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The Mystery Bug: Helicobacter Pylori. Effect of Eradication on Platelet Counts in Patients with Immune Thrombocytopenic Purpura

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Abstract: ***Aim and Objectives: 1. To determine the prevalence of H.pylori infection in patients diagnosed to have Immune Thrombocytopenic Purpura. 2. To assess the effect of eradication of H.pylori infection on the platelet count of patients with ITP 3. To identify and evaluate other factors that may influence the changes in platelet counts of the studied patient group. Setting and Design: This study was Prospective Interventional study conducted at (JIPMER), Puducherry, India. Methods and Materials: All ITP patients who attended the haematology clinic were considered for study. Baseline blood investigations and endoscopic antral biopsy was done for all the patients. Diagnosis of H.pylori was done by urease test or histologically by Giemsa staining. If either or both the tests were positive, the patient was considered to have a positive H.pylori status. The standard triple therapy was used for eradication of H.pylori and those who tested negative for H.pylori, were included in the second group, and continued on their conventional therapy for ITP. Both the groups of patients were followed up at 2, 6 and 10 weeks of enrolment. Their clinical status, number of bleeding episodes and adverse reaction to drugs if any, were recorded. Blood investigation for platelet count was done for all patients at the follow up visit. Statistical Analysis: Statistical analysis of the findings was done using Graph Pad Instat Software, version 3 (Graph Pad, San Diego, CA, USA). Results: 1. Higher incidence of ITP among females and in patients with low socioeconomic status and more number of family members. 2. Significant increase in platelet counts in case group after 6-10 weeks of eradication of H.pylori infection. Conclusion: There is a significant improvement in platelet counts of Immune thrombocytopenic purpura patients at 6 -10 weeks of therapy.***

Keywords: Immune Thrombocytopenic Purpura, Helicobacter pylori, Platelet count, bleeding episodes

1. Introduction

Helicobacter pylori (H.pylori) is a microaerophilic gram negative organism which infects nearly half of the human population (1).It has been implicated in many upper gastro intestinal diseases like chronic gastritis, peptic ulcer, gastric cancer and gastric lymphoma. Recently, certain extra-intestinal and autoimmune conditions have also been reported to be associated with H. pylori infection. The most important among them are iron deficiency anaemia and immune thrombocytopenic purpura (ITP) (2). The Maastricht III consensus, which gives guidelines for management of H.pylori infections, also recommends testing for H.pylori infection in patients with ITP (2).

Several mechanisms have been proposed to explain the association between

H. pylori and ITP (3, 4).Antibodies against H.pylori, are thought to cross react with the platelets, causing thrombocytopenia (5). Other proposed mechanisms include a modulation of host immunity, by H.pylori, which favours the emergence of autoreactive B-1 cells and the enhancement of phagocytic capacity of monocytes (6).

Some recent studies have found a persistent increase in the platelet count in over half of the patients with chronic ITP who were given H.pylori eradication treatment (4). These findings emphasize the need for evaluation of H.pylori as an etiologic factor of ITP (7).

India is a country with a high prevalence of H. Pylori with a reported incidence of more than 60% (8).To the best of our knowledge, no studies have been reported from India on the prevalence of H. pylori infection in patients with ITP or the effect eradication of H. pylori on platelet counts in patient with ITP.

2. Review of Literature

Immune Thrombocytopenic Purpura

Earlier known as Idiopathic Thrombocytopenic Purpura, Immune Thrombocytopenic Purpura (ITP), comprises a heterogeneous group of disorders characterized by autoimmune-mediated platelet destruction and impairment of thrombopoiesis (9). It can be either primary, where no obvious cause leading to thrombocytopenia can be identified or secondary, where the low platelet count is mediated by immune reactions to other conditions like auto immune diseases or even medications. Primary immune thrombocytopenic purpura (ITP) remains a diagnosis of exclusion.

Immune thrombocytopenia can be secondary to medications or to a concurrent disease, such as an autoimmune condition (eg, systemic lupus erythematosus [SLE], antiphospholipid antibody syndrome [APS], immune thyroid disease, or Evans syndrome), a lymphoproliferative disease (eg, chronic lymphocytic leukemia or large granular T-lymphocyte lymphocytic leukemia), or chronic infection, eg, Helicobacter pylori, human immunodeficiency virus (HIV), or hepatitis C virus (HCV) (5).

ITP is treated conventionally with steroids of the glucocorticoid group. The commonly used drug is prednisone in a dose of 1mg/kg (10).

