53. Schiavotto C, Ruggeri M, Rodeghiero F. Failure of repeated courses of high-dose intravenous immunoglobulin to induce stable remission in patients with chronic idiopathic thrombocytopenic purpura. Ann Hematol 1995;70:89-90.
54. Godeau B, Lesage S, Divine M, Wirquin V, Farcet JP, Bierling P. Treatment of adult chronic autoimmune thrombocytopenic purpura with repeated high-dose intravenous immunoglobulin. Blood 1993;82:1415-21.
55. Schiavotto C, Ruggeri M, Rodeghiero F. Adverse reactions after high-dose intravenous immunoglobulin: incidence in 83 patients treated for idiopathic thrombocytopenic purpura (ITP) and review of the literature. Haematologica 1993; 78:35-40.
56. Ruggeri M, Castaman G, De Nardi G, Rodeghiero F. Acute renal failure after high-dose intravenous immune globulin in a patient with idiopathic thrombocytopenic purpura. Haematologica 1993;78:338.
57. Gaines AR. Acute onset hemoglobinemia and/or hemoglobinuria and sequelae following Rh(o)(D) immune globulin intravenous administration in immune thrombocytopenic purpura patients. Blood 2000;95:2523-9.
58. CDC Recommendations of the Advisory Committee on Immunisation Practices: use of vaccines and immuneglobulins in persons with altered immunocompetence. Morbidity and Mortality Weekly Report 1993;42:1-12.
59. McMillan R. Long-term outcomes after treatment for refractory immune thrombocytopenic purpura. N Engl J Med 2001; 344:1402.
60. Andersen JC. Response of resistant idiopathic thrombocytopenic purpura to pulsed high-dose dexamethasone therapy. N Engl J Med 1994;330:1560-4.
61. Kappers-Klunne MC, van't Veer MB. Cyclosporin A for the treatment of patients with chronic idiopathic thrombocytopenic purpura refractory to corticosteroids or splenectomy. Br J Haematol 2001;114:121-5.
62. Emilia G, Messora C, Longo G, Bertesi M. Long-term salvage treatment by cyclosporin in refractory autoimmune haematological disorders. Br J Haematol 1996;93:341-4.
63. Ahn YS, Rocha R, Mylvaganam R, Garcia R, Duncan R, Harrington WJ. Long-term danazol therapy in autoimmune thrombocytopenia: unmaintained remission and age dependent response in women. Ann Intern Med 1989;111:723-9.
64. Schiavotto C, Castaman G, Rodeghiero F. Treatment of idiopathic thrombocytopenic purpura (ITP) in patients with refractoriness to or with contraindication for corticosteroids and/or splenectomy with immunosuppressive therapy and danazol. Haematologica 1993;78 Suppl 2:29-34.
65. Proctor SJ, Jackson G, Carey P, Stark A, et al. Improvement of platelet counts in steroid-unresponsive idiopathic immune thrombocytopenic purpura after short-course therapy with recombinant α2b interferon. Blood 1989;74:1894-7.
66. Godeau B, Durand JM, Shaeffer A, Bierling P, et al. Dapsone for chronic autoimmune thrombocytopenic purpura: a report of 66 cases. Br J Haematol 1997;97:336-9.
67. Brox AG, Howson-Jan K, Fauser AA. Treatment of idiopathic thrombocytopenic purpura with ascorbate. Br J Haematol 1988;70:341-4.
68. Strother SV, Zuckerman KS, LoBuglio AF. Colchicine therapy for refractory idiopathic thrombocytopenic purpura. Arch Intern Med 1984;144:2198-200.
69. Snyder HW Jr, Cochran SK, Balint JP Jr, Bertram JH, Mittelman A, Guthrie TH Jr, et al. Experience with protein A-immunoadsorption in treatment-resistant adult immune thrombocytopenic purpura. Blood 1992;79:2237-45.
70. Gasbarrini A, Franceschi F, Tartaglione R, Landolfi R, Pola P, Gasbarrini G. Regression of autoimmune thrombocytopenia after eradication of Helicobacter pylori. Lancet 1998;352: 878.
71. Emilia G, Longo G, Luppi M, Gandini G, Morselli M, Ferrara L, et al. Helicobacter pylori eradication can induce platelet recovery in idiopathic thrombocytopenic purpura. Blood 2001;97:812-4.
71 bis.Franchini M, Veneri D. Helicobacter pilori infection and immune thrombocytopenic purpura. Haematologica 2003, 88:1087-91.
72. Veneri D, Franchini M, Gottardi M, D'Adda M, Ambrosetti A, Pizzolo G. Efficacy of Helicobacter pylori eradication in raising platelet count in adult patients with idiopathic thrombocytopenic purpura. Haematologica 2002;87:1177-9.
73. Skoda RC, Tichelli A, Tyndall A, Hoffmann T, Gillessen S, Gratwohl A. Autologous peripheral blood stem cell transplantation in a patient with chronic autoimmune thrombocytopenia. Br J Haematol 1997;99:56-7.
74. Howard J, Hoffbrand AV, Prentice HG, Mehta A. Mycophenolate mofetil for the treatment of refractory auto-immune haemolytic anaemia and auto-immune thrombocytopenia purpura. Br J Haematol 2002;117:712-5.
75. Stasi R, Pagano A, Stipa E, Amadori S. Rituximab chimeric anti-CD20 monoclonal antibody treatment for adults with chronic idiopathic thrombocytopenic purpura. Blood 2001; 98:952-7.
76. Delgado J, Bustos JG, Jimenez-Yuste V, Hernandez-Navarro F. Anti-CD20 monoclonal antibody therapy in refractory immune thrombocytopenic purpura. Haematologica 2002; 87:215-6.
77. Saleh MN, Gutheil J, Moore M, Bunch PW, Butler J, LoBuglio AF. A pilot study of the anti-CD20 monoclonal antibody rituximab in patients with refractory immune thrombocytopenia. Semin Oncol 2000;27 Suppl 12:99-103.
78. Lim SH, Hale G, Marcus RE, Waldmann H, Baglin TP. Campath-1 monoclonal antibody therapy in severe refractory autoimmune thrombocytopenic purpura. Br J Haematol 1993; 84:542-4.
79. Willis F, Marsh JC, Bevan DH, Killick SB, Lucas G, Smith EC. The effect of treatment with Campath-1H in patients with autoimmune cytopenias. Br J Haematol 2001;114:891-8.
80. Kuter DJ, Begley CG. Recombinant human thrombopoietin: basic biology and evaluation of clinical studies. Blood 2002; 100:3457-69.

