Clinical Presentations of Idiopathic Thrombocytopenic Purpura

Abstract: Back Ground: ITP is the most common cause of thrombocytopenia in children and is a result of immunologically mediated increased platelet destruction, which is usually acute and self-limiting but may be a recurrent or chronic disorder. Aim of Study: Assessment of clinical manifestation of ITP in children. Patient and Methods: A prospective study was performed on 60 patients diagnosed as idiopathic thrombocytopenic purpura admitted to central child teaching hospital from 1st of January to 30 September 2011. Diagnosis was based on clinical and laboratory tests including bone marrow aspiration. Results: The age ranged from 6 months to 13 years and majority of cases were between 1-5 years, 68.3 % were males & 31.7 % were females, male to female ratio 2.1 : 1. History of upper respiratory tract infection preceded the onset of illness in 71.7 % of patients. Where the Duration between Preceding URTI & ITP Up to 1 week in 30.2 % and > 1 week in 79.8 % Concerning the distribution of cases according to the season of the year: One case (1.7%) found during January, four cases (6.7%) during February, six cases (10.0%) during March, five cases (8.3%) during April, ten cases (16.7%) during May, eight cases (13.3%) during June, 14 cases (23.3%) during July, five cases (8.3%) during August, and seven cases (11.7%) during September. This distribution was significant (P<0.05). The commonest presentation was cutaneous bleeding in 60 %, Mucous Membrane bleeding 1.7 %, both 38.3 %, Splenomegaly with bleeding 6.7 %, Hepatomegaly with bleeding 8.3 %, Lymphadeno-pathy with bleeding 5 %. In 69 % of cases the initial platelet count was less than 20,000 /mm³, 37.9 % had anemia. Bone marrow examination done in 56 case and all were normal. In this study, one case die due to intracranial hemo – rrhage. we didn’t include the management because the patients receive different types of treatments (mostly by steroid, some by IVIG & anti-D), most of patients examined by bone marrow aspiration which was normal, and also we didn’t include the follow up because of most of the patients didn’t come for follow up. Conclusion: The disease is self-limited, most cases at age of 1-5 years, male affected more than female, most cases preceded by upper respiratory tract infections, most cases presented by skin bleeding.

Keywords: ITP, DISEASE, BLEEDING.

<table>
<thead>
<tr>
<th>CBC</th>
<th>Complete blood count</th>
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<tbody>
<tr>
<td>HIV</td>
<td>Human immunodeficiency virus</td>
</tr>
<tr>
<td>ITP</td>
<td>Idiopathic Thrombocytopenic Purpura</td>
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<tr>
<td>IVIG</td>
<td>Intravenous Immunoglobulin</td>
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<td>MPV</td>
<td>Mean Platelet Volume</td>
</tr>
<tr>
<td>SLE</td>
<td>Systemic Lupus Erythematosus</td>
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<td>URTI</td>
<td>Upper Respiratory Infection</td>
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INTRODUCTION:

Immune (Idiopathic) Thrombocytopenic Purpura

Immune thrombocytopenic purpura (ITP) is a syndrome characterized by:

1. Thrombocytopenia (platelet count less than 150,000/mm3)
2. Shortened platelet survival
3. Presence of antiplatelet antibody in the plasma
4. Increased megakaryocytes in the bone marrow.

This condition may be acute, chronic, or recurrent. In the acute form, the platelet count returns to normal (>150,000/mm3) within 6 months after diagnosis. In the chronic form, the platelet count remains low beyond 6 months. In the recurrent form, the platelet count decreases after having returned to normal levels. In adults, the chronic form is more common, whereas in children, the acute form is more common (Lanzkowsky, P. (Ed.). 2005). In acute ITP, it is...
associated with purpura, petechiae, mucocutaneous bleeding and occasionally hemorrhage into internal tissues (Kliegman, R. M. et al., 2007). In 70–80% of children who present with acute ITP, spontaneous resolution occurs within 6 mo. Approximately 20% of children who present with acute ITP go on to have chronic ITP (Kliegman, R. M. et al., 2007).

