

CASE REPORT

Ceftriaxone induced thrombocytopenia in a child

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Abstract: A variety of diseases, hereditary conditions, toxins and drugs may cause thrombocytopenia. Thrombocytopenia induced by ceftriaxone has been rarely reported. In this case, ceftriaxone-induced thrombocytopenia is presented in a 2-year-old girl due to rare presentation (Ref. 10). Full Text in free PDF www.bmj.sk. Key words: ceftriaxone, thrombocytopenia.

Many medications can induce antibodies that cause thrombocytopenia. Platelet reactive drug-dependent antibodies (DDAbs) against quinine/quinidine, sulfonamide antibiotics, rifampicin, or ranitidine have been well characterized (1–4). Ceftriaxone is a third-generation cephalosporin that has been rarely reported to cause drug-induced immune thrombocytopenia (DITP) in a few cases (5, 6). Cephalosporins are known to cause severe drug-induced hemolytic anemia (DIHA). Most frequently, the second-generation cephalosporin cefotetan and the third-generation cephalosporin ceftriaxone are involved (7). DITP is less frequent but has also been observed with second-generation and with third-generation cephalosporins (8–10). We report a girl who developed thrombocytopenia during ceftriaxone therapy, which resolved promptly after the cessation of therapy.

Case report

A 2-year-old girl was referred to our hospital due to thrombocytopenia for further evaluation. It was learned that she was given intramuscular ceftriaxone (50 mg/kg/once – a day) for 6 days because of bronchopneumonia before admission to our hospital. On the 7th day of therapy, thrombocytopenia (76,000/mm³) was noticed. The personal and family history were unremarkable. Physical examination was normal. Laboratory studies revealed hemoglobin 12.4 g/dL, white blood cell count 5,000/mm³, platelet count 6,000/mm³ and normal renal and liver functions. Ceftriaxone was immediately withdrawn. Three days after the cessation of ceftriaxone, thrombocyte count was measured as 225,000/mm³.

Discussion

The patients are sensitized by prior exposure to the drug and experience acute cell destruction either upon re-exposure or af-

ter continuous drug administration for at least 1 week (6). Previously, a few cases of thrombocytopenia apparently induced by ceftriaxone were reported in literature (5, 6). The mechanism of drug-induced thrombocytopenia is one of immunological platelet destruction. DDAbs bind to neoantigens on PLTs via their Fab fragments and most frequently recognize epitopes on the glycoprotein (GP) complexes Ib/IX/V and/or IIb/IIIa (6). Grossjohann et al (6) described two patients with ceftriaxone induced thrombocytopenia. The responsible DDAbs reacted with epitopes residing on GPIIb/IIIa and on GPIX. DDAbs from all patients showed individual reaction patterns and clear cell lineage specificity. In addition, the DDAbs were dependent on the substitution at position 3 of the ceftriaxone molecule. Epitopes on GPIIb/IIIa and GPIX were involved. The antibodies will then attach to the drug-platelet complex and cause platelet destruction through complement activation (6).

In our case, ceftriaxone-dependent antiplatelet antibody could not be demonstrated due to lack of laboratory facilities. Nevertheless, drug induced thrombocytopenia was diagnosed on clinical and laboratory findings.

In conclusion, we would like emphasize that thrombocytopenia may be seen during ceftriaxone therapy. Therefore, clinicians should be aware of ceftriaxone-induced thrombocytopenia in children.

References

1. Chong BHX, Berndt MC, Horn S, et al. Characterization of the binding domains on platelet glycoproteins Ib-IX and IIb/IIIa complexes for the quinine/quinidine-dependent antibodies. *Blood* 1991; 77: 2190–2199.
2. Curtis BR, McFarland JG, Wu GG, et al. Antibodies in sulfonamide-induced immune thrombocytopenia recognize calcium-dependent epitopes on the glycoprotein IIb/IIIa complex. *Blood* 1994; 84: 176–183.
3. Burgess JK, Lopez JA, Gaudry LE, et al. Rifampicin-dependent antibodies bind a similar or identical epitope to glycoprotein IX-specific quinine-dependent antibodies. *Blood* 2000; 95: 1988–1992.
4. Gentilini G, Curtis BR, Aster RH. An antibody from a patient with ranitidine-induced thrombocytopenia recognizes a site on glycoprotein IX that is a favored target for drug-induced antibodies. *Blood* 1998; 92: 2359–2365.

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5. **López-Gómez M, Arrebola Nacle JP, López-Ruz MA, et al.** Ceftriaxone-induced thrombocytopenia. *Rev Clin Esp* 2004; 204: 441–442.
6. **Grossjohann B, Eichler P, Greinacher A, et al.** Ceftriaxone causes drug-induced immune thrombocytopenia and hemolytic anemia: characterization of targets on platelets and red blood cells. *Transfusion* 2004; 44: 1033–1040.
7. **Arndt PA, Garratty G.** Cross-reactivity of cefotetan and ceftriaxone antibodies, associated with hemolytic anemia, with other: cephalosporins and penicillin. *Am J Clin Pathol* 2002; 118: 256–262.
8. **Hull RL, Brandon D.** Thrombocytopenia possibly caused by structurally related third-generation cephalosporins. *DICP* 1991; 25: 135–136.
9. **Christie DJ, Lennon SS, Drew RL, et al.** Cefotetan-induced immunologic thrombocytopenia. *Br J Haematol* 1988; 70: 423–426.
10. **Aljitawi OS, Krishnan K, Curtis BR, et al.** Serologically documented loracarbef (Lorabid)-induced immune thrombocytopenia. *Am J Hematol* 2003; 73: 41–43.

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