

## Case Reports

### Autoimmune Neonatal Thrombocytopenia (AINT) Successfully Managed with Intravenous Immunoglobulins (IVIg)

Ajay Kumar  
Vikas Loiwal  
Piyush Gupta

About 30% of infants born of mothers with active idiopathic thrombocytopenic purpura (ITP) have thrombocytopenia resulting from the transplacental transfer of antiplatelet antibodies. The disease is usually self-limiting with thrombocytopenia lasting for 2-3 months(1,2). Indications for therapy include marked thrombocytopenia. With a platelet count of  $<50 \times 10^9/L$  or life threatening hemorrhage such as intracranial bleeding(3). Corticosteroid therapy has yielded equivocal results. Exchange transfusion or platelet administration may be only of temporary value in arresting acute bleeding. Intravenous immunoglobulin (IVIg) has been successfully used for achieving sustained increase in platelet counts in pregnant women with ITP who were refractory to high dose corticosteroids(4). We present our experience with the use of IVIg in autoimmune neonatal

thrombocytopenia (AINT) and intracranial hemorrhage.

#### Case Report

A 28-year-old female with history of spontaneous as well as post-traumatic skin, mucosal, subconjunctival and gum bleeds and increased menstrual loss for last six years delivered a 2.0 kg male baby by normal vaginal route after 32 weeks of gestation. At the time of delivery, the mother had ecchymotic rashes all over body, with intact spleen and platelet count of  $46 \times 10^9/L$ . During antenatal period, she had a Hb of 12.3 g/dl, RBC count of  $3.82 \times 10^{12}/L$ , MCV of 113 fl, MCH of 32.2 pg, MCHC of 28.5 g/dl, TLC of  $10.5 \times 10^7/L$ , DLC(%) -  $P_{77}L_{18}M_{1}E_4$ , and ESR of 46 mm fall in 1st hour. Peripheral smear revealed mild degree of hypochromia and anisocytosis with decreased platelets. Absolute platelet count was  $19 \times 10^9/L$ . PT and PTTK were normal. Bleeding time was more than 15 minutes with a normal clotting time. Platelet antibodies tested positive against compatible platelets. LE cell and ANF were negative. A diagnosis of autoimmune thrombocytopenic purpura (ATP) was made and she received prednisolone 60 mg per day for almost one month prior to delivery.

The child was born vaginally without any complications and cried immediately. A small cephalhematoma over the right parietal bone was immediately noticed. After 2 hours, baby developed petechial bleeds over back. The spots gradually progressed to palms, thighs and legs over next 6 h. There was no hepatosplenomegaly. At 10 hours of age he became sluggish and developed shrill cry. After another four hours, multifocal clonic seizures were observed. Blood sugar and serum calcium were

*From the Department of Pediatrics, University College of Medical Sciences and Guru Teg Bahadur Hospital, Delhi 110 095.*

*Reprint requests: Dr. Ajay Kumar, 13, S.R. Hostel, G.T.B. Hospital Campus, Delhi 110 095.*

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normal. CSF was uniformly hemorrhagic. There was associated hypoglycemia.

Investigations done at the time of birth revealed: Hb-21 g/dl, TLC- $14.1 \times 10^9/L$ , DLC(%)  $P_{36}L_{60}M_2E_2$ , Platelet count- $20 \times 10^9/L$ , RBC count- $4.12 \times 10^{12}/L$ , MCV-99.2 fl, MCH-34.4 pg, MCHC-34.6 g/dl, PT-14 sec (control 14 sec) and PTTK-50 sec (control 38 sec). Cultures of CSF and blood were sterile. Fundus revealed no abnormality. Ultrasonography of skull or a CT scan could not be done at this time. Platelet counts on 3rd, 7th and 30th day of life were 50,162 and  $263 \times 10^9/L$ , respectively.

As soon as the thrombocytopenia was documented (*i.e.*, after 2 h of birth), therapy with IVIg was planned. During the time period, when the drug was being procured, 2 mg/kg of prednisolone was given through a feeding tube. IVIg therapy could be instituted only after 8 hours of birth. Intravenous immunoglobulins were given in a dose of 400 mg/kg/day for the first five days of life. No seizures were noticed after first 36 hours of life. The general condition gradually improved and activity, cry returned to normal by 4th day of starting therapy. No fresh bleeding spots appeared and an ultrasound of the skull done at day seven of life did not reveal any abnormality. The child was discharged after four weeks of stay in the hospital. Neurological examination done before discharge was found to be normal.

### Discussion

The diagnosis of AINT, suspected on the basis of the clinical state, is confirmed either by the presence of free antiplatelet antibody or measurement of platelet associated IgG (PAIgG) in the mother's serum(5). In the present case, diagnosis of autoimmune neonatal thrombocytopenia was based on the finding of thrombocytopenia during the first 72 hours of life, evidence of

maternal ATP and absence of an alternate cause for the neonatal thrombocytopenic state. The other possible causes of thrombocytopenia in mother like pregnancy induced hypertension and lupus erythematosus were excluded by appropriate clinical as well as laboratory data.

The neonate presented with classical skin bleeds without any reticuloendothelial involvement. Imaging studies were not possible to document the existence of an intracranial bleed. The clinical features, in the absence of a metabolic or septic setting, pointed towards an intracranial bleed. A normal ultrasound of the head after a week suggested that the bleed was predominantly subarachnoid. A low platelet count of  $20 \times 10^9/L$  prompted us to use IVIg early in the disease. A rapidly progressive course, punctuated with intracranial bleed, justified this decision. The response was gratifying and sustained. However, there was some unavoidable delay in instituting this therapy. An earlier therapy might have prevented the development of intracranial bleed. In such cases therapy should be directed by the levels of platelet count ( $<50,000/mm^3$ ) rather than waiting for an intracranial bleed to occur(3).

Limited information is available concerning the use of IVIg in AINT. Ballin *et al.*(6) used IVIg for AINT in 11 newborns. Six of them had cutaneous bleeds and platelet counts on first day of life ranged from 5 to  $74 \times 10^9/L$  (median:  $25 \times 10^9/L$ ). Immunoglobulin was given in a dose of 1g/kg/day for 2 days in 10 children and 0.4 g/kg/day for 5 days in one infant. In this study, concomitant steroids were also used. Platelet count increased to more than  $50 \times 10^9/L$  in 75% of total patients. Results of the above study and the present report might indicate a useful place of IVIg therapy in the management of AINT.

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