Etiology and pathogenesis:-

The most frequent cause of ITP is immune mediated platelet destruction due to auto antibodies: A recent history of viral illness is described in 50–65% of cases of childhood ITP. The reason why some children respond to a common infection with an autoimmune disease remain unknown (Lanzkowsky, P. (Ed.). 2005; Kliegman, R. M. et al., 2007). Most common infectious viruses have been described in association with ITP, including rubella, varicella, measles, parvovirus, influenza, EBV and HIV (Imbach, P. et al., 2008). Other infections that might cause ITP are: Helicobacter pylori, Cytomegalovirus, and hepatitis C virus (Cines, D. B., & Blanchette, V. S. 2002; Cines, D. B. et al., 2009). Acute ITP had been also observed following vaccination in children notably; measles-mumps-rubella vaccine (MMR), Pneumococcus, Haemophilus influenzae b, hepatitis B vaccine and varicella zoster vaccine (France, E. K. et al., 2008; Jadavji, T. A. J. et al., 2003). After binding of the antibody to the platelet surface, circulating antibody-coated platelets are recognized by the Fc receptor on the splenic macrophages, ingested, and destroyed (Kliegman, R. M. et al., 2007). In the circulation platelet survival is markedly decreased, splenic sequestration accounts for the shortened survival in most patients, but the liver, and even the reticuloendothelial cells of the bone marrow, can play a major role in the sequestration of antibody-coated platelets, especially in patients with very low platelet counts or continued thrombocytopenia after splenectomy. The spleen has also been implicated as a site of antibody formation (Levine, S. P. 2003).

Incidence:– The true incidence of ITP is unknown because the disease is often transient. The estimated incidence is about 1 in 10,000 children per year (Lanzkowsky, P. (Ed.). 2005; Craig, J. I. O. et al., 2006).

CLINICAL MANIFESTATIONS. The classic presentation of ITP is that of a previously healthy 1–4 yr old child who has sudden onset of generalized petechiae and purpura. There is a history of a preceding viral infection 1–4 wk before the onset of thrombocytopenia. Often there is bleeding from the gums and mucous membranes, particularly with profound thrombocytopenia (platelet count <10 × 10^9/L) (Kliegman, R. M. et al., 2007; Jonas, M. M. et al., 2003; & Paul, S. J. 2002). Bleeding from gastrointestinal tract, or kidneys is not uncommon. Hematemesis and melena are infrequent (Lanzkowsky, P. (Ed.). 2005). Findings on physical examination are normal, other than the finding of petechiae and purpura. Splenomegaly is rare, as is lymphadenopathy or pallor. An easy to use classification system has been proposed from the U.K. to characterize the severity of bleeding in ITP on the basis of symptoms and signs, but not platelet count:

1. No symptoms
2. Mild symptoms: bruising and petechiae, occasional minor epistaxis, very little interference with daily living
3. Moderate: more severe skin and mucosal lesions, more troublesome epistaxis and menorrhagia
4. Severe: bleeding episodes—menorrhagia, epistaxis, melena—requiring transfusion or hospitalization, symptoms interfering seriously with the quality of life.

The presence of abnormal findings, such as hepatosplenomegaly or remarkable lymphadenopathy, suggests other diagnoses (leukemia) (Kliegman, R. M. et al., 2007; Jonas, M. M. et al., 2003; & Bussel, J., & Cines, D. 2000). Fewer than 1% of patients have intracranial hemorrhage.

