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BRIEF REPORT

Extreme reactive thrombocytosis in a healthy 6 year-old child☆

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Abstract Thrombocytosis is usually found by chance in children. Reactive or secondary thrombocytosis is the more common form, with infectious diseases being the most prevalent cause of it. Regarding the number of platelets there are four degrees of thrombocytosis; in its extreme degree the number of platelets exceeds 1,000,000/mm^3. We describe a case of extreme reactive thrombocytosis in a healthy 6-year-old child. He required critical care admission for diagnosis and treatment (maximum number of platelets 7,283,000/mm^3). We review the different causes of thrombocytosis in childhood, the differential diagnosis, and the available treatments in case of extreme thrombocytosis.

PALABRAS CLAVE
Pediatría;
Cuidados intensivos;
Neumonía;
Trombocitosis;
Trombosis

Trombocitosis extrema reactiva en un niño sano de 6 años

Resumen La trombocitosis es un hallazgo casual frecuente en pediatría. En niños, predominan las formas secundarias, siendo las infecciones su causa más prevalente. Se distinguen 4 grados de trombocitosis en función del número de plaquetas; en la forma extrema, se supera el 1.000.000/mm^3. Se presenta un caso de trombocitosis extrema reactiva en un niño sano de 6 años, que requirió ingreso en cuidados intensivos para tratamiento y diagnóstico (cifra máxima

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de plaquetas de 7.283.000/mm^3). Se revisan las diferentes causas de trombocitosis en la infancia, se describe el diagnóstico diferencial y se discute sobre los diferentes tratamientos disponibles ante un caso como el descrito.

Introduction

Thrombocytosis is defined as a platelet count of more than two standard deviations higher than the upper limit of normal values (0.5×10^{9}/L).[1,2] It is frequently found by chance in paediatrics. We present a case of extreme thrombocytosis in a healthy child. We describe the treatment, the differential diagnosis and the complications that occurred during its evolution.

Case study

Admission to hospital: A 6-year-old boy attended the emergency department with fever for 12 days, cough, pain in the left side and asthenia. He had no personal or family history of interest. His general condition was fair, with oxygen saturation at 92–93%, tachypnea and left basal hypoventilation. Oxygen therapy was initiated through nasal cannula and the following were requested: (a) complete blood count (CBC): haemoglobin 9.1 g/dL, white blood cells 32,850/mm^3 (70% neutrophils) and platelets 5,301,000/mm^3; (b) coagulation test: normal; (c) biochemistry: normal with C-reactive protein 9.1 mg/dL and procalcitonin 0.36 ng/mL; (d) chest X-ray: left basilar infiltrate and small associated effusion, and (e) blood culture.

With a presumptive diagnosis of pneumonia, he was admitted with oxygen therapy and intravenous treatment with amoxicillin/clavulanic acid (100 mg/kg/day). The thrombocytosis was confirmed and considered to be possibly reactive.

Second day of hospitalisation: owing to an increase in pleural effusion, the patient was transferred. At the receiving hospital, following another chest scan, a watchful waiting approach was maintained, replacing the amoxicillin/clavulanic acid with intravenous cefotaxime (200 mg/kg/day). In addition, a new CBC was performed and increased thrombocytosis was observed. The haemotology service was consulted and recommended carrying out further diagnostic tests, which were normal: (a) red blood cell morphology; (b) coagulation study with protein C and antithrombin III; (c) autoimmunity: antinuclear antibodies, antineutrophil cytoplasmic antibodies and perinuclear antineutrophil antibodies, rheumatoid factor, serum immunoglobins, immune reconstitution, and (d) paraneoplastic phenomena: alpha-foetoprotein and chorionic gonadotropin.

Third day of hospitalisation: in a further ultrasound scan a new consolidation with pleural effusion was found in the right lung base. It was decided not to modify the treatment and to wait to see how the patient evolved.

Fifth day of hospitalisation: the patient had headache, more severe abdominal pain, a worsening of the right pleural effusion and increasing thrombocytosis. Treatment with aspirin (5 mg/kg/day) was initiated, since the headache and abdominal pain were considered to be vasomotor phenomena. Clarithromycin (10 mg/kg/day) was added to treat atypical pathogens. Further supplementary tests were also performed, and these too were negative: (a) blood tests for *Chlamydophila pneumoniae*, *Chlamydophila psittaci*, *Chlamydia trachomatis*, *Coxiella burnetii*, influenza A virus and influenza virus; (b) polymerase chain reaction (PCR) for adenovirus, *Mycoplasma pneumoniae* and respiratory syncytial virus; (c) *Francisella tularensis* antibodies, *Legionella pneumophila* serogroup 1, immunoglobin M (IgM) for *Legionella pneumophila* serogroup 1 and cytomegalovirus; (d) stool culture for adenovirus, rotavirus and faecal parasites; (e) hidden blood in stool, faecal calprotectin; (f) echocardiogram; (g) cranial computed tomography (CT) (normal), and (h) chest and abdomen CT (pleural effusion and consolidation without associated masses).

Sixth day of hospitalisation: the effusion was drained, yielding 820 mL of exuded pleural fluid. An adenosine deaminase not indicative of tuberculosis was detected, the pneumococcal capsular antigen assay (BinaxNOW®) was negative, with no microorganisms in the Gram stain and negative Zielh-Neelsen stain. The pleural fluid analysis also included a culture of mycobacteria, fungi and bacteria, PCR for mycobacteria and an anatomical pathology examination.

At the same time as the pleural drainage, suction and biopsy of bone marrow were carried out together with flexible fibre optic bronchoscopy and bronchoalveolar lavage. In the bone marrow analysis a culture of mycobacteria, fungi and bacteria was requested together with PCR for cytomegalovirus and Epstein–Barr virus, in addition to JAK II and BCR-ABL hybrid genetic testing. The bronchoalveolar lavage analysis included an anatomical pathology study and a culture of mycobacteria, fungi and bacteria, immune reconstitution, PCR for enterovirus, rhinovirus, coronavirus, parainfluenza virus and influenza A, B and C viruses. All these tests were normal or negative.

Seventh and eighth days of hospitalisation: the pleural effusion, the fever and the abdominal pain decreased and the laboratory values normalised.

Ninth day of hospitalisation: in the laboratory tests performed before removing the drainage an increased platelet count was confirmed (7,283,000/mm^3). It was decided to carry out a platelet apheresis to avoid a possible thrombosis. The right femoral vein was cannulated for this purpose and the platelet count was successfully reduced (2,252,000/mm^3).