(83.3%) with 4 vessels disease were *H. pylori* seropositive.(χ_2 = 2.616, df = 4, p = 0.624). There was no association between *H. pylori* infection and angiographic evidence of CAD. Out of the 207 patients who participated in this study, 166 patients (80.2%) were *Ch. pneumoniae* seronegative and 41 patients (19.8%) were *Ch. pneumoniae* seropositive. Fourteen of 66 patients (21.2%) with normal angiogram and 27 of 141 patients (19.1%) with abnormal angiogram were *Ch. pneumoniae* seropositive (p value with Fisher's exact test = 0.713, χ_2 = 0.120 df = 1, chi-square p = 0.729). Comparing the extent of CAD with *Ch. pneumoniae* showed that 5 of 45 patients (11.1%) with one vessel disease, 15 of 37 patients (40.5%) with 2 vessels disease, 6 of 53 patients (11.3%) with 3 vessels disease and 1 of 6 patients (16.7%) with 4 vessels disease were *Ch. pneumoniae* seropositive. (χ_2 = 14.678, df = 4, p = 0.005). There was no association between *Ch. pneumoniae* infection and angiographic evidence of CAD. In contrast to other studies we have not demonstrated an association between *H. pylori* and *Ch. pneumoniae* seropositivity and angiographic evidence of CAD.

Mendall et al[2] in a general practice-based case control study demonstrated an OR of 2.28 (95%, CI =1.25 - 4.15). In contrast, one meta-analysis of 18 studies involving more than 10000 people ,which was performed by Danesh and Peto,[6] showed no significant evidence of correlation between *H. pylori* infection and CAD. In addition, atherosclerosis risk in communities, who studied 15792 patients between 45-64 year-old ,after adjustment for age, gender and race demonstrated adjusted hazard ratio of 0.97 (95%, CI =0.52 - 1.78), which did not show any correlation between this infection and CAD .

This present study has 2 important advantages to other studies; one is the sufficient homogeneity of the social status of the patients. Most of the patients who participated in this study were from a low socioeconomic class with similar hygiene level. Their daily diets were also very similar. Another point in this study is that atherosclerosis was proven in the case group and rejected in the control group by angiography, which is a sensitive and specific technique in diagnosing CAD. There is no significant association between CAD and *H. pylori* and *Ch. pneumoniae* infection.

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Screening for glucose-6-phoshate dehydrogenase deficiency in Behçet's disease

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Glucose-6-phoshate dehydrogenase deficiency occurs with high frequency in the Middle East.[1] Iraq lies at the center of the Middle East and is well known to have relatively a high frequency of G6PD deficiency. Previous reports from Iraq have estimated the frequency of G6PD deficiency to be 8.9% or 12.4%.[1] However a more recent report using the methemoglobin reduction test has put the frequency at 6.3%.[1]

Another disease, which occurs in the Middle East and particularly in Iraq, is Behçet's disease.[2] Behçet's disease has a well-known association with certain genetic characteristics. Thus, the association between human leukocyte antigen (HLA) B51 and Behçet's disease is well known.[2] More recently an association with genetically controlled slow acetylator status was reported and this was found to be related to the disease severity and HLA B51.[2] Thus in this report we try to answer the question if there is any association between G6PD deficiency and Behçet's disease.

Forty-one Behçet's disease patients, 25 males and 16 females, with ages ranging from 19-46 years, were recruited in the study. Patients were registered

at Behçet's disease Clinic at Medical City Teaching Hospital, Baghdad, Iraq. All Behçet's disease patients fulfilled the International Study Group Criteria (ISGC) for the diagnosis of Behçet's disease.[3] Their informed consent was obtained. The ethical committee approved the study.

Thirty-seven healthy individuals, 19 males and 18 females, with ages ranging from 25-38 years, participated in the study. None of the participants had a history of serious illness and the findings of the physical examinations were normal. Written informed consent was obtained from all patients. Two ml of blood were withdrawn and put in a tube containing 0.3 ml of acid citrate dextrose from each subject or Behçet's disease patient. The methemoglobin reduction test was used to screen for G6PD deficiency.[4] Screening for G6PD deficiency in both Behçet's disease patients and normal control subjects using methemoglobin reduction test did not detect any case of G6PD deficiency. Therefore, we can conclude that there is no association between Behçet's disease and the genetic abnormality, which leads to G6PD deficiency. It may be argued that the sample of Behçet's disease patients is small. But we have to take into consideration that this number represents an adequate number for this uncommon disorder. In addition, the same number of patients was used to determine the 2 other genetically controlled traits, which are the frequency of HLA B51 and acetylation. Results of that parallel study showed that the frequency of patients with positive HLA B51 was 68.2%. Another genetically controlled trait studied in the same population was acetylation. It is well established that acetylation demonstrates genetic polymorphism; slow acetylators being homozygous for an autosomal recessive gene.[2] Results of the previous study showed that all Behçet's disease patients were slow acetylators.[2] In addition there was an association between the frequency of slow acetylators, positive HLA B51, and severity of Behçet's disease.[2] Therefore, the fact that this study failed to show an association between Behçet's disease and G6PD deficiency is significant. The frequency of G6PD deficiency in a large sample reported in a recent study using the methemoglobin reduction test was 6.3%.[1] The present report does not rule out that the frequency of G6PD deficiency in Behçet's disease is similar to that reported in the general population.

Dapsone is a drug well known to cause hemolysis in G6PD deficient patients. Dapsone has been used successfully in our department for the last 20 years in treatment of Behçet's disease with no incidence of major reported hemolysis.[5] This observation has been subsequently confirmed in a double blind controlled study.[5] The fact that Behcet's disease has no association with G6PD deficiency, it helps to explain why dapsone has been used safely.

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Abruptio placentae following snake bite in a Sudanese woman

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The fact that venomous snake bites during pregnancy result in a high fetal wastage and may cause maternal mortality makes this an important, albeit uncommonly encountered, entity in obstetrics.[1,2] Thus, it is essential that all emergency physicians become familiar with the recognition and treatment of venomous snake bites.

A 29-year-old Sudanese woman, gravida 4 para 3, full term with normal spontaneous vaginal deliveries at home were presented to the New Halfa Teaching Hospital, Sudan with snake bite in her right leg for 8 hours. She was in her 32 weeks gestation. The patient's pulse rate was 90 beats/minute, the blood pressure was 110/70 mm Hg and her temperature was 37.2ºC. No complain of abdominal pain; vaginal bleeding or hematuria and urine examination was free of microscopic hematuria. The patient's renal function's tests were within normal values. Ultrasound confirmed the gestational age and the fetal activity. The patient