Helicobacter pylori infection and immune thrombocytopenic purpura

Helicobacter pylori is a recently re-discovered Gram-negative bacterium which has revolutionized the understanding of the pathogenesis of peptic ulcer disease and hence its treatment.[1] *H. pylori* is etiologically related to many digestive tract diseases including peptic ulcer disease, chronic active gastritis, primary low-grade B cell gastric lymphoma and gastric carcinoma.[2,3] The seroprevalence of *H. pylori* has also been investigated in many other diseases and a positive correlation has been found in an increasing number of conditions outside the digestive tract, such as cardiovascular, respiratory, neurological, skin and autoimmune disorders.[4,5] These last include rheumatoid arthritis, autoimmune thyroiditis and autoimmune neutropenia. The interaction between *H. Pylori* and the immune system was also confirmed by recent studies that reported an association between *H. pylori* infection and idiopathic thrombocytopenic purpura (ITP) and, in many cases, a significant increase in platelet count after bacterial eradication.[6,8] The relationship between infection with *H. pylori* and the development of ITP opens a new exciting and controversial area of investigation with important implications for both pathogenesis and patients' management. However, the data reported in literature are limited[9-24] and mostly regard single case reports or small series of patients so that the evi-

Table 1. *Helicobacter pylori* infection and idiopathic thrombocytopenic purpura: review of the published data.