Helicobacter pylori

Marshall and Warren in 1984 reported the presence of a

curved bacillus in the antral biopsies of patients with active chronic gastritis, duodenal or gastric ulcers (11). This bacterium was cultured and later identified to be *Helicobacter pylori*. This work won them the Nobel Prize in 2005. *H. Pylori* has been, from then, implicated in the etiology of gastritis and gastritis- associated diseases like gastric ulcer, duodenal ulcer, and primary gastric B-cell lymphoma (MALToma) (12).

Extra gastric manifestations of H.pylori

Recently there has been increasing evidence of the association of H.pylori with many diseases other than acid peptic disease and its sequelae. It is thought to be associated with a number of extra gastric pathologies including hepatocellular carcinoma, gallstones formation and cholangiocellular carcinoma, as well as enteric diseases and inflammatory bowel diseases. An infectious etiology and association of novel species of Helicobacter is hypothesized in these conditions (13). H.pylori has also been implicated in conditions like ITP and sideropenic anaemia (6). H.pylori is even thought to be associated with cardiovascular disease (13). The majority of the reports are anecdotal, epidemiologic, or eradication studies, but there are also relevant in vitro studies. ITP represents one disease showing a strong link with H. pylori infection (14).

H.pylori and Immune Thrombocytopenic Purpura

Among the proposed extra gastric manifestations of H.pylori infection, ITP is one of the diseases in which a strong association has been established (13). Many theories have been proposed to explain the development of thrombocytopenia in the presence of H.pylori infection. The immune response to infection generating antibodies that cross react with the platelet antigens and causing failure of platelet aggregation is one of the most popular theories (15). Other theories include molecular mimicry, platelet aggregation, and the induction of a Th1 phenotype that favours the onset and/or persistence of ITP. The role of bacterium-related factors, such as the CagA (cytotoxin-associated gene A) protein, is still under investigation (6). Platelet production may be impaired by infection of megakaryocyte (MK) bone marrow- dependent progenitor cells or decreased production of thrombopoietin (TPO) (5).

H.pylori eradication in ITP

The role of H.pylori in the etiology of ITP having been established, many studies have tried to evaluate the effect of eradication of H.pylori infection on the platelet count of patients with ITP and H.pylori infection. Studies from Japan have showed a recovery of platelet counts in nearly half of the patients with ITP, in whom H.pylori was eradicated (6). Some studies have shown complete recovery in upto 75% of patients diagnosed to have ITP and treated with H.pylori eradication regimen, and partial recovery in the rest (4). No significant difference was found in those patients who were negative for H.pylori. These findings are to be validated by larger trials as most of the reported studies included only small study groups. Eradication therapy is simple and inexpensive, with limited toxicity and the advantage of avoiding long-term immunosuppressive treatment for those who respond. Although the evidence and follow-up are limited, it appears reasonable to routinely screen patients with ITP for H.pylori, particularly in those populations with a high background prevalence of H.pylori infection (6).

Aims and Objectives

1) To determine the prevalence of H.pylori infection in patients diagnosed to have Immune Thrombocytopenic Purpura.
2) To assess the effect of eradication of H.pylori infection on the platelet count of patients with ITP who are positive for H.pylori infection.
3) To identify and evaluate other factors that may influence the changes in platelet counts of the studied patient group.

Patients and Methods

This study was conducted in a prospective controlled interventional design. Approval was obtained from the Institute Research and Ethics Committee. Informed consent was taken from all the patients included in the study.

Patients

All consecutive patients who attended the haematology clinic with a diagnosis of ITP, made as per the clinical criteria proposed by the American Society of Haematology, were considered for enrolment into the study.

Those patients with-

- Platelet count less than 20, 000/mm^3.
- Active life threatening bleeding at time of enrolment.
- History of use of proton pump inhibitors or antibiotics in the two weeks before presentation, or
- History of eradication therapy for H.pylori within two years prior to enrolment.
- Were excluded from the study.

Details of the patient including biodata and relevant details related to the disease and treatment were collected and noted as per the proforma (Annexure 1).

3. Methods

Blood investigation for complete haemogram was done at the time of enrolment of the patients.

Diagnosis of H.pylori infection

All recruited patients were subjected to upper gastro intestinal endoscopy (UGIE) using a video endoscope (EG 3400; Pentax, Montvale, NJ, USA) under topical anesthesia using 2% lignocaine viscous. Four gastric mucosal biopsies (two each from the gastric corpus and the antrum) were taken using standard endoscopic biopsy forceps. Two of these were used for urease test and the rest two for histology after Giemsa staining. All endoscopies were done by experienced consultant endoscopists of our institute.

