It looked like a myocardial infarction after eating crawfish…Ever heard of Haff disease?

If you diagnose a case let us know…we need to identify the toxin.

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In April, a 26 year old female ate boiled crawfish that she purchased at a seafood restaurant/market. She ate at 1:00 p.m. By 5:00 she had nausea, vomiting, chest pain, shortness of breath, and profuse sweating. By 7:00 she went to the local hospital emergency room and was hospitalized. She had been in good health, took no drugs, and drinks very moderately. The physical examination was normal and a complete evaluation for myocardial infarction was negative. The only abnormality was a level of creatine phospho kinase (CPK) of 1024, (normal = 0-162) and the diagnosis was rhabdomyolysis of undetermined origin. By 11:00 p.m. her CPK rose to 8670. The following day she felt better and was discharged. Two days later her CPK was 379.

Two days after the initial case another woman, 33, ate crawfish from the same establishment at 8:00 p.m. By 11:00 she had severe stomach cramps, chest and back pain, nausea, hematuria. At 1:00 a.m. she was admitted to a local hospital for chest pain and shortness of breath. Her physical exam was normal and there was no evidence of myocardial infarction. Her initial CPK was 884, the following day it rose to 8600, and then dropped to almost normal a day later.

Within seven days of the first case, there were seven additional people who were hospitalized with similar histories of chest pain, shortness of breath, nausea, and sweating. All had a history eating crawfish purchased at the same establishment and developed symptoms within 3 to 16 hours (mean 8 hours) of their meals. Five were hospitalized and recovered swiftly without sequelae. All had a rise in CPK to 6000-8000 which returned to normal in a few days.

The cases all lived in several small towns within a 30 mile radius. Several controls who also ate crawfish did not get sick. Inquiries into other hospitals in the area did not reveal any additional cases.

The Office of Public Health conducted an environmental and epidemiologic investigations. The environmental inspection did not indicate any violations or evidence of cross contamination with chemicals or pesticides, and food preparation was deemed satisfactory. Crawfish samples were submitted to the Office of Public Health laboratory for analysis of heavy metals, pesticides and herbicides. All results were negative.

Creatine phosphokinase is an enzyme that is found in all muscle cells (cardiac and other muscles). High CPK in the blood means “damaged muscle cells”. The enzymes more specific to the heart muscle (CK MB and troponin) were normal in all cases. Causes of rhabdomyolysis are numerous: vigorous exercise, certain drugs such as lipid lowering drugs, some herbal teas, substance abuse (cocaine, ecstasy, alcohol), snake bites, bee stings and some infectious diseases. None of these cases had any of these risk factors. Other suspected causes include a long list of metals and metalloids (copper, arsenic, chromium, barium…) and some pesticides, fungicides or herbicides.

A bibliographic review indicates that this outbreak is characteristic of Haff disease. A review of Haff disease was published in Emerging Infectious Diseases, Volume 6 No. 2/March - April 2000, Dispatches Haff Disease: From the Baltic Sea to the U.S. Shore. Udo Buchholz, Eric Mouzin et al.

In 1924, an outbreak of sudden, severe muscular pain and rigidity (often with dark urine) was described near the Königsberger Haff shores along the Baltic coast. Most patients survived without sequelae but a few died. During the next decade some 1,000 cases occurred in similar small outbreaks. The cases all had recently consumed cooked fish (turbot, eel, pike). Seabirds and cats also seemed to have been affected. Several hypotheses were considered, particularly arsenic poisoning, however all were disproven.

In the U.S. there have been very few cases reported:
- 1984: 2 cases from Texas
- 1985: 2 cases from California
- 1986: 2 cases from California
- 1997: 4 cases from California
  2 cases from Missouri

(Continue on next page)
The 1997 US cases were defined as an illness in a person with unexplained rhabdomyolysis who had eaten fish within 24 hours of onset of symptoms. All ate buffalo fish that originated from Louisiana and Missouri. Buffalo fish (Ictiobus cyprinellus) is a bottom-feeding freshwater fish similar to carp. The laboratory marker used to define rhabdomyolysis is a fivefold or greater elevation in creatine kinase (CK) levels, with a muscle/brain (MB) fraction <5%. The same definition applies to the recent Louisiana cases except for a history of crawfish consumption.

Samples of food were recovered and analyzed (Buchholz U, 2000). Recovered leftovers and uncooked fish from the same lot were tested for the toxin of ciguatera or saxitoxin, the toxin of paralytic shellfish poisoning, and the toxins of blue-green algae. To characterize the physicochemical properties of the toxin, extract from both cooked and uncooked fish was partitioned into water-soluble, nonpolar lipid (hexane) and polar lipid (chloroform) fractions. These fractions were then administered intraperitoneally and orally to laboratory mice. Fractions causing toxicity to mice were further analyzed for identification of the toxin.

The origin of the buffalo fish eaten by four of the patients was traced to the same wholesaler in Louisiana who receives fish from approximately 25 fishermen who fish rivers in Louisiana. No fish or unusual animal die-off was noted in the areas where buffalo fish were caught.

The next step in the current investigation is to identify the toxin and find the source. This is a long and complicated process. Samples of suspected food will be sent to the FDA laboratory for further identification.

**Bulletin**

Notice: Varicella vaccination will be required for all first time school enterers and child care attendees beginning in the fall of 2003.

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**Newborn Screening for Congenital Hypothyroidism, LA, 1988-1999**

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Between January 1, 1988 and December 31, 1999 there were 827,863 newborns in Louisiana that were tested for congenital hypothyroidism (CH). One hundred thirty (130) infants were diagnosed with congenital hypothyroidism. This represents an incidence of 1.6 per 10,000 births or 1 in 6,250 births. The rate of congenital hypothyroidism is slightly higher in whites vs non-whites (1.5 vs 1.3/100,000 live births), a pattern similar to the U.S. rates.

The number of cases reported ranged from 7 to 15 per year with no significant trend. The total number of births has steadily decreased from 73,887 in 1988 to 67,034 in 1999. The incidence of CH per 10,000 newborns ranged from 1.1 to 2.2 (Figure 1). Sixty percent of the cases reported from 1990-1999 were females. White females had the most cases (48), followed by white males (24), non-white males (19), and non-white females (17; Figure 2).

Newborn screening for CH is done on all newborns using “filter paper” blood specimens. This screening for CH constitutes a major progress in early detection of a treatable condition, thereby preventing mental retardation. Screening in the DHH-OPH State Laboratory is done by assay of the thyroxine levels (T4). Then thyroid-stimulating hormone (TSH) levels are obtained for specimens with T4 results in the lowest 12% of the daily batch. Screening for CH is now also done in all fifty states; private laboratories performing screening tests must report positive results to the Office of Public Health.

In Louisiana this condition is much more common in newborn girls than in newborn boys, which has also been reported in the

*Figure 1: Cases of Congenital Hypothyroidism, Louisiana 1988-1999*