		Patients			
Authors	*Total number*	*Infected (%)*	*Bacterial eradication (%)*	*Platelet response (%)**	*Median follow-up (months)*
Gasbarrini *et al.*[9] (1998)	18	11 (61.1)	8 (72.7)	8 (100.0)	4.0
Garcia Perez *et al.*[10] (1999)	1	1	1	1	32.0
Grimaz *et al.*[11] (1999)	1	1	1	1	21.0
Tohda *et al.*[12] (2000)	1	1	1	1	6.0
Emilia *et al.*[13] (2001)	30	13 (43.3)	12 (92.3)	6 (50.0)	8.3
Soldinger *et al.*[14] (2001)	1	1	1	1	3.0
Jarque *et al.*[15] (2001)	56	40 (71.4)	23/32 (71.9)†	3 (13.1)	24.0
Kumagai *et al.*[16] (2001)	1	1	1	1	32.0
Veneri *et al.*[17] (2002)	35	25 (71.4)	15/16 (93.7)‡	11 (73.3)	11.7
Emilia *et al.*[18] (2002)	7	3 (42.9)	3 (100.0)	2 (66.7)	–
Kohda *et al.*[19] (2002)	48	27 (56.2)	19/19 (100.0)§	12 (63.2)	14.8
Mukai *et al.*[20] (2002)	2	2	2	2	2.5
Michel *et al.*[21] (2002)	51	15 (29.4)	–	–	–
Hino *et al.*[22] (2003)	30	21 (70.0)	18 (85.7)	10 (55.6)	15.0
Hashino *et al.*[23] (2003)	22	14 (63.6)	13 (92.9)	5 (38.5)	15.0
Ando *et al.*[24] (2003)	61	50 (82.0)	27/29 (93.1)¶	16 (59.2)	11.0
Total	365	226 (61.9)	145/165 (87.9)	80 (55.2)	14.0

**Complete or partial response. †Bacterial eradication was obtained in 23 of 32 treated patients (71.9%). ‡Bacterial eradication was obtained in 15 of 16 treated patients (93.7%). §Bacterial eradication was obtained in 19 of 19 treated patients (100.0%).¶Bacterial eradication was obtained in 27 of 29 treated patients (93.1%).*

dence of a link between *H. pylori* and ITP remains speculative. The aim of this concise review was to consider the studies so far published on *H. pylori* infection and ITP in order to evaluate the seroprevalence of *H. pylori* infection and the response to eradication treatment in ITP patients and to clarify, if possible, the role of this bacterium in the pathogenesis of immune thrombocytopenic purpura.

Source of materials

This review collects the studies so far published in the medical literature on ITP and *Helicobacter pylori* infection. MedLine reports from January 1998 to June 2003 were searched via PubMed using the following terms: '*thrombocytopenia*', '*autoimmune thrombocytopenic purpura*', '*immune thrombocytopenic purpura*', '*idiopathic thrombocytopenic purpura*', and '*ITP*'. All these terms were searched for alone and in combination with '*therapy*', '*infection*', '*Helicobacter pylori*', '*Helicobacter pylori infection*', '*H. pylori*', and '*H. pylori infection*'. This search generated 31 references, 16 of which[9–24] reported bacterial seroprevalence and the results of eradication treatment in adult ITP patients infected by *H. pylori*. These studies are listed chronologically in Table 1.

Prevalence of Helicobacter pylori infection in ITP patients

It is well known that seroprevalence of *H. pylori* infection in otherwise healthy individuals varies greatly from country to country, that it has decreased over the last decades in industrialized countries and that it increases with age.

Table 1 summarizes the clinical data from the medical literature on ITP patients and *H. pylori* infection, including the seroprevalence. Although

the studies reported so far are few and regard small series of patients, most of the authors found a high prevalence of *H. pylori* infection in ITP patients. However, since the majority of previous studies involving a relatively large number of patients were conducted in Italy and Japan, in which the prevalence of *H. pylori* infection in the healthy population is high (> 70% over the age of 50 years),[25,26] the prevalence of *H. pylori* infection in ITP patients in these countries may not be different from the prevalence in the healthy population. Only one French study[21] recorded a low prevalence (29%) of *H. pylori* infection in ITP patients, similar to that found in control subjects. Although this may reflect the great variability in the rate of infection in different areas, we must highlight that the authors chose the serologic antibody method to detect *H. pylori* infection. This assay does not necessarily indicate an active *H. pylori* infection, since antibody titers may remain positive for years after successful treatment, and is a more laborious and less reliable assay than the urea breath test, which was the method used in almost all the other reported studies.[27] On the whole, even considering the study by Michel *et al.*,[21] 226 out of the 365 ITP patients studied (62%) so far have been found to have *H. pylori* infection. The data must be interpretated cautiously because of the variability in rate of *H. pylori* infection in different areas and generations. In fact, in our opinion, for meaningful results, the seroprevalence in ITP patients must be compared with that in healthy populations in different geographical areas, corrected for age distribution. Individual cohorts should have their own regional, age-matched controls, although most of the previous studies did not have adequate controls.