Diagnosis of positivity for *H.pylori* was done by urease

test or histology by Giemsa staining.

Urease test was done using a urea solution prepared and standardized in our institute. (**Urea test broth** is prepared by adding 20 g of urea, 9.5 g of Na_2HPO_4, 9.1 g of KH_2PO_4, 0.1g of yeast extract and 0.01 g of phenol red. The pH is made to 6.8±0.2 at 25°c). Non invasive tests (urea breath test) may provide a more rapid diagnosis and be less expensive but offer similar accuracy (17). The solution was observed at room temperature till 24 hours of inoculation with two gastric mucosal biopsies. The change of colour of the solution from yellow to pink was considered as positive test for *H.pylori* infection.

Histology was done by Giemsa staining of the biopsies for identification of

H.pylori

If either or both the tests were positive, the patient was considered to have a positive *H.pylori* status. If both the tests were negative, the patient was considered to be negative for *H.pylori* infection.

Patient groups

Those patients who were found to be positive for H.pylori were included in the first group and were given eradication therapy for H.pylori in addition to the other conventional therapy for ITP. The standard triple therapy, was used for eradication of H.pylori, which included

Omeprazole 20mg bd

Clarithromycin 500mg bd xfor 14 days. Amoxicillin 1g bd

The drugs for ITP were continued during and after the H.pylori eradication therapy.

Those patients who tested negative for H.pylori, included in the second group, were continued on their conventional therapy for ITP with no additional therapy.

Follow up

Both the groups of patients were followed up at 2, 6 and 10 weeks of enrolment. Their clinical status, number of bleeding episodes and adverse reaction to drugs if any, were recorded. Blood investigation for platelet count was done for all patients at the follow up visit.

Patients in the first group who received H.pylori eradication therapy were subjected to a follow up UGIE and endoscopic biopsies were taken to confirm H.pylori eradication. Patients who continued to be positive for H.pylori even at follow up UGIE, were administered second line salvage regimen for H.pylori eradication, which included the drugs.

Omeprazole 20mg bd.
Bismuth subcitrate 240mg bd. x For 14 days. Tetracycline hydrochloride 500mg bd
Metronidazole 500mg bd

