Table 1 - Correlation of cefoxitin resistance among methicillin resistant and methicillin susceptible *Staphylococcus aureus*.

***Staphylococcus aureus* strains**	**Fox S/SOA Negative**	**Fox R/SOA Positive**	**Fox R/SOA Negative**	**Total**
MSSA	436	Nil	Nil	**436 (100)**
MRSA	Nil	108	1	**109 (100)**
Total	**436**	**108**	**1**	**545**

MSSA - *methicillin sensitive Staphylococcus aureus,*
MRSA - *methicillin resistant Staphylococcus aureus*, Fox - Cefoxitin, S - Susceptible, SOA - Salt Oxacillin agar, R - resistant

confirmatory test was also cefoxitin resistant (**Table 1**). Cefoxitin a cephamycin antibiotic is a very potent inducer of mecA expression; hence, resistant to it can be used as a predictor for the presence of mecA gene in MRSA strains.[4] Previous studies, described high sensitivity and specificity of cefoxitin resistance with the disk diffusion method in confirmed MRSA strains.[4-6] In the present study, cefoxitin resistance by disc diffusion method among MRSA strains had a sensitivity and specificity of 100%. Inclusion of cefoxitin **(**30 µg**)** disk in the routine sensitivity plates for *Staphylococcus aureus* appears to be a very simple, sensitive, specific, and cost effective method for confirmation of MRSA strains. The clinical laboratories, particularly in the developing regions of the world with over stretched health care budget, and limited recourses at their disposal, cannot afford the expensive molecular biological techniques for confirmation of MRSA. Testing of these strains routinely for cefoxitin resistance by disk diffusion test appears to be a better predictor of mecA mediated resistance in *Staphylococcus aureus*. This will not only be a cost effective, highly specific and sensitive test, but also the MRSA confirmation report can be made available to the clinicians in a much shorter time and can be very helpful in better management of the patient by timely prescribing the specific antimicrobial therapy, and implementation of strict infection control measures to control the spread of MRSA in the hospital.

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From the Department of Infection Control, Internal Medicine, Surgey, King Fahad Hospital and Tertiary Care Center, Al-Hofuf, Al-Hasa, Kingdom of Saudi Arabia. Address correspondence and reprint requests to Dr. Bodh R. Panhotra Consultant Microbiology and Infection Control Officer, King Fahad Hospital, Al-Hofuf, Al-Hasa 31982, Kingdom of Saudi Arabia. Tel. +966 (3) 5750000. Fax. +966 (3) 5752255. E-mail: drpanhotra2000@yahoo.co.in/ drpanhotra30@hotmail.com

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Role of maternal factors in the etiology of neural tube defects in Jordan

Amira T. Masri, *MD.*

Neural tube defects (NTDs) are among the most common human congenital malformations. Jordan lacks an ongoing surveillance system for congenital anomalies, however, the incidence of NTD in Jordan is high; 1.6 per 1,000 live births based on one study carried out in the north of Jordan.[1] The exact cause of these defects is not known, however, they are currently considered to be "complex" genetic disorders with both genetic, and environmental factors play an important role in their causation. Nutritional factors appear to be an important contributor to the etiology of NTDs. Several other maternal factors have also been established to be contributory to this risk, including socioeconomic status, various environmental factors, genetic factors, maternal illness, and medication during pregnancy, maternal age and reproductive history.[2] The risk of one child having spinal dysraphism is estimated at 0.1-0.2%, but with one affected sibling the risk of a second affected

child increases to 2-5%, and the risk of a third affected child increases again to 10-15%.[2] A small percentage of birth defects is preventable, hence, knowledge of any additive factor that increases the risk for a common birth defect, such as NTDs is relevant, and might help in planning preventive strategies. To identify whether risk factors associated with NTD in our population are similar to those in other populations or added factors play a role, this case control study was carried out at Jordan Univerisity Hospital using our patient population for the past 10 years. The charts of all patients hospitalized with NTD over a 10-year period between January 1993 and December 2002 were reviewed. A control group of 227 healthy live births born during the same period were also included for the chart review. It is noteworthy that for every patient 2 controls or more were chosen for the review. Controls were chosen from the same year as the patients. The charts were reviewed for maternal age, parity, birth order, previous abortions, previously malformed newborns, maternal illnesses, and drug history during pregnancy. The controls were reviewed in a similar way. Forty-five patients with NTD were included in the study. The average maternal age at birth in the study group was 27.34 years, while it was 28.45 years in the controls. There was no significant difference between the 2 groups (p=0.11). In addition, there was no significant difference in the 2 groups when further subdivided by age. Spontaneous abortions were reported in 47.5% of NTD mothers compared with 27.9% of the controls, and the difference between the 2 groups was significant (p<0.05). Regarding parity, 31.7% of mothers of NTD children were primiparous compared with 20.4% in the controls. While there was no significant difference, a trend towards correlation with primipara was noted, 31.7% versus 20.4%. In addition, neither birth order, nor maternal illness nor drug history was significant in this study.

The NTD rates vary from one population to another, and have also been found to vary by geography, time, maternal demographic characteristics, and maternal reproductive history. The effect of maternal age as a risk factor for NTDs is generally considered to be small, and when an association can be found, risk tends to be elevated in older or very young mothers.[2] In our group of patients, we failed to elicit such a role for maternal age. A history of spontaneous abortion was reported in 47.5% of NTD mothers compared with 27.9% in the controls, and this showed a significant relation (p=0.01). An association between NTDs, and previous spontaneous abortions has been noted in several studies,[3] and there are several suggested hypotheses for the explanation of this observation; Firstly, the trophoblastic cell rest theory, the remaining from a previous aborted pregnancy interferes with normal embryogenesis and secondly, previously lost fetus was affected with NTD,[3] however, genetic factors might also be incriminated as well. Some birth defects can be associated with higher birth order, but in this study, there was no correlation between birth ordered and risk of NTD. Some authors reported a correlation between multiparity and NTD[2,3] others have shown both a "modest risk in mothers of parity 3 or more" and an increased risk in primiparous mothers.[2] While there was no significant difference in our study, a trend towards correlation with primipara was noted. Maternal illness during pregnancy has also been considered a risk factor. Pregestational diabetes increases the risk of having a child with a malformation in the central nervous system including spina bifida to 2-10 fold higher than the risk in the general population, in addition a febrile illness in the first trimester has been associated with a 2-fold increase in the risk.[4] In utero exposure to drugs such as valproic acid, and carbamazepine increase the risk of spina bifida to 1-2%. In this study neither maternal illness nor drug history was significant. Controlled interventional studies have clearly shown a 72% reduction in the risk of NTDs with the use of folic acid dietary supplements,[5] and based on the results of this study periconceptional supplementation with folic acid might be useful in reducing the risk of NTD in mothers with high risk history.

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From the Department of Pediatrics, Division of Child Neurology, Jordan University Hospital, Amman, Jordan. Address correspondence and reprint requests to Dr. Amira Masri, PO Box 1612, Code 11941, Amman, Jordan. Tel. +962 (6) 5332477. E-mail: masriamira69@hotmail.com

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