Pathogenesis of ITP associated with Helicobacter pylori infection

We recently reported some other proof of the importance of *H. pylori* infection in the pathogenesis of ITP in a subset of adults.[28] In fact, we found a difference in HLA class II allele patterns between ITP patients with or without *H. pylori* infection. Patients with ITP had a lower frequency of HLA-DRB1*11 and –DQB1*03 alleles than did healthy controls. Analyzing the ITP patients with respect to *H. pylori* infection, we observed that this low frequency was a typical feature of *H. pylori*-negative patients. Although this finding may contribute to stratifying different subgroups of ITP patients with probably different pathogeneses of thrombocytopenia and, finally, different responses to eradication treatment, it does not clarify the pathophysiology of immune thrombocytopenia associated with *H. pylori* infection. Some authors studied the presence of autoantibodies against platelets in ITP *H. pylori*-infected patients and found that bacterial eradication and platelet recovery were accompanied by the disappearance of autoantibodies in most cases.[9,19] These results provide further support to the autoimmune hypothesis of ITP associated with *H. pylori* infection in which there would be a cross mimicry between *H. pylori* and platelet antigens. Michel and colleagues[21] investigated this possible molecular mimicry by testing platelets eluates from *H. pylori*-positive patients with ITP for *H. pylori* antibodies, but none of the 3 patients investigated was found to be positive. However, this series seems too small to exclude a possible cross-reactivity between *H. pylori* antibodies and platelet-membrane antigens. Finally, another possible pathogenetic mechanism is the occurrence of a B-cell clonal expansion.[29] In fact, on the basis of previous observations of the association between gastric extranodal marginal zone B-cell lymphoma and *H. pylori* infection and the regression of some cases after bacterial eradication,[30] we cannot exclude a similar mechanism also for *H. pylori*-associated ITP. Thus, chronic *H. pylori* infection could stimulate the emergence of autoreactive clonal B cells producing platelet-reactive antibodies.

Effect of Helicobacter pylori eradication on platelet count in ITP patients

The first observations of a positive association between *H. pylori* infection and ITP and that eradication of the bacterium was accompanied by an increase in platelets in most cases were made by Gasbarrini and colleagues in 1998.[9] Since then, many other authors have verified and, in the majority of cases, confirmed these intriguing preliminary observations. Table 1 lists these studies, analyzing platelet response after bacterial eradication. We do, however, advise caution in interpreting these data, since the patients enrolled in the studies, the bacterial eradication protocols, the evaluation of platelets response and the follow-up periods differed greatly from study to study and make comparisons of the results difficult. In spite of this caveat, it is clear that the majority of patients in whom *H. pylori* infection was eradicated subsequently had a platelet increase. In fact, 80 out of the 145 patients (55.2%) who became *H. pylori*-negative obtained a complete or partial platelet response. Among the studies including the largest series of patients, only Jarque and colleagues[15] observed a very low platelet response (13%) after eradication of *H. pylori* infection in 56 adult patients with chronic ITP. Recently, we demonstrated that also *H. pylori*-positive ITP patients who relapse after steroid therapy and those with severe (< 30×10^9/L platelets), refractory ITP requiring continuous immunosuppressive therapy may be rescued with this bacterial eradication treatment. In fact,[17,31] *H. pylori* eradication significantly increased the platelet count in 6 out of 9 such patients (66.7%) and dra-

matically improved these patients' quality of life, liberating them from immunosuppressive therapy. Another important finding, which indirectly confirms the etiologic role of *H. pylori* in a subset of ITP patients, is that in the majority of the studies reported, platelet count did not differ before and after treatment in those patients in whom the bacterium was not eradicated. However, the data so far published indicate that bacterial eradication resolves the majority but not all cases of *H. pylori* positive ITP. The variability in the response rate to treatment, which could depend on an interaction between genetic host factors (HLA) and bacterial factors (strains), will be clarified when the pathogenesis of this condition becomes completely understood.

