Table 2 - Diagnosis of Hajj caravan patients according to ICD-10.

| Diagnosis | ICD-10 codes | n | (%) |
|---|---|---|---|
| Disease of musculoskeletal and connective tissue | M00-M99 | 8 | (24.2) |
| Disease of respiratory system | J00-J99 | 7 | (21.2) |
| Disease of nervous sytem | G00-G99 | 5 | (15.1) |
| Disease of gastroenteritis | K00-K93 | 5 | (15.1) |
| Cases of general surgery as hernia, amputation, acute abdomen and others | R00-R99 | 4 | (12.1) |
| Disease of blood and blood forming organs | D50-D89 | 2 | (6.1) |
| Disease of skin and soft tissue | L00-L99 | 2 | (6.1) |
| **Total** | | **33** | **(100)** |

good and 81.8% had acceptable condition. **Table 1** also highlights the duration of stay of patients before and after Hajj day showing that majority of patients 48.4% staying for 2-4 days until 9th of Dhu-Al-Hijjah but after 9th of Dhu-Al-Hijjah maximum patients 42.4% left the hospital on the same or next day. **Table 2** shows us the presentation of different diagnosis with their ICD-10 codes.[7] There were only 7 codes in which various diagnoses fall but the diagnosis of patients with musculoskeletal and connective tissue disorder were at higher rate 24. 2%.

Our study gives us a picture of how it was made possible for the ill to perform Hajj by the hospital management. Other studies regarding Hajj,[3-6] and more than this had been conducted in past but it is a different type of study ever done for the first time in the KSA. Our study indicates that the provided selection criteria is followed closely and no undue effects were experienced by allowing patients to attend Hajj. Moreover, hospital should carefully select patients who should be allowed to go with the Hajj caravan in order to avoid unnecessary morbidity and mortality.

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# Acetylator phenotype in Iraqi patients with discoid lupus erythematosus

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Lupus erythematosus (LE) is usually divided into 2 main types, discoid (DLE) and systemic (SLE). Discoid lupus erythematosus is a relatively benign disorder of the skin most frequently involving the face. There are hematological and serological changes in half of the patients.[1]

Polymorphic N-acetylation has been linked to variation in drug response, susceptibility to adverse reactions and increased incidence of certain spontaneous disorders including cancer.[2] The association between LE and acetylation has received much attention with conflicting results. While drug–induced lupus syndrome is more frequent in slow than rapid acetylators,[2] the association of spontaneous SLE with the slow acetylator status is controversial. Although some reports confirmed this association, other repots failed to find any association.[3] The association between DLE and acetylation has received little attention. There are only 2 reports that failed to show an association between acetylation and DLE.[4,5] The present paper examined the acetylator status in Iraqi DLE patients. Iraqi population as well as other Middle Eastern populations are characterized by a predominance of slow acetylators.[6] Therefore, it is interesting to

examine this problem in a predominantly slow acetylator population.

Twenty DLE patients and 30 healthy volunteers participated in the study. Approval to conduct this study was granted by the appropriate local ethical committee. The nature of the trial was explained to each subject and the consent of each was obtained. Excluded from this study were individuals with glucose-6-phosphate dehydrogenase deficiency or allergy to sulfonamides. Non of the subjects had a history of drug-induced lupus prior to phenotype determination, were receiving drugs that would interfere with acetylation, or were on any drugs known to be polymorphically N-acetylated. The study included a total of 20 patients with DLE who attended the Department of Dermatology, Baghdad Teaching Hospital, Medical City, Baghdad. Diagnosis of DLE patients was carried out by a specialist dermatologist and was based upon clinical and histological criteria and the absence of lupus related articular or visceral damage (gut, liver, or heart disease were excluded). Investigations included a complete blood picture and erythrocyte sedimentation rate, a general urine examination, anti-nuclear antibody titer, venereal disease research laboratories test, and anti double stranded deoxyribonucleic acid antibody. Patients included 8 (40%) males, and 12 (60%) females, whose ages ranged from 17-58 years, with mean 37.20±10.88. The age at the onset of the disease ranged from 7-48 years (mean 29.40±9.28). The mean duration of disease was 7.81 years (range 0.25-30). All patients had negative antinuclear antibodies. Thirty healthy volunteers were recruited, and all had no history of major illness and no abnormal physical findings during examination or investigation. Their age ranged from 16-52 years (mean 26.30±9.81). The group included 9 (30%) males and 21 (70%) females. After an overnight fast, each subject received a single oral 100mg of dapsone, obtained from (Al-Nile Company for Pharmaceuticals and Chemical Industries, Cairo, Egypt). Drinking of caffeine containing beverages was not allowed throughout the study period. A blood sample (5ml) was obtained 3 hours after drug intake by venepuncture that was added to a 10ml polyethylene tube containing 50µl of heparin (Heparin Leo 5000 iu/ml, Leo Pharmaceutical Products, Denmark). Plasma was separated within one hour after collection by centrifugation at 3000rpm for 10 minutes. The samples were subsequently stored frozen at -20°C pending analysis. A rapid, simple, one-stage protein precipitation method for the estimation of plasma dapsone (DDS) and monoacetyldapsone (MADDS) concentrations by a high performance liquid chromatography was used,[7] as was described in a previous study.[6] Statistical analyses were carried out, using SPSS software, version 10. Results were presented as mean±standard deviation, differences between groups were assessed by chi square test and an estimate was considered to be statistically significant if $p$ value was <0.05.[8]

