

similar to that of a previous attack of the disease, pericarditis is not recurring, and pain management should be started while corticosteroids are avoided. Sometimes it can precede clinical evidence of a recurrence and thus patient reexamination is mandatory to rule out recurrent pericarditis. Female gender, previous use of corticosteroids, and frequent recurrences must be considered as risk factors for recurrent pain. The outcome is not so good with a higher incidence of recurrences and a trend to a higher rate of constrictive pericarditis. Thus, this is a subset of patients who require more intensive follow-up.

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Association of Aspirin Use With Vitamin B₁₂ Deficiency (Results of the BACH Study)

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We examined the prevalence of vitamin B₁₂ deficiency and its association with medication use and characteristics, including infection with *Helicobacter pylori* (*H. pylori*), in 255 patients hospitalized for cardiovascular disease. In almost half of the study population, vitamin B₁₂ deficiency was found; patients using acetylsalicylic acid were more frequently vitamin B₁₂ deficient in comparison to nonusers ($p = 0.02$). Fifty-one percent of the patients were infected with *H. pylori*, and fewer infected patients were vitamin B₁₂ deficient. ©2004 by Excerpta Medica, Inc. (Am J Cardiol 2004;94:975–977)

Vitamin B₁₂ is involved in the remethylation of homocysteine into methionine.¹ This process is catalyzed by the enzyme methionine synthase, facilitated by vitamin B₁₂ and folate. Low vitamin B₁₂ levels will therefore elevate homocysteine concentrations. Functional studies, wherein high homocysteine concentrations were used, have reported diminished endothelial function, which can lead to atherosclerosis.^{2,3} In the secondary prevention of ischemic heart disease, acetylsalicylic acid has been an adequate drug for years.^{4–6} However, patients using a daily low dose of acetylsalicylic acid often develop gastrointestinal complications as an adverse event, in which peptic ulcers and gastric bleeding are most common.^{7–9} This

can interfere in the vitamin B₁₂ absorption process and therefore lead to lower vitamin B₁₂ blood levels. Here we report the prevalence of vitamin B₁₂ deficiency in patients hospitalized for cardiovascular disease. Additionally, vitamin B₁₂ deficiency and its association with *Helicobacter pylori* (*H. pylori*) infection, medication use, and patients' characteristics were studied.

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Patients were recruited between April and October 2002 at the Coronary Care Unit of the Heart-center of the University Medical Center Nijmegen, The Netherlands. Information on demographics, diagnosis, medication use, and history of coronary heart disease was collected. Fasting blood samples were taken from consecutive patients after admission and processed immediately.

Assays of serum vitamin B₁₂ and *H. pylori* antibodies were performed in the hospital laboratory. Samples were run at the same time to minimize assay variability. The Immulite analyzer (DPC, Los Angeles, California) was used for the quantitative measurement of vitamin B₁₂ serum levels. Vitamin B₁₂ levels <150 pmol/L were classified as clinical deficiency, between 150 to 250 pmol/L as subclinical deficiency, and >250 pmol/L as normal.

Testing for antibodies against *H. pylori* infection was performed with a commercially available enzyme-linked immunosorbent assay (Pyloriset, Orion Corp., Orion Diagnostica, Espoo, Finland). The specific antibodies against *H. pylori* infection measured in the serum samples were classified following the manufacturer's instructions. Patients were considered *H. pylori* seropositive when values were >20 U/ml.

The relation among demographic characteristics, *H. pylori* serology, medication use, and categories of

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vitamin B₁₂ levels were evaluated with a chi-square test. For multivariate logistic regression analysis, vitamin B₁₂ status was dichotomous categorized and compared with the main demographic characteristics, *H. pylori* antibody levels, and acetylsalicylic acid use.

A total of 255 consecutive cardiovascular patients were studied, of whom 152 were men (mean age 65 ± 12 years). Patients were hospitalized because of (un)stable angina pectoris (46%), acute myocardial infarction (23%), arrhythmia (14%), heart failure (14%), and other reasons (8%). More than half of the patients (54%) were using acetylsalicylic acid for secondary prevention of ischemic heart disease. A total of 60 patients (24%) did have an episode of use of acid suppression medication.