Conclusions

The data so far reported confirm the existence of an association between *H. pylori* infection and idiopathic thrombocytopenic purpura. The pathogenesis of ITP associated with *H. pylori* is still not well defined, but it seems that genetic host factors (i.e., the HLA class II system) and bacterial factors (i.e., the variability of *H. pylori* strains) may both play important roles. These factors may also account for the variability in the response to eradication treatment observed in ITP *H. pylori*-infected patients.

Although the data are still limited, there are clear indications that eradication of *H. pylori* has a beneficial effect on platelet recovery in ITP patients. In our opinion, these positive results justify placebo-controlled trials on larger numbers of patients.

Until the results of such trials are available, we advise investigation for *H. pylori* and bacterial eradication in all patients with chronic ITP found to have the infection, since this management may be sufficient to avoid the toxicity and disadvantages of long-term immunosuppressive treatments.

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References

1. Gasbarrini A, Franceschi F. Autoimmune diseases and Helicobacter pylori infection. Biomed Pharmacother 1999; 53:223-6.
2. Tsang KW, Lam SK. Helicobacter pylori and extra-digestive diseases. J Gastroenterol Hepatol 1999;14:844-50.
3. Parsonnet J, Hansen S, Rodriguez I, Gelb AB, Warnke RA, Jellum E, et al. Helicobacter pylori infection and gastric lymphoma. N Engl J Med 1994;330:1267-71.
4. De Luis DA, Varela C, de la Calle H. Helicobacter pylori infection is markedly increased in patients with autoimmune atrophic thyroiditis. J Clin Gastroenterol 1998;26:259-63.
5. Gupta V, Eden AJ, Mills MJ. Helicobacter pylori and autoimmune neutropenia. Clin Lab Haematol 2002;24:183-5.
6. British Society for Haematology. Guidelines for the investigation and management of idiopathic thrombocytopenic purpura in adults, children and in pregnancy. Br J Haematol 2003;120:574-96.
7. Cines DB, Blanchette VS. Immune thrombocytopenic purpura. N Engl J Med 2002; 346:995-1008.
8. McCrae KR, Bussel JB, Mannucci PM, Remuzzi G, Cines DB. Platelets: an update on diagnosis and management of thrombocytopenic disorders. Hematology 2001. Educational Program Book. p. 282-305.
9. Gasbarrini A, Franceschi F, Tartaglione R, Landolfi R, Pola P, Gasbarrini G. Regression of autoimmune thrombocytopenia after eradication of Helicobacter pylori. Lancet 1998; 352: 878.
10. Garcia Perez A, Valverde de La Osa J, Gimenez Samper M, Alonso Garcia I. Resolution of an autoimmune thrombocytopenic purpura after eradication treatment of Helicobacter pylori. Sangre 1999; 44:387-8.
11. Grimaz S, Damiani D, Brosolo P, Skert C, Geromin A, de Pretis G. Resolution of thrombocytopenia after treatment for Helicobacter pylori: a case report. Haematologica 1999; 84:283-4.
12. Tohda S, Ohkusa T. Resolution of refractory idiopathic thrombocytopenic purpura after eradication of Helicobacter pylori. Am J Hematol 2000; 65:329-30.
13. Emilia G, Longo G, Luppi, M, Gandini G, Morselli M, Ferrara L, et al. Helicobacter pylori eradication can induce platelet recovery in idiopathic thrombocytopenic purpura. Blood 2001;97:812-4.
14. Soldinger E, Pilia MC, Piubello W, Nadali G. Multi-resistant idiopathic thormbocytopenia successfully treated by eradication of Helicobacter pylori. Dig Liv Dis 2001;33:732.
15. Jarque I, Andreu R, Llopis I, De la Rubia J, Gomis F, Senent L, et al. Absence of platelet response after eradication of Helicobacter pylori infection in patients with chronic idiopathic thrombocytopenic purpura. Br J Haematol 2001; 115:1002-3.
16. Kumagai T, Sekigawa K, Hashimoto N, Shirato R. Remission of idiopathic thrombocytopenic purpura by eradicating Helicobacter pylori after omeprazole monotherapy. Int J Hematol 2001;74:237-8.
17. Veneri D, Franchini M, Gottardi M, D'Adda M, Ambrosetti A, Krampera M, et al. Efficacy of Helicobacter pylori eradication in enhancing platelet count in adult patients with idiopathic thrombocytopenic purpura. Haematologica 2002; 87:1777-9.
18. Emilia G, Luppi M, Morselli M, Potenza L, D'Apollo N, Torelli G. Helicobacter pylori infection and idiopathic thrombocytopenic purpura. Br J Haematol 2002;118:1198-9.
19. Kohda K, Kuga T, Kogawa K, Kanisawa Y, Koike K, Kuroiwa G, et al. Effect of Helicobacter pylori eradication on platelet recovery in Japanese patients with chronic idiopathic thrombocytopenic purpura and secondary autoimmune thrombocytopenic purpura. Br J Haematol 2002;118:584-8.
20. Mukai M, Kon Y, Notoya A, Kohno M. Helicobacter pylori associated with idiopathic thrombocytopenic purpura. Am J Med 2002;113:169-71.
21. Michel M, Khellfar M, Desforges L, Lee K, Schaeffer A, Godeau B, et al. Autoimmune thrombocytopenic purpura and Helicobacter pylori infection. Arch Intern Med 2002; 162:1033-6.
22. Hino M, Yamane T, Park K, Takubo T, Ohta K, Kitagawa S, et al. Platelet recovery after eradication of Helicobacter pylori in patients with idiopathic thrombocytopenic purpura. Ann Hematol 2003;82:30-2.
23. Hashino S, Mori A, Suzuki S, Izumiyama K, Kahata K, Yonezumi M, et al. Platelet recovery in patients with idiopathic thrombocytopenic purpura after eradication of Helicobacter pylori. Int J Hematol 2003;77:188-91.
24. Ando K, Shimamoto T, Tauchi T, Ito Y, Kuryiama Y, Gotoh A. Can eradication therapy for Helicobacter pylori really improve the thromocytopenia in idiopathic thrombocytopenic purpura? Our experience and literature review. Int J Hematol 2003;77:239-44.
25. Asaka M, Kimura T, Kudo M, Takeda H, Miyazaki T, Miki K, Graham DY. Relationship beteen Helicobacter pylori to serum pepsinogen in an asymptomatic Japanese population.