LABORATORY FINDINGS
1. Platelet count:
   a. Always less than 150,000/mm3.
   b. Often less than 20,000/mm3 in patients with severe generalized hemorrhagic manifestations.
   c. MPV increased (on automated counting equipment); normal MPV,8.9±1.5 \( \mu \)m3

2. Blood smear:
   a. Blood smear normal, apart from thrombocytopenia (if active infection, increased neutrophils, lymphocytes, or atypical mononuclear cells may be present).
   b. Anemia present only in proportion to amount of blood loss.
3. Bone marrow:
   a. Increased megakaryocytes, often immature and with absence of budding
   b. Normal erythroid and myeloid cells
   c. Increased eosinophils occasionally seen
   d. Erythroid hyperplasia if significant blood loss.

   (Marrow findings are diagnostic of ITP only when consistent with the clinical state and presence of a low platelet count and when other causes of secondary thrombocytopenia are excluded. The main purpose of performing a bone marrow examination is to exclude other hematologic disorders, e.g., leukemia.)

4. Coagulation profile:
   a. Bleeding time—usually abnormal
   b. Prothrombin time (PT), activated partial thromboplastin time (APTT), and fibrinogen level—normal (Lanzkowsky, P. (Ed.). 2005).

   Other laboratory tests should be done as indicated by the history and physical examination. In adolescents with new-onset ITP, an antinuclear antibody test should be done to evaluate for SLE. A Coombs test should be done if there is unexplained anemia to rule out Evans syndrome (autoimmune hemolytic anemia and thrombocytopenia) or before instituting therapy with IV anti-D (Kliegman, R. M. et al., 2007; Paul, S. J. 2002).

DIFFERENTIAL DIAGNOSIS. The well-appearing child with moderate to severe thrombocytopenia, an otherwise normal complete blood cell count (CBC), and normal findings on physical examination has a limited differential diagnosis (Kliegman, R. M. et al., 2007).

ITP must be differentiated from:

- Exposure to some medications may give clinical picture similar to ITP (e.g., heparin, quinine, sulfa, rifampin, carbamazepin, valproic acid and H1 blocker)
- Familial non immune thrombocytopenia :
  - Wiskott-Aldrich syndrome : X-linked recessive immunodeficiency characterized by thrombocytopenia, eczema, and recurrent infections.
  - Bernard Soulier syndrome : There is a deficiency in glycoprotein Ib lead to defect in adhesion. Platelet are giant & few and do not aggregate with ristocetin
- A megakaryocytic thrombocytopenia

These disorders are suspected when the patient fail to respond to therapy and by finding on bone marrow aspiration.

- Disorders that associated with ITP, like HIV infection, SLE, humoral immunodeficiency, especially when there is atypical presentation.

TREATMENT: Acute ITP in childhood is usually a self–limiting condition. Medical treatment does not seem to alter the natural history of the disease. Treatment may be required to minimize the danger of life–threatening intracranial hemorrhage, a more rapid rise in platelet count to the theoretically safe level of >20 × 10^9/L, although there are no data indicating that early therapy prevents intracranial hemorrhage. Antiplatelet antibodies bind to transfused platelets as well as they do to autologous platelets. Thus, platelet transfusion in ITP is usually contraindicated unless life-threatening bleeding is present (Kliegman, R. M. et al., 2007; Jonas, M. M. et al., 2003; & Paul, S. J. 2002). Treatment is indicated for children with platelet counts less than 20,000/mm3 and significant mucous membrane bleeding, and those with platelet counts less than 10,000/mm3 and minor purpura (Lanzkowsky, P. (Ed.). 2005).

General Measures:
The general management of children with ITP includes:

- Avoiding aspirin and other drugs that can impair platelet function.
- Avoiding intramuscular injection.
- If the platelet count is very low, children should avoid stimulus which carries significant risks of severe trauma (Tarantino, M. D. 2002).