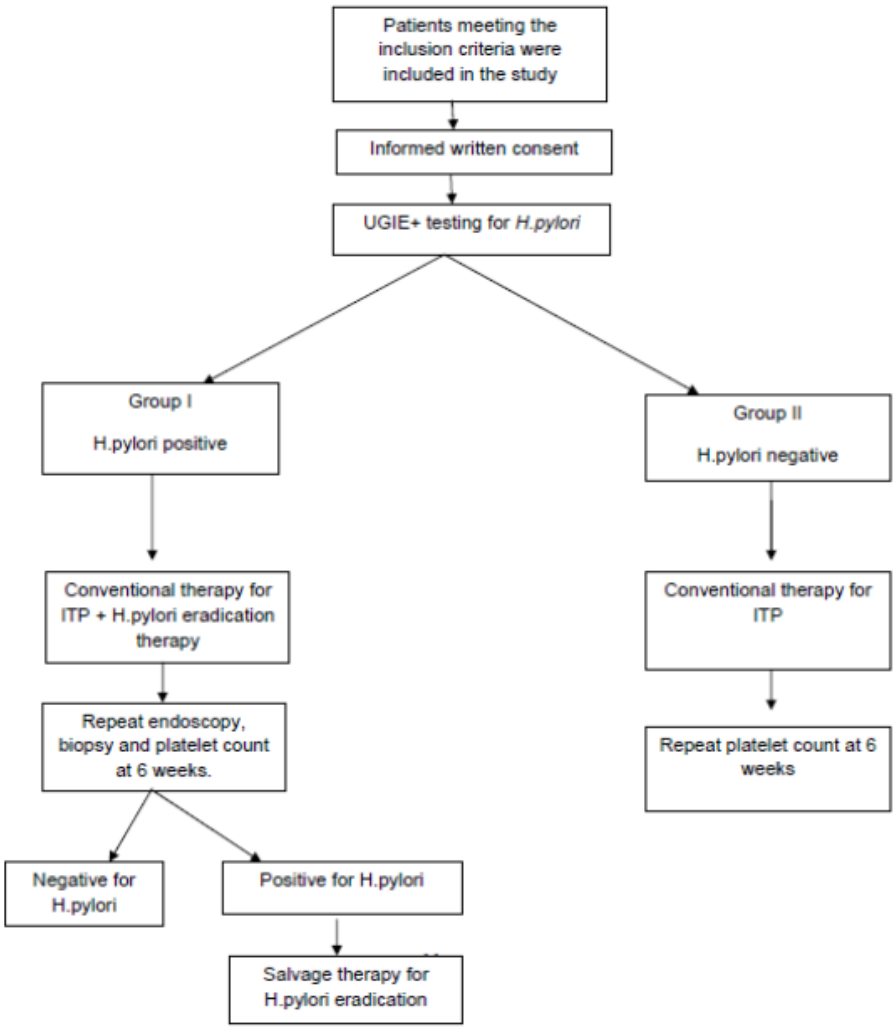
Parameters studied

- Platelet counts in both the groups of patients.
- Demographic details.
- Compliance to drugs.
- Adverse drug reactions.
- Number of bleeding episodes.

Statistical analysis

Statistical analysis of the findings was done using Graph Pad Instat Software, version 3 (Graph Pad, San Diego, CA, USA). Mann Whitney U test and Kruskal-Wallis test were used to compare the mean platelet counts in the two groups. Multivariate analysis, of the pre-intervention characteristics of those patients who showed significant improvement in platelet counts with H.pylori eradication, was done to determine if any of them had a positive effect on the improvement. Characteristics like baseline platelet count, age sex, demographic parameters, duration of disease, presence of dyspeptic symptoms, were studied.

The study design is summarised in the flowchart (Fig 1).



4. Observation and Results

Table 1: Comparison of Prevalence of H.Pylori and Gender Distribution in Case and Control Group of ITP Patients.

Patient	H.pylori positive ITP (CASE)	H.pylori negative ITP (CONTROL)	Total patients (80)
Male	9	17	26
Female	31	23	54
Total	40	40	

Table 2: Comparison of Follow Up Platelet Counts in Case and Control Group Of ITP Patients

Duration (weeks)	H.pylori positive (Mean ± SEM)	H.pylori negative (Mean ± SEM)	P value (one tailed)
2	179800 ± 31028	165200 ± 32090	0.3760
6	255000 ± 29196	180000 ± 40363	0.0363
10	292000 ± 40573	121000 ± 54000	0.0121

Group analysis of follow up platelet counts by KRUSKAL-WALLIS test is insignificant. (p value - 0.4742, KRUSKAL-WALLIS statistic- 6.577).

Table 3: Comparison of Various Parameters in Case snd Control Group of ITP Patients

Parameters	H.pylori positive (Mean ± SEM)	H.pylori negative (Mean ± SEM)	P value (one tailed)
Per Capita Income (Rs)	514 ± 65	1278± 295	0.0105
BMI	22 ± 1.2	25 ± 1.7	0.0916
No. Of Bleeding Episodes	4.3 ± 0.7461	6.8 ± 0.8919	0.0227
Duration Of Symptoms (Months)	10.6 ± 2	29 ± 7	**0.0118**
No. Of Family Members	6 ± 0.5	4 ± 0.5	0.0384

4.1 Observation

A total of 80 patients of ITP were included in the study from the hematology clinic. Among all patients, those who tested positive for H.pylori were taken as case group and those who tested negative for H.pylori were taken as controlgroup.

TABLE 1 shows the prevalence of H. pylori infection in patients with ITP. Out of 80 cases studied, 40 (9 males and 31 females) of them proved to be H.pylori positive by biopsy and geimsa staining. 40 (17 males and 23 females) of 80 proved to be H.pylori negative by biopsy and giemsa staining method. Hence the prevalence of H.pylori infection in studied patients of ITP was found to be 50%.

TABLE 2 shows the comparison of follow up serial platelet counts of case and control group in ITP patients. Platelet count estimation of patients was done at an interval of 2 weeks, 6 weeks and 10 weeks. At 2 week mean platelet count of case group is 1, 79, 800 and the standard error of mean is 31, 028 whereas control group has a mean platelet count of 1, 65, 200 and the error of mean was found to be 32, 090. P value is 0.3760 and is insignificant.

At 6 weeks case group has a mean platelet count of 2, 55, 000 and the error of mean is 29, 196.control group shows the mean platelet count of 1, 80, 000 and the error of mean was found to be 40, 363. P value is 0.0363 and is significant.

