).

The tuberculostatic therapy was stopped and was initiated specific therapy for NHL – T. In evolution the fever has quickly disappeared, general condition improved clearly, the child was able to walk and feed. The pleural drain was stopped also (Fig. 6).

Patient parameters, tumor parameters and biological data had led to the development of a prognostic index. The fact that the patient can self-handle in a limited way (more than 50% of the time spent in bed) and the prognostic factors: low age, elevated LDH, adenopathy involving multiple lymph nodes, III-th stage lymphoma, fall into a high risk group, with limited prognosis. For the present the patient are a good response to chimiotherapy.

Discussion

The dilemma of diagnostic started from the fact that the patient had an appearance of pleural empyema, most commonly with bacterial etiology (staphylococcus),

supported by the presence of leukocytosis with polynucleosis and inflammatory syndrome. The unfavorable clinical outcome under large antibiotic therapy, and the positive ADA test reorientated the diagnosis to Tuberculous pleurisy. The diagnosis was cut off by the appearance of the adenopathic block and the anatomo-pathological result of lymphoblastic T lymphoblastic. Reverse, the purulent appearance of the pleural fluid we explain it by the increased number of white blood cells in the fluid. Véronique Minard-Colin shows in his review that T cell lymphoma is the second most common subtype of non-Hodgkin lymphoma (NHL) in children and adolescents. The annual incidence per million inhabitants ranges from 10 in children between 5 and 14 years old, like our child (6). Chaignaud and co established sience 1998 that there is a significantly greater association of T-cell lymphoma and pleural effusions than with Hodgkin's disease (7). The author discusses pleurisy associated with mediastinal T-cell lymphoma. In our case the firs manifestation was pleural effusion, the adenopathic block appearing after 3 weeks of evolution. No mediastinal tumor mass was decent in time.

Conclusions

The particular evolution of our case was at the base of tardiv diagnosis. Maybe a immunohistochemical analysis performed from pleural liquid would have passed the diagnosis earlier. Rapid recognation of the malignant pathology is essential to the vital prognosis of any patient.

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