Initial approaches to the management of ITP include the following:

- **No therapy** other than education and counseling of the family and patient for patients with minimal, mild, and moderate symptoms, as defined earlier. This approach emphasizes the usually benign nature of ITP and avoids the therapeutic roller coaster that ensues once interventional therapy is begun. This approach is far less costly, and side effects are minimal (Kliegman, R. M. et al., 2007).
2. **Intravenous immunoglobulin (IVIG).** IVIG at a dose of 0.8–1.0 g/kg/day for 1–2 days induces a rapid rise in platelet count (usually>20x 10^9/L) in 95% of patients within 48 hr. IVIG appears to induce a response by down regulating Fc-mediated phagocytosis of antibody-coated platelets. IVIG therapy is both expensive and time-consuming to administer. Additionally, after infusion, there is a high frequency of headaches and vomiting, suggestive of IVIG-induced aseptic meningitis (Kliegman, R. M. *et al*., 2007).

**IVGG toxicity:**

a. **Anaphylaxis** in IgA-deficient patients because of preexisting IgA antibodies that react with small amounts of IgA present in commercially available gamma globulin.

b. **Post infusion headache** in 20% of patients; transient and possibly severe (in severe cases, administer postinfusion dexamethasone 0.15–0.3 mg/kg IV).

Severe headache in ITP may suggest the presence of intracranial hemorrhage and if clinically indicated may require a CT scan.

c. **Fever and chills** in 1–3% of patients; prophylactic acetaminophen (10–15 mg/kg, 4 hourly, as required) and diphenhydramine (1 mg/kg, 6–8 hourly, as required) to reduce the incidence and severity.

d. **Coombs-positive hemolytic anemia** because of the presence of blood group antibodies (anti-A, anti-B, and anti-D) present in IVGG.

e. **Hepatitis C virus (HCV)** infection reported in patients receiving IVGG; however, no reports of HIV infection with any licensed preparation in the United States has been described (Lanzkowsky, P. (Ed.). 2005).

3. **Intravenous anti-D therapy.** For Rh positive patients, IV anti-D at a dose of 50–75µg/kg causes a rise in platelet count to>20x 10^9/L in 80–90% of patients within 48–72 hr. When given to Rh positive individuals, IV anti-D induces mild hemolytic anemia. RBC-antibody complexes bind to macrophage Fc receptors and interfere with platelet destruction, thereby causing a rise in platelet count. IV anti-D is ineffective in Rh negative patients.

**Adverse drug reactions**

**Fever**

**Chills**

**Headache**

Drop in hemoglobin and hematocrit attributed to anti-D-related hemolysis. These symptoms are directly attributable to anti-D-related hemolysis and a positive Coombs reaction (Lanzkowsky, P. (Ed.). 2005; Kliegman, R. M. *et al*., 2007; Tarantino, M. D. 2002; & Gaines, A. R. 2000).

4. **Prednisone.** Corticosteroid therapy has been used for many years to treat acute and chronic ITP in adults and children. Doses of prednisone of 1–4 mg/kg/24 hr appear to induce a more rapid rise in platelet count than in untreated patients with ITP. Whether bone marrow examination should be performed to rule out other causes of thrombocytopenia, especially acute lymphoblastic leukemia, before institution of prednisone therapy in acute ITP is controversial (Kliegman, R. M. *et al*., 2007).

The mechanism of action of steroids in ITP is multifactorial; by inhibiting phagocytosis of antibody coated platelets in the spleen and prolonging platelet survival, by improving capillary resistance, and inhibiting platelet antibody production. A course of prednisone, 2 mg /kg/day (maximum 60 mg/ day), is given in divided doses. Prednisone is reduced in stepwise fashion at 5 to 7 days intervals irrespective of the platelet count and is stopped at the end of 21–28 days, regardless of the response. A shorter course of prednisone at 4 mg/ kg /day for 4 days has also been used with success (Cines, D. B., & Blanchette, V. S. 2002). In severe cases, methylprednisolone (Solu-medrol) 30 mg/ kg/day, (maximum1g /day) for 3 days produces a more rapid response than steroids in conventional doses (Lanzkowsky, P. (Ed.). 2005). Prolonged use of steroids in ITP is undesirable. Large doses or prolonged steroid usage may perpetuate the thrombocytopenia and depress platelet production. It also leads to side effects including weight gain, cushingoid facies, fluid retention, acne, hyperglycemia, hypertension, mood swings, pseudotumor cerebri, cataracts, growth retardation, and avascular necrosis (Lanzkowsky, P. (Ed.). 2005). Each of these medications may be used to treat exacerbations of ITP, which commonly occur several wk after an initial course of therapy. In the special case of intracranial hemorrhage, multiple modalities should be used, including platelet transfusion, IVIG, high-dose corticosteroids, and prompt surgical consultation, with plans for emergency splenectomy (Kliegman, R. M. *et al*., 2007).