At 10 weeks case group has a mean platelet count of 2, 92, 000 and the error of mean is 40, 573.wheras control group shows the mean platelet count of 1, 21, 000 and the error of mean was found to be 54, 000. P value is 0.0121 and is insignificant.

Group analysis of follow up platelet counts was also done wrt platelet count at the time of enrolment, both in case and control group. 8 sets of data were considered for this analysis. Initial platelet count and counts at 2, 6 and 10 weeks of follow up for both case and control group were analysed. Analysis was done with KRUSKAL-WALLIS TEST.

P value came out to be 0.04742 and is insignificant. Kruskal-wallis statistic score- 6.577.

Table 3 shows the comparison of per capita income of case and control group in ITP patients. Analysis of the data shows that ITP patients with H.pylori positivity have mean per capita income of Rs. 514 and the standard error of mean is Rs.65 whereas the control group has a mean per capita income of Rs.1278 and the standard error of mean is Rs.295. P value is 0.0105 which is less than 0.05 and is significant. Comparing gender in case and control group of ITP patients. Among 10 patients belonging to the case group, 9 are females and 1 is male so female: male ratio is 9:1.

In the control group 7 patients are female and 3 is male out of total 10. Hence the female:male ratio in control group is 7:3.
Out of total 20 ITP patients studied female:male ratio is 4:1.

Comparison of BMI of case and control group in ITP patients. Analysis shows that case group has a mean BMI of 22 and standard error of mean is 1.2 whereas control group has a mean BMI of 25 and the standard error of mean is 1.7

P value is 0.0916 which is statistically insignificant.

Comparison of number of bleeding episodes in case and control group in ITP patients. Statistical analysis shows that mean of number of episodes in case group is 4.3 and standard error of mean is 0.75. In control group mean of number of episodes is 6.8 and the standard error of mean is 0.9

P value is 0.0227 and is significant.

Comparison of duration of symptoms (in months) from the time of diagnosis in case and control group of ITP patients. Data analysis shows that mean duration of symptoms in case group is 10 months and 20 days and standard error of mean is 2 months. In control group mean duration of symptoms in case group is 29 months and the standard error of mean is 7 months.
P value is 0.0118 and is significant.

Comparing the number of family members in case and control group of ITP patients. Mean of number of family members in case group is 6 and the standard error of mean is 0.5 whereas mean number in control group is 4 and the standard error of mean is 0.5

P value is 0.0384 which is less than 0.05 and is significant.

5. Discussion

In the present study it was found that ITP is found predominantly in females. The gender ratio is highly suggestive of this finding. However no study was found to conclusively state the incidence of ITP and prevalence of H.pylori infection in ITP patients.

Follow up study does not show any clear change in platelet counts after eradication of H.pylori between 2 to 6 weeks duration. However increase in platelet count is statistically significant between 6 to 10 weeks. This can be explained by the immunological phenomenon that antibody formation against H.pylori causes destruction of platelets. As eradication of H.pylori leads to decrease in antibody formation against platelet, destruction is reduced and increase in platelet count in patients of ITP becomes significant in 6-10 weeks duration.

Per capita income is a direct indicator of the socioeconomic status of the patient. We found in this study that H.pylori positive patients have significantly lower per capita income compared to the H.pylori negative group. This finding may be explained by the poor sanitation in the low socioeconomic group leading to higher incidence of

H.pylori infection, rate of transmission directly or indirectly among the studied case group. Most common mode of transmission being feco- oral route; low per capita income of the patient is associated with the higher incidence of the infection.

Incidence of ITP is significantly higher in females. Female to male ratio 31:9. This can be attributed to the higher incidence of autoimmune conditions in females. This finding compares well with similar autoimmune conditions such as SLE, rheumatoid arthritis, grave's disease, Hashimoto's thyroiditis and multiple sclerosis.

Comparison of BMI among the case and control group shows lower BMI in H.pylori positive patients and higher BMI in H.pylori negative patients, but the difference is not significant. The association of BMI with H.pylori infection in patients with ITP is also not very clear. However a study done at AIIMS by singh et al found that higher body mass index and higher per capita income were associated with successful H. pylori eradication and duodenal ulcer healing, respectively (17).

Comparison of number of bleeding episode in case and control group of ITP patients shows higher number of bleeding episodes in control group as compared to case group which is difficult to explain on the basis of this study and need further research.

Duration of symptoms of disease is longer in control group as compared to the case group. This is due to remission of symptoms in case group due to eradication of Helicobacter and subsequent increase in platelet counts.