Gastroenterology 1992;102:760-6.
26. Russo A, Eboli M, Pizzetti P, Di Felice G, Ravagnini F, Spinelli P. Determination of Helicobacter pylori seroprevalence among Italian blood donors. Eur J Gastroenterol Hepatol 1999;11:867-73.
27. Morselli M, Potenza L, Luppi M, Torelli G, Emilia G. Immune thrombocytopenic purpura and Helicobacter pylori infection. Arch Intern Med 2003;163:120.
28. Veneri D, Gottardi M, Guizzardi E, Zanuso C, Krampera M, Franchini M. Idiopathic thombocytopenic purpura, Helicobacter pylori infection and HLA class II alleles. Blood 2002; 100:1926-7.
29. Roark JH, Bussel JB, Cines DB, Siegel DL. Genetic analysis of autoantibodies in idiopathic thrombocytopenic purpura reveals evidence of clonal expansion and somatic mutation. Blood 2002;100:1388-98.
30. Wotherspoon AC, Doglioni C, Diss TC, et al. Regression of primary low-grade B-cell gastric lymphoma of mucosa associated lymphoid tissue type after eradication of Helicobacter pylori. Lancet 1993;342:575-7.
31. Franchini M, Krampera M, Veneri D. Helicobacter pylori eradication in adults with idiopathic thrombocytopenic purpura. Am J Med 2003;114:420-1.

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