5. **Splenectomy.** Splenectomy should be reserved for patients with acute life threatening bleeding, and those whose symptoms lasted more than one year (chronic ITP) and older than 4 years. Its effective in controlling thrombocytopenia in 64 – 88 % of cases (Kliegman, R. M. *et al*., 2007). Splenectomy is associated with lifelong risk of overwhelming sepsis by encapsulated organisms. Before splenectomy corticosteroids, IVIG, or anti-D given to raise platelet count (Lanzkowsky, P. (Ed.). 2005). Presplenectomy immunization with meningococcal, pneumococcal and haemophilus influenza type b vaccines and subsequent penicillin prophylaxis are necessary for
all age groups (Cines, D. B. & Blanchette, V. S. 2002). When possible, surgery should be performed using laparoscopic technique (Kahn, M. J., & McCrae, K. R. 2004).

Other modalities of treatment are uncommonly used, and tried only after failure of response to immunoglobulin, steroids, or splenectomy, these include (Lanzkowsky, P. (Ed.). 2005; Levine, S. P. 2003; & Bennet, C. M. et al., 2006):
- Imunosuppressive therapy like: danazol, vincristine, vinblastine, cyclosporine, cyclophosphamide and azathioprine.
- Rituximab.
- Plasmapheresis
- Platelet transfusion.

Management of life threatening hemorrhage:
- Platelet transfusion
- Methylprednisolone 500 mg/m² IV per day for 3 days
- Intravenous immunoglobulin 2g/kg.
- Emergency splenectomy.

These measures can be used singly or in combination, depending on the severity and response to treatment. Patients refractory to these measures may benefit from vincristine sulfate (1.5 mg/ m² IV maximum 2 mg) and plasmapheresis (Lanzkowsky, P. (Ed.). 2005).

Prognosis
- Excellent; 50% recover usually within 1 month and 70–80% recover within 6 months.
- Spontaneous remission after 1 year is uncommon, although may occur even after several years.
- Age older than 10 years, insidious onset, and female gender are associated with the development of chronic ITP.
- Of all chronic patients, 50–60% eventually stabilize without any therapy and without need for splenectomy (Lanzkowsky, P. (Ed.). 2005).

Aims of the study
- Assessment of clinical manifestation of ITP in children.
- To enlighten the possible risk factors of acute ITP regarding to age, sex, residence, history of antecedent event, & date variation.

3. To study the laboratory finding at diagnosis.

PATIENTS AND METHODS:-
A prospective study was performed on 60 patients diagnosed as acute idiopathic thrombocytopenic purpura, who were admitted to central child teaching hospital from 1st of January to 30 September 2011. Data were collected according to preceded questionnaire from information regarding age, sex, date of admission, Residence, Rural or Urban, Antecedent event (the events like upper respiratory tract infections, fever, etc), Onset of event, Duration between antecedent event and generalized petechiae and purpura, site of bleeding, associated clinical features, lab tests which include CBC, blood smear, bone marrow exam.

The criteria of inclusion for the study group were:
- Signs and symptoms of ITP.
- The necessary laboratory investigation.

Statistical Analysis :
Statistical Package for Social Sciences version 18 (SPSS 18) was used for data input and analysis. Discrete variables presented as numbers and percentages. Continuous variables presented as mean and standard deviation (SD). Chi square test for independence used to test the significance of association between two discrete variables. t test used to test the significance of difference in mean between two independent samples. Chi square test for goodness of fit used to test the significance of observed distribution. Findings with P value less than 0.05 were considered significant.