A clinical deficient serum vitamin B₁₂ level (<150 pmol/L) was detected in 20 patients (8%); 93 patients (37%) had subclinical deficient vitamin B₁₂ levels between 150 and 250 pmol/L (Table 1). (Sub)clinical deficiencies were more common in patients who were >65 years of age (odds ratio [OR], 1.31, 95% confidence interval [CI] 0.80 to 2.17, p = 0.05) and in men (OR 1.2, 95% CI 0.77 to 2.11, p = 0.06). Patients using acetylsalicylic acid (n = 137, 55%) were more frequently deficient (OR 1.83, 95% CI 1.10 to 3.05, p = 0.02); this association was still significantly present after adjustment for age, gender, and *H. pylori* (OR 1.75, 95% CI 1.03 to 2.98, p = 0.02) (Figure 1).

More than half of the patients (51%) were infected with *H. pylori*. The infection lowered the risk of vitamin B₁₂ deficiency (OR 0.61, 95% CI 0.37 to 1.00). After adjusting for gender, age, and acetylsalicylic acid use, this association was still present (OR 0.50, 95% CI 0.29 to 0.87) (Table 2).

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Low levels of vitamin B₁₂ were common in our population of patients hospitalized for cardiovascular disease, who mostly had ischemic heart disease. For almost 10% of our population, this deficiency was clinical relevant and should be treated. Vitamin B₁₂ deficiency was strongly related to the use of acetylsalicylic acid, even after adjustment for age and gender of the patients. Infection with *H. pylori* was often detected, using a standard serology test, in our study population. Because infected patients were less vitamin B₁₂ deficient, infection with *H. pylori* was found to be protective.

Explanations for the association between use of acetylsalicylic acid and vitamin B₁₂ deficiency may be found in the absorption mechanism of vitamin B₁₂. The harmful side effects of acetylsalicylic acid on the stomach mucosa are well known. This could lead to reduction of secretion of intrinsic factor or hypochlorhydria, both factors in the development of vitamin B₁₂ deficiency. Moreover, vitamin B₁₂ plays an important role in the homocysteine metabolism, in which low vitamin B₁₂ could lead to hyperhomocysteinemia. High levels of homocysteine are associated with endothelial damage,¹⁰ followed by atherosclerosis, where acetylsalicylic acid is a common therapy. Here the process ends at its starting point; the beneficial effects of acetylsalicylic acid on platelet aggregation effect seem to be inhibited by its side effects on gastric mucosa.

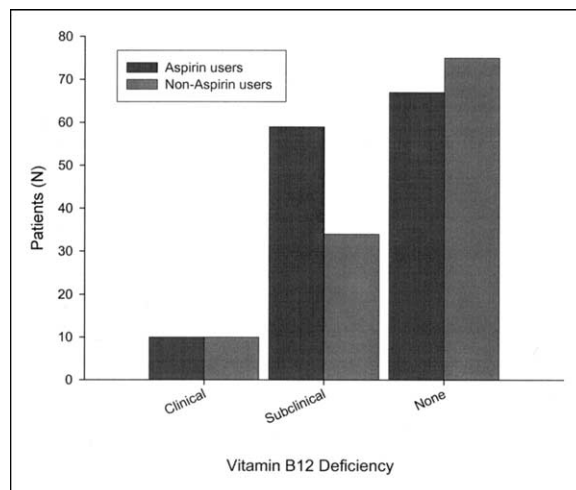


FIGURE 1. Vitamin B₁₂ levels and its association with acetylsalicylic acid use in hospitalized cardiovascular patients.

