Health Hazards Arising from Home-Prepared Foods Available in the Markets of the Gulf

Sir,

In many developing countries, some foods available in the market are prepared at home. This practice may lead to health hazards especially with ineffective food control systems. Many foodborne diseases may spread due to such a practice. In the Arabian Gulf countries, such as Bahrain, Qatar and United Arab Emirates two kinds of home-prepared fermented fish sauces are available in local markets; namely Mehiawah and Tareeh. These fish sauces are made by a traditional process handed from generation to generation. In general the sauces are made from dried Indian sardine, salt, spices and water, and kept in a container for about one week for fermentation. The two products are usually sold in glass bottles.

An attempt was made to examine the microbiological quality of these products. Five samples, three of Tareeh and two of Mehiawah were obtained from homes and from the local market in Bahrain, respectively. The products were tested for total plate count, Staphylococcus aureus and Salmonella count as described in AOAC. 

The results revealed that the total plate count was high in all fish sauces studied. Staphylococcus aureus were detected in three samples and ranged from 10 to $3.1 \times 10^6$ colony forming units/ml (CFU/ml), while Salmonella was not detected in any of the samples (Table 1). It can be concluded that marketing of home-prepared fish sauces should not be encouraged. Outbreaks of food poisoning due to Staphylococcus aureus are mainly caused by improper hygienic practices which result in food being contaminated by food handlers such as sneezing over it, contact with septic cuts, or from unhealthy carriers. It is well documented that Staphylococcus is found in the nose, throats, ears and on the hands of many people.

Standards and regulations for fermented fish sauces should be established as soon as possible. This will help those interested in producing such food commercially.

Further in-depth studies of the microbiological and chemical quality of various foods prepared at home and available in the markets of the region should be encouraged.

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References

Stevens-Johnson Syndrome Secondary to Pyrimethamine Sulphadoxine

Sir,

Fansidar® (Roche), is a combination of 500 mg sulphadoxine $N^2$(5,6-dimethoxy-4-pyrimidinyl)-sulphanilamide, and 25 mg pyrimethamine: 2,4-diamino-5-(p-chlorophenyl)-6-ethylypyrimidine, in one tablet. It is indicated mainly for the treatment and prophylaxis of plasmodium falciparum malaria, in chloroquine-resistant areas. This drug is given here, in one single dose (two tablets) to newly recruited people from such areas. We wish to call attention to a case of Stevens-Johnson syndrome (SJS) complicating a single oral dose of this drug, in an Indonesian woman (30-year-old), recently recruited to Dammam. She was admitted to Dammam Central Hospital, with acute onset of fever, generalized haemorrhagic skin rash with bullous eruptions, and conjunctival haemorrhage, with severe constitutional symptoms.

Management and follow-up included dressing of her lesions, antibiotics by the intravenous route (gentamicin, and clindamycin), and one litre of blood, in addition to supportive therapy. Corticosteroids were avoided since the condition was already established and to avoid exposing the patient to uncontrollable infection to which she was already susceptible. So our policy was based on the opinion that corticosteroids if they were not given very early, would cause more harm than benefit. Our patient was discharged completely cured, without any complications after 32 days. Our diagnosis (SJS) was first made by clinical history, by exclusions, by exhaustive investigations and was verified by the drug monitoring services of the local poison control centre. The term SJS, is given to Erythema multiforme exudativum, Ectodermosis.

Table 1

Results of bacteriological examination of two fermented fish sauces

<table>
<thead>
<tr>
<th>Food sample</th>
<th>SPC CFU/ml</th>
<th>Staphylococcus CFU/100 ml</th>
<th>Salmonella CFU/100 ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tareeh 1</td>
<td>$4.4 \times 10^4$</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Tareeh 2</td>
<td>$5.0 \times 10^3$</td>
<td>10</td>
<td>ND</td>
</tr>
<tr>
<td>Tareeh 3</td>
<td>$6.5 \times 10^6$</td>
<td>$3.1 \times 10^6$</td>
<td>ND</td>
</tr>
<tr>
<td>Mehiawah 1</td>
<td>$1.3 \times 10^5$</td>
<td>$1.1 \times 10^5$</td>
<td>ND</td>
</tr>
<tr>
<td>Mehiawah 2</td>
<td>$1.3 \times 10^6$</td>
<td>ND</td>
<td>ND</td>
</tr>
</tbody>
</table>

ND = Not detected.
SPC = Standard aerobic plate count.
CFU = Colony forming units.
Neutropenia Related to Captopril

Siri,

In a recent issue of your journal (Saudi Med J 1989; 10(2): 111-112) Drs E. S. Husain and M. J. Akhtar described two patients who developed neutropenia while receiving captopril therapy. In general, cases of drug induced myelosuppression can be divided into three types:

1. Predictable and dose-dependent, as seen after administration of alkylating agents and other chemotherapeutic drugs.
2. Unpredictable but partially dose-dependent, as seen after administration of phenothiazines.
3. Immuneologic, idiosyncratic and dose-independent, as seen after administration of a variety of unrelated drugs.

The unpredictability of the type 2 reaction may rest on the fact that the metabolism and detoxification of certain drugs are individually determined by genetic factors, such as capacity to methylate, acetylate, or glucuronidate, and clinical factors such as renal failure.

Captopril apparently does not cause a type 1 myelosuppression, and not surprisingly, the mere addition of captopril to culture of normal myeloid stem cells (CUNM) in vitro has failed to disclose direct stem-cell cytotoxicity. However, there is evidence supporting both type 2 and 3 reactions with angiotensin-converting enzyme inhibitors (class-related side-effects). Neutropenia has been reported with both captopril and enalapril in a similar frequency.

In the first case which was referred to by Drs Husain and Akhtar, the authors did not point out that both glibenclamide and procainamide therapy have been associated with the development of various haematologic reactions, e.g. agranulocytosis, leucopenia, haemolytic anaemia, aplastic anaemia and pancytopenia. More than 75% of heart failure patients who developed neutropenia on captopril therapy were receiving procainamide.

As pointed out by the authors, neutropenia with captopril occurs almost exclusively in patients with impaired renal elimination due to kidney failure or in patients with collagen vascular disorders. Therefore, the second patient described by the author was a high-risk patient. However, according to the FDA approved product-prescribing information, the incidence of neutropenia is 3.7% if renal failure and collagen vascular diseases co-exist, thus the percentage mentioned in the report of 7.2% needs to be updated.

Finally, we would like to stress the final statement in the article namely ‘Patients on captopril therapy should be advised to contact their doctor as soon as they develop fever or sore throat’. This is in line with captopril’s package insert and is derived from extensive experience with the drug. It is obvious that at least patient No. 1 did not abide by this rule as she had a fever of 3 days duration prior to her being readmitted on 5 January 1986.

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References
References