Note: P value is the probability of error/chance (sampling error, error because of sampling). The finding is not considered significant if the probability of error more than 5% (P>0.05), here you cannot say there is no relation but say there is no significant relation because still the probability of trueness is more than 90% in some instances but the probability of chance is high (> 5%).
RESULTS
1-Age:
The sample is consisted of 60 child aged 1 to 156 months with mean age 49.2 ± 38.4 month. Dominant age group was 12-59 months (1-5years) (P<0.05, table 1, figure 1) Table 1: Demographic characteristics of study sample.

<table>
<thead>
<tr>
<th>Variables</th>
<th>N (%)</th>
<th>Chi-square</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (month); mean±SD</td>
<td>60 (100.0)</td>
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<tr>
<td>Age Group</td>
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<tr>
<td>&lt; 12 month</td>
<td>5 (8.3)</td>
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<tr>
<td>12-59 month</td>
<td>35 (58.3)</td>
<td>22.500</td>
<td>0.006</td>
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<tr>
<td>≥ 60 month</td>
<td>20 (33.3)</td>
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<td></td>
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<tr>
<td>Gender</td>
<td></td>
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</tr>
<tr>
<td>Male</td>
<td>41 (68.3)</td>
<td></td>
<td></td>
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<tr>
<td>Female</td>
<td>19 (31.7)</td>
<td>8.067</td>
<td>0.005</td>
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<td>Residence</td>
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<tr>
<td>Urban</td>
<td>34 (56.7)</td>
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<tr>
<td>Rural</td>
<td>26 (43.3)</td>
<td>1.067</td>
<td>0.302</td>
</tr>
</tbody>
</table>

N: number, %: percent, SD: standard deviation.

Figure1: Distribution of study sample according to age.

2-Sex Differences
As shown in table (1), Males were 41 (68.3%) and females 19 (31.7%). and males significantly constituted about two thirds of cases (P<0.05, table 1, figure 2)
3. Residence (Rural or Urban) Thirty four patients constitute (56.7%) of patients were from urban areas and 26 patients constitute (43.3%) from rural areas as shown in (Table 1). while the observed distribution regarding residence was not significant (P>0.05, table 1).

4. Date of admission - Concerning the distribution of cases according to the month of the year: One case (1.7%) found during January, four cases (6.7%) during February, six cases (10.0%) during March, five cases (8.3%) during April, ten cases (16.7%) during May, eight cases (13.3%) during June, 14 cases (23.3%) during July, five cases (8.3%) during August, and seven cases (11.7%) during September. This distribution was significant (P<0.05, table 2, figure 3).

Table 2: Distribution of ITP cases according to the month of the presentation

<table>
<thead>
<tr>
<th>Month of the year</th>
<th>N (%)</th>
<th>Chi-square</th>
<th>P value</th>
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</thead>
<tbody>
<tr>
<td>January</td>
<td>1 (1.7)</td>
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<tr>
<td>February</td>
<td>4 (6.7)</td>
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<td></td>
</tr>
<tr>
<td>March</td>
<td>6 (10.0)</td>
<td></td>
<td></td>
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<tr>
<td>April</td>
<td>5 (8.3)</td>
<td>16.800</td>
<td>0.032</td>
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<tr>
<td>May</td>
<td>10 (16.7)</td>
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<td></td>
</tr>
<tr>
<td>June</td>
<td>8 (13.3)</td>
<td></td>
<td></td>
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<tr>
<td>July</td>
<td>14 (23.3)</td>
<td></td>
<td></td>
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<tr>
<td>August</td>
<td>5 (8.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>September</td>
<td>7 (11.7)</td>
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<td></td>
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<tr>
<td>Total</td>
<td>60(100.0)</td>
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</table>
5. The preceding upper respiratory tract infection:
- It was significant to find 43 cases (71.7%) preceded with upper respiratory tract infection (P< 0.05, table 3).
- Duration between upper respiratory tract infections and appearance of ITP lesions was 3 days in minimum, maximum 23 days, and in average was 11.6 ± 4.8 days (table 3).
- Those preceded with URTIs of more than one week were the majority; 30 cases (79.8%). This distribution was significant (P< 0.05, table 3, figure 4).