TABLE 1 Baseline Characteristics

Variable	Serum Vitamin B ₁₂		
	No Deficiency (<150 pmol/L) (n = 142)	Subclinical Deficiency (150–250 pmol/L) (n = 93)	Clinical Deficiency (>250 pmol/L) (n = 20)
Men	81 (57%)	56 (60%)	15 (75%)
Women	61 (43%)	37 (40%)	5 (25%)
Age (yrs)			
<55	31 (22%)	20 (22%)	5 (25%)
55–65	29 (20%)	13 (14%)	3 (15%)
>65	82 (58%)	60 (64%)	12 (60%)
<i>H. pylori</i> seropositive	78 (57%)	39 (44%)	9 (45%)
Aspirin use	67 (48%)	59 (65%)	10 (50%)

TABLE 2 Risk Factor for Vitamin B₁₂ Deficiency

Variable	Vitamin B ₁₂ deficiency	
	Unadjusted OR (95% CI)	Adjusted OR (95% CI)
Aspirin	1.83 (1.10–3.05)	1.75 (1.03–2.98)
Age	1.31 (0.80–2.17)	1.47 (0.85–2.56)
Gender	1.27 (0.77–2.11)	1.42 (0.83–2.45)
<i>H. pylori</i> infection	0.61 (0.37–1.00)	0.50 (0.29–0.87)

We have previously found low serum levels of vitamin B₁₂ in another cardiovascular population of 223 patients.¹¹ In 14% and 30% of that population, low serum (<150 pmol/L) and low-normal (150–250 pmol/L) vitamin B₁₂ levels were found, respectively. In a study of 105 Dutch elderly subjects, which also included 25% of chronic cardiovascular patients, the median plasma vitamin B₁₂ concentration was 230 pmol/L. Plasma vitamin B₁₂ was low (≤150 pmol/L) in 25% and low-normal (160 to 260 pmol/L) in 35% of these subjects.¹² We found the same amount of low-normal patients but more clinically deficient patients in our cardiac patient population. These studies did not find any risk factor for these low levels.

The high prevalence of infection with *H. pylori* in our population could be a result of the high mean age of our study population. Elderly patients have a higher risk to be infected with these bacteria because hygiene status was lower in their youth. A reason for the protective effect of infection with *H. pylori* on vitamin B₁₂ deficiency in cardiovascular patients could be the way the bacteria works on the gastric mucosa. Apparently, more gastric acid is produced as a reaction of the stomach in the presence of the bacteria. Therefore, more vitamin B₁₂ could be released from its protein binding in the patient's diet. This leads to higher amounts of free vitamin B₁₂, and all available intrinsic factors will come into play in this situation.

Our study population used a lot of concomitant medication, which could lead to confounding in the associations that we studied. Nonsteroidal anti-inflammatory drugs are known to cause similar damage to the stomach as acetylsalicylic acid.¹³ The Dutch elderly population frequently used vitamin preparations, which resulted in a maximum daily vitamin intake.^{14,15} With use of a vitamin B preparation, a low daily dietary intake of vitamin B12 can be compensated.^{16,17} However, the number of patients using nonsteroidal anti-inflammatory drugs or vitamin preparations did not differ between vitamin B₁₂ deficient and nondeficient patients.

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Prevalence of Diabetes Mellitus and Related Conditions in Asian Indians Living in the United States

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This study is the first attempt to evaluate the prevalence of diabetes mellitus and related conditions in Asian Indians living in the United States. A community-based survey of 1,046 Asian Indian immigrants living in and around the Atlanta metro area of Georgia was conducted and found an overall prevalence of diabetes mellitus of 18.3% (22.5% in men and 13.6% in women). This prevalence of diabetes mellitus in Asian Indians is much higher than in whites, blacks, and Hispanics living in the

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Many studies have reported that Asian Indians have an unusually high prevalence of diabetes mellitus.^{1,2} This high prevalence of diabetes in migrant Asian Indians has been shown to be much higher than the population residing in India and is also higher than the other racial groups in the host countries.² Asian Indians now constitute 1% (1.9 million) of the United States population³ and are one of the fastest growing minority groups. No study has examined and compared the prevalence of diabetes mellitus in Asian Indians with other racial groups in the United States. This study examined the prevalence of diabetes mellitus and its association with other co-morbid medical conditions in Asian Indians living in Atlanta, Georgia.

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