The frequency of slow acetylators in DLE patients, whose MADDS and DDS ratios were <0.30, was 13 of 20 (65%), with a 95% confidence interval (CI) of 49.4-80.6%, (7, 53.8% females, and 6, 46.2% males). Their ages ranged from 17-58 years (mean 37.38±12.58), their ages at onset of the disease ranged from 7-48 years (mean 28.46±11.30), and the duration of disease ranged from 1-30 years (mean 8.92±9.53). The plasma concentrations of DDS ranged from 0.19-7.77 µg/ml (mean 1.83±2.12), and MADDS ranged from 0.01-1.20 µg/ml (mean 0.23±0.33). The plasma MADDS and DDS ratios ranged from 0.03-0.27 µg/ml (mean 0.12 ±0.07) **Table 1**.

The frequency of non-acetylators in DLE patients whose MADDS and DDS ratio are zero, was 7 of 20 (35%), (5, 71.4% females and 2, 28.6% males). Their ages ranged from 29-52 years (mean 36.86±7.63), their ages at onset of disease ranged from 27-36 years (mean 31.14 ± 3.39), and the duration of the disease ranged from 0.25-20 years (mean 5.75±7.03). The plasma concentrations of DDS ranged from 0.49-2.66 µg/ml (mean 1.51±0.92 µg/ml). The plasma concentrations of MADDS were zero. There were no rapid acetylators in DLE patients.

The frequency distribution of the plasma MADDS and DDS ratio in 30 healthy volunteers (control) revealed that the frequency of slow acetylators whose MADDS and DDS ratios were <0.30 was 22 (73.3%) of 30 with a 95% CI of 59.9-86.7%. They included 17 (77.3%) females and

Table 1 - Frequency distribution of acetylator phenotype in lupus patients and healthy controls with their statistical significance.

| Acetylator phenotype | Subjects n | Frequency % |
|---|---|---|
| ***None*** | | |
| DLE | 7 | 35 |
| Control | 0 | 0 |
| ***Slow**** | | |
| DLE | 13 | 65 |
| Control | 22 | 73.33 |
| ***Rapid*** | | |
| DLE | 0 | 0 |
| Control | 8 | 26.66 |

DLE - discoid erythematosus lupus, * Not significant compared to control *p=0.28*

5 (22.7%) males, their ages ranged from 16-52 years, (mean 27.86%±10.74). Their plasma MADDS and DDS ratios were from 0.01-0.28 µg/ml (mean 0.11±0.088). While the frequency of rapid acetylators in control whose MADDS and DDS ratios were >0.30 were 8 (26.7%) of 30. They included 4 (50.0%) females and 4 (50%) males. Their ages ranged from 16-29 years, (mean 22.00±4.87). Their plasma MADDS and DDS ratios were from 0.36-1.63 µg/ml (mean 0.86±0.57). There were no non-acetylators in healthy controls (**Table 1**). The slow acetylators in the DLE patients, as a group, was not significantly different from the slow acetylators in the control group ($p>0.05$).

In this study, patients with DLE were shown to be either slow acetylators or non-acetylators, that is the plasma concentration of MADDS was undetected in plasma. In this respect, the results of this study differs from the 2 previous reports from the European countries which concluded that the frequency of slow acetylators was not different between DLE patients and normal control group.[4,5] In a parallel study, the incidence of slow acetylators in patients with spontaneous SLE was reported not different from that found in the control group.[3] Taken together, the 2 studies about the acetylator phenotype in SLE and DLE support the held view that the 2 diseases do not represent a spectrum but can be considered to be overlapping diseases with different etiologies.[1] An interesting finding in this study was that some patients were non-acetylator. In a previous report about half of the patients with Behcets' disease were found to be non-acetylators.[6] This finding can not be explained by a technical error in the method used since non-acetylator in this study as well as the previous were found in the patients and not in the control group. On both occasions patient and control samples were run together. The significance of this finding, needs further investigation to determine the genotype of non-acetylators, in order to understand this phenomena.

In conclusion, in a population of slow acetylators, it appears that slow or very slow acetylators phenotype can be considered as genetic trait predisposing to the development of DLE. This finding as well as the occurrence of non-acetylators needs further investigation to determine the genotype in a lager sample.

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# Risk factors of coronary heart disease among Jordanians

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Coronary heart disease (CHD) is the first leading cause of death in developed countries. According to Ministries of health statistics, it was also found to have a significant prevalence in Jordan and the rest of the Arabic countries.[1] Prevention is of primary importance, and proper prevention requires correction of risk factors in persons at high risk. This will significantly lower the mortality rate, which reduces the economic burden resulting from stroke on patients' families and health service organizations. Numerous surveys and epidemiological studies revealed the major risk of coronary heart disease (CHD), which included diabetes, low-density lipoprotein-cholesterol (LDL-C), hypertension (HTN), smoking, inflammatory diseases and others, which could be managed effectively.[2] However, the relative significance of these factors remains controversial among many publications.[3] Nevertheless, it was well established that some environmental, genetic and ethnic factors played a significant role in these controversial conclusions.

We have conducted this study, which retrospectively involved a total of 201 patients who underwent catheterization at the Islamic and Ibn-Al-Hytham Hospitals, Amman, Jordan, in the period between 1995 and 2000. Experienced