Comparison of number of family members in both groups indicates that prevalence of H.pylori infection is more in families with more members. Most of our patients belong to the lower socioeconomic strata and overcrowding is frequently present among such family structure. This can be a cause of poor sanitation and higher rates of infection and thus a probable explanation of higher prevalence of infection in families with more members.

6. Conclusions

1) Prevalence of H.pylori infection in studied patients of ITP is 50%.
2) There is a significant increase in platelet count in case group after 6 to 10 weeks of eradication of H.pylori infection.
3) Higher prevalence of H.pylori infection in studied group with low per capita income and more number of family members.
4) Higher prevalence of ITP in female patients, female to male ratio is 31:9.

7. Summary

The study was undertaken to study the effect of **HELICOBACTER PYLORI ERADICATION ON PLATELET COUNTS IN PATIENTS WITH IMMUNE THROMBOCYTOPENIC PURPURA.**

In this study a total of 80 patients with immune thrombocytopenic purpura who presented to the hematology clinic of JIPMER Pondicherry were included. An upper GI endoscopy was done for all these patients and presence of H.pylori infection was determined by rapid urease test and histology by geimsa stain for the biopsy specimen. A detail proforma was filled for all patients including their demographic and clinical details. Statistical analysis was done using Mann-Whitney test and Kruskal-Wallis test.

Following observations were made:

1) Higher incidence of ITP among female patients.
2) Significant increase in platelet counts in case group after 6-10 weeks of eradication of H.pylori infection.
3) Higher prevalence of H.pylori infection in patients with lowsocioeconomic status.
4) Higher prevalence of H.pylori infection in studied group with more number of family members.

8. Limitations

1) Small sample size.
2) Tissue processing and histopathological examination after endoscopy takes long time so it was possible to collect endoscopy report only after 3 weeks of endoscopy.
3) Complete longer follow up study was not possible for all the patients due to poor compliance, migration, problems faced by patients belonging to distant and remote areas, increasingly making it difficult for them to come for regular follow up visits.

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Proforma

Name:
Age:
Sex:
Case No:
MRD no:
Address:
Phone number:

Epidemiological Parameters

District of residence:
Number of family members:
Type of house:
Number of rooms in the house:
Mode of defecation:
Type of toilet:
Source of drinking water:
Monthly income of family:
Occupation of patient:
Occupation of main earning member:
Smoking- yes/no, if yes, give details:
Alcohol- yes/no, if yes, give details:

Clinical Parameters

Weight:
Height:
Duration of symptoms:
Bleeding episodes in past:

Approximate number of episodes so far Sites of bleed encountered so far Hospitalisations for bleed- yes/no
Diagnosis of ITP made in which year- Drugs received so far for ITP-
Dyspepsia– yes/no Previous endoscopy- yes/no
Previous H pylori testing- yes/no
Previous eradication therapy- yes/no, if yes, give details
Details of previous PPI use:
Previous ulcer surgery- yes/no

Basic Investigations

Hb:
TC:
DC:
Platelet count:
ESR:
Peripheral smear:

Previous investigations

1) Bone marrow aspiration/biopsy:
2) Previous lowest platelet count:
3) USS abdomen:
4) Other investigations in the past:
5) Endoscopy report with date:
6) RUT:
7) Biopsy report:
8) Treatment details with date:
9) Compliance:
10) Adverse effects noticed:

Follow Up

Repeat endoscopy report:
RUT:
Biopsy:
Platelet count:

JIPMER HO9PFFAL, PtJDUCHERRY- BOM 006
CONSENT FORM

I Hospital No. have been told by my doctor that I have a bleeding disease called ITP and one of the accepted treatments for this disease is a combination of throe drugs, which are to be given to me only If I have a particular infection called H. Py/on. To know accurately whether I have this infection I need to undergo an endoscopy and biopsy. My doctor has explained to me about the procedure and its possible complications.

In my full senses, I agree for the same. I also agree for taking my blood sample for platelet count eatlmatlon. I also know that this treatment is part of a clinical study and I give full permission to use my clinical details for research

Daw:

Signature of Witness

Name:

Designation:

Signature / Thumb Impression of the Patient I Guardian

Guardian

Relationship: FuB Address

Suggestions for Future Research

- Larger studies need to be conducted to be able to conclusively establish the role of H.pylori eradication on platelet counts in patients with ITP.
- Complete follow up studies should be conducted for a longer period of time to asses the pattern of change in platelet counts.