Table 3: Relation between ITP and preceding URTI

<table>
<thead>
<tr>
<th>Variables</th>
<th>N (%)</th>
<th>Chi-square</th>
<th>P value</th>
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<tr>
<td>Preceding URTI</td>
<td>43 (71.7)</td>
<td>9.600</td>
<td>0.002</td>
</tr>
<tr>
<td>Duration between Preceding URTI &amp; ITP (days);</td>
<td></td>
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<tr>
<td>mean±SD</td>
<td>11.6 ± 4.8</td>
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</tr>
<tr>
<td>Duration between Preceding URTI &amp; ITP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Up to 1 week</td>
<td>13 (30.2)</td>
<td>6.721</td>
<td>0.010</td>
</tr>
<tr>
<td>&gt; 1 week</td>
<td>30 (79.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>43 (100.0)</td>
<td></td>
<td></td>
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</tbody>
</table>

N; number, %; percent, SD; standard deviation; URTI; upper respiratory tract infection.
6. Clinical Manifestations:
Table 4, Fig 5, show the clinical features of cases presented in our study, presenting site of bleeding is skin 60%, skin & mucous membrane 38.3%, mucous membrane only 1.7%. Splenomegaly with bleeding 6.7%, Hepatomegaly with bleeding 8.3%, Lymphadenopathy with bleeding 5%.

Table 4: Clinical findings for the study sample:

<table>
<thead>
<tr>
<th>Findings</th>
<th>N (%)</th>
<th>Chi-square</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presenting Site of ITP</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Skin</td>
<td>36 (60.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mucous Membrane</td>
<td>1 (1.7)</td>
<td>31.300</td>
<td>0.034</td>
</tr>
<tr>
<td>Both</td>
<td>23 (38.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Splenomegaly with bleeding</td>
<td>4 (6.7)</td>
<td>45.067</td>
<td>0.005</td>
</tr>
<tr>
<td>Hepatomegaly with bleeding</td>
<td>5 (8.3)</td>
<td>41.667</td>
<td>0.004</td>
</tr>
<tr>
<td>Lymphadenopathy with bleeding</td>
<td>3 (5.0)</td>
<td>48.600</td>
<td>0.004</td>
</tr>
</tbody>
</table>

N; number, %; percent.

Figure 4: Distribution of study sample according to duration between preceding URTI and onset of ITP.

Figure 5: Distribution of study sample according to site of presenting ITP lesions.
7. Lab. Findings:
Table 5, Fig. 6 & 7, show the initial lab. Finding.

<table>
<thead>
<tr>
<th>Findings</th>
<th>N (%)</th>
<th>Chi-square</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCV%: mean±SD</td>
<td>60 (100.0)</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Presence of Anemia</td>
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<td></td>
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<tr>
<td>Yes</td>
<td>22(37.9)</td>
<td>3.379</td>
<td>0.066</td>
</tr>
<tr>
<td>No</td>
<td>36(62.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>60(100.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelets (10^9/ml) : mean±SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 20</td>
<td>40(69.0)</td>
<td>39.759</td>
<td>0.005</td>
</tr>
<tr>
<td>20-50</td>
<td>17(29.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50-150</td>
<td>1(1.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>60(100.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N; number, %; percent, SD; standard deviation; URTI; upper respiratory tract infection.

![Figure 6: Distribution of study sample according to platelet level.](image)

There is one case dead due to intracranial hemorrhage noticed during this study.

