importantly, prior administration of antibiotics does not interfere with the test. This simple and quick test has a high degree of sensitivity and specificity and good likelihood ratios.

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# HIV Related Immune Thrombocytopenia

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Till October 1990, Human Immunodeficiency Virus (HIV) has been reported in 4,082 individuals out of 5,80,824 tested in India(l). To-date over 700 patients with HIV-related thrombocytopenia have been reported in the world literature(2).

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The need to create an awareness of tilling out HIV infection in patients presenting with thrombocytopenia has prompted us to document this case and discuss the therapeutic options.

## Case Report

A 6-year-old boy presented with a history of easy bruisability for 2 months which had increased over a period of 8 days. He had 2 episodes of epistaxis. He had received a blood transfusion at a private hospital 3 years ago for anemia. Eight months prior to this presentation, the child had received rifampicin and isoniazid for 6 months for primary complex. On examination, the child had ecchymotic patches over both the lower extremities and on the left cheek. Systemic examination was normal. Investigations showed hemoglobin 10.8 g/dl, total leucocyte count 6,800 per cu mm with 45% polymorphs and 55% lymphocytes. The platelet count was 52,000 per cu mm with a normocellular bone marrow (M : E : : 3 : 1). The megakaryocytes though structurally normal' were increased in number. The antiplatelet antibody titre was 1:32 by the immunofluoescence techniques. Micro - ELISA (Wellcozyme HIV recombinant UK 56/57) for

HIV antibodies was positive on two occasions and Western blot (EPIBLOT - HIV Western Blot Kit) was also positive. Mother's ELISA was negative. The patient's immunoglobin levels and CD4/CD8 ratio were normal *(Table I)*. Oral corticosteroids were started a week later as the platelet count dropped to 35,000 per cu mm. Repeat platelet count after 1 month of corticosteroid therapy was 72,000 per cu mm and steroids were then gradually tapered over a period of 2 months. Apart from intermittent spontaneous ecchymotic patches there was no clinical deterioration. On a regular follow up the platelet count, however, continued to remain in the range of 50,000 to 100,000 per cu mm.

**Discussion**

AIDS in children has increased the incidence of immune thrombocytopenia(2). The overall incidence of thrombocytopenic purpura in HIV infection has been reported to be 13% and 30% in 2 different studies(3,4).

Although various hemostatic abnormalities have been described in HIV infection, many questions have been raised regarding the pathogenesis and treatment of HIV-related thrombocytopenia. When thrombocytopenia occurs in an otherwise HIV infected adult, it is considered to be a diagnostic criterion for the AIDS-related complex. By the CDC classification isolated thrombocytopenia in an HIV infection child would be classified as P-2F(5).

Although the exact mechanism of thrombocytopenia in HIF infections is ill defined, it appears to be heterogenous. Autoantibody may play a prominent role in mucosally infected patients, whereas, immune complexes may be more important in parenterally acquired disease(2). The presence of anti-IgG containing immune complexes on the platelet surface may explain many of the immunologic features of this form of thrombocytopenia. This could explain the paradox of a positive titre of antiplatelet antibodies in immunocompromized patients as seen in our case.

The optimal treatment of HIF associated thrombocytopenia remains uncertain. The initial responses with various therapeutic options like zidovudine, intravenous gammaglobulin (IVIG), prednisolone and

**TABLE I**–*Values of Immunoglobulin Levels and T Cell Phenotyping*

| | Test | % | Absolute counts | Normal |
|---|---|---|---|---|
| T-cell phenotyping | $CD_3$ | 48.5 | 1814 | 45-65 |
| | $CD_4$ | 32 | 1197 | - |
| | $CD_8$ | 22 | 823 | - |
| | Ratio $Cd_4/CD_8$ | | 1.45 | > 1 |
| Immunoglobin level | IgG | | 948 | 500-1663 |
| (mg/dl) | IgA | | 141 | 66-344 |
| | IgM | | 273 | 39-290 |

splcnectomy ranges from 30-100%(2). With dapsone, the response has been more durable lasting for a mean period of 18.4 months(6). The other therapeutic modalities tried hpve been anti-RhD, danazol, vincristine, ascorbate and alpha-interferon.

The possible mechanism of action in zidovudine is postulated to be viral clearance from the megakaryocytes, enhancement of platelet production and suppression of autoantibody formation. IVIG appears to be the most effective treatment with various regimes being tried(7,8).

Corticosteroids have been used as a salvage drug should IVIG fail. Prednisolone is a double-edged sword because of the risk of further immunosuppression. However, in children there are reports of better and sustained response with prednisolone as compared to that with IVIG (9).

In our patient, since zidovudine and IVIG could not be given because of the cost, we used steroids. Splenectomy has also shown a favorable response but an associated 25% risk of progression of AIDS has been reported(10). It is important to remember that 10-20% of HIV positive immune thrombocytopenia patients have spontaneous remission of thrombocytopenia. However normalization of the platelet count may herald the onset of AIDS. Withholding the treatment may be appropriate in the absence of bleeding even if the platelet count is less than 50,000 per cu mm.

In conclusion, it may be stated that individualization of therapy is important in HIV-related thrombocytopenia. With the increasing incidence of AIDS, an emphasis must be laid on the need for testing blood for HIV before transfusion and avoiding blood transfusion unless absolutely indicated.

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# Fatal Fulminant Hepatic Failure Due to Sodium Valproate in an Adolescent

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Since its introduction in 1973, sodium valproate (VPA) has been used extensively due to its efficacy against a wide range of seizure disorders. It appears to be well tolerated at therapeutic doses. Severe hepatotoxicity, resulting in death, is extremely rare(l-3). Till date all reports of fatal hepatotoxicity have come from Europe and North America(l-3). We report one such case involving an Indian adolescent.

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## Case Report

A 14-year-old boy with past history of seizure disorder was referred to us, for jaundice and encephalopathy in March 1993. In January 1993 he was put on valproate (600 mg/day) and phenobarbitone (60 mg/day) following a second episode of right focal seizure with secondary generalization. There was no recurrence of seizures while he was on this therapy for the next 10 weeks. In March he developed moderate grade fever an3 vomiting. Two days later he developed altered sensorium and lapsed into coma. Jaundice was also noted at this stage and he was shifted to this hospital. Both anticonvulsants were stopped after the onset of jaundice. Examination revealed him to be deeply comatose. Vital signs were normal except for tachycardia (120/min). Icterus was present; stigmata of chronic liver disease were absent. Liver span was 12 cm. There was no splenomegaly or free fluid in the abdomen. Pupillary size was 4 mm bilaterally, oculocephalic reflexes were elicitable. All deep tendon reflexes were exaggerated. Planters showed bilateral extensor response.

Investigations were as follows: hemoglobin 12.2/dl, TLC 12,200/ul polymorphs 72%, lymphocytes 21%), blood sugar 103 mg/dl, sodium 125 mEq/L, potassium 3.6 mEq/L, bilirubin 19.6 mg/dl (conjugated