BONE MARROW EXAMINATION:
Sixty five patients had bone marrow exam., All of which were normal. For patients didn’t examine the bone marrow due to family rejection.

DISCUSSION:
In our study we want to clarify the most common presenting features of ITP in attempt to identify the risk factors of this disease, but it is a descriptive study, so we didn’t include the management because the patients receive different types of treatments (mostly by steroid, some by IVIG & anti-D), most of patients examined by bone marrow aspiration which was normal, and also we didn’t include the follow up because it was poor. In this study the distribution of cases according to the age and sex of the patients; Males were 41 (68%) and females 19 (31.7%). And males significantly constituted about two thirds of cases. Which compatible with (Khashan, A. 2008) & in (Lanzkowsky, P. (Ed.). 2005), but incompatible with (Nariman, F. I. 1998), (Sawsan, S. A., 2006) & (Hussein, K. M. 2010).
In our study the dominant age group was 12-59 month (66.6 %), this result was consistent with many studies like (Al-Nadawi, M. N. et al., 2002), (Nariman, F. I. 1998), (Hussein, K. M. 2010), (Zeller, B. et al., 2000), (Jacobs, P. et al., 2003). History of preceding infection usually viral upper respiratory infection is noted in 71.7 % that’s similar to a finding by (Khashan, A. 2008), (Hussein, K. M. 2010), (Nariman, F. I. 1998), & (Al-Nadawi, M. N. et al., 2002). The distribution of cases according to the duration of the disease between the antecedent event and generalized petechiae and purpura in which the highest duration noticed was more than 1 wk in which 79.8 % of cases, while the least patient recorded of duration less than 1 wk 30.2%, which is against what reported by (Khashan, A. 2008), & (Erduran, E. et al., 2003). The distribution of cases according to the month of the year: One case (1.7%) found during January, four cases (6.7%) during February, six cases (10.0%) during March, five cases (8.3%) during April, ten cases (16.7%) during May, eight cases (13.3%) during June, 14 cases (23.3%) during July, five cases (8.3%) during August, and seven cases (11.7%) during September. The largest number in July & May, may be due to increased cases of viral infections. The distribution of cases according to the residence: Where 34 (56.7%) four patients constitute (56.7%) of patients were from urban areas and 26 patients constitute (43.3%) from rural areas. While the observed distribution regarding residence was not significant (P>0.05). The presenting signs & symptoms were skin bleeding (petechiae & purpura) 60 % which is the highest, Mucous Membrane (epistaxis & gum bleeding) 1.7 % , both 38.3 %, the least presentations were Splenomegaly with bleeding 6.7 %, Hepatomegaly with bleeding 8.3 %, Lymphadenopathy with bleeding 5 %, these abnormal findings may be due to the causative agent like viral disease. All of these were consistent with (Khashan, A. 2008), (Hussein, K. M. 2010), (Nariman, F. I. 1998), & (Al-Nadawi, M. N. et al., 2002). Majority of our cases had initial platelet count < 20.000/mm³ (69% of cases) from (Hussein, K. M. 2010), (Nariman, F. I. 1998), & (Al-Nadawi, M. N. et al., 2002). where’s anemia present in 37.9 % which may be due to bleeding or nutritional anemia

CONCLUSION
1. The majority of cases of ITP and children had mild bleeding symptoms, and self-limited.
2. The most affected patients were between 1-5 yr age group.
3. Male affected more than female.
4. Most of cases preceded by upper respiratory tract infection or by fever.
5. The most presenting sign was skin bleeding.
6. Bone marrow examination was done routinely in most cases and the results were normal.

Recommendations
1. The disease should be suspected in the risky age group of 1-5 yr, who presented with petechia or mucous membrane bleeding.
2. The disease is a self-limited, and should wait to know the platelets count and the full blood count to decide the treatment.
3. Routine bone marrow examination is not necessary in the typical cases but should be done in all atypical cases.

REFERENCES:


