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Rhabdomyolysis After Cooked Seafood Consumption (Haff Disease) in the United States vs China

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Background: Haff disease is a syndrome of myalgia and rhabdomyolysis that occurs after eating cooked seafood.
Methods: For this descriptive analytical article, a literature search identified the scientific articles on Haff disease and/or rhabdomyolysis after eating cooked seafood in the United States and China. Analysis of those articles focused on identifying the seafood vectors of Haff disease, describing the most commonly recurring clinical and laboratory manifestations of Haff disease, and comparing the Haff disease toxidrome with other similar seafood-borne toxidromes. Statistically significant differences were determined using unpaired *t* tests and Fisher exact tests.
Results: Twenty-nine confirmed cases of Haff disease were identified in the United States, and 60 cases were identified in China during 1984-2014. Most of the US cases followed consumption of buffalo fish, and most of the Chinese cases followed consumption of freshwater pomfret. However, Haff disease also followed consumption of the same species of boiled crayfish (*Procambarus clarkii)* in the United States (n=9) and China (n=6). US patients with crayfish-transmitted Haff disease reported significantly more nausea with and without vomiting, chest pain, body and back pain, dyspnea, and diaphoresis than the Chinese patients and were more frequently misdiagnosed as having myocardial infarctions.
Conclusion: The bioaccumulation of a new, heat-stable freshwater and/or brackish/saltwater algal toxin, similar to palytoxin but primarily myotoxic and not neurotoxic, is suspected of causing Haff disease. At present, only the rapid identification of the seafood vectors of Haff disease will limit disease outbreaks and prevent further cases.

Keywords: *Neurotoxins, rhabdomyolysis, seafood*

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INTRODUCTION

Although the health benefits from polyunsaturated fatty acid–rich seafood include protection from myocardial infarction and stroke, fish and shellfish can bioconcentrate algal toxins and cause cluster outbreaks of seafood poisoning. Haff disease is a syndrome characterized by myalgia often accompanied by myoglobinuria after the ingestion of cooked fish and crustaceans.[1-7] Haff disease targets and damages striated muscle and causes rhabdomyolysis with the release of myoglobin, creatine kinase (CK), and other muscle cell enzymes. The proposed mechanisms for acute renal failure in patients with rhabdomyolysis include the following: (1) blockage of the renal tubules with precipitated myoglobin, (2) cortical damage from oxygen free radicals, and (3) myoglobin-induced vasoconstriction of renal arterioles. The differential diagnosis of Haff disease includes other causes of seafood poisoning by heat-stable toxins—typically bioaccumulated red algal and blue-green algal toxins.[8,9]

Haff disease is named for the Königsberg Haff, or lagoon, that borders the Baltic coasts of Russia and Poland, the site of the first reports of the disease in 1924.[9] The US Centers for Disease Control and Prevention (CDC) defines Haff disease (also known as Haff-Iuksov-Sartlan disease and Iuksovsk-Sartlansk disease in Eastern Europe) as a painful myotoxic condition that meets the following criteria: (1) a positive cooked seafood ingestion history within 24 hours, (2) a markedly elevated (specifically a 5-fold or greater rise over normal) serum CK level, and (3) a CK-muscle/brain (MB) fraction <5%.[8,9]

The objectives of this retrospective case series analysis were (1) to identify the most common seafood vectors of Haff disease in the United States and China; (2) to describe and to compare the most commonly recurring clinical and laboratory manifestations of Haff disease following crayfish and fish consumption in the United States and China—the two nations reporting the greatest number of Haff disease cases meeting the current CDC disease definition; and (3) to compare the Haff disease toxidrome with similar seafood poisoning toxidromes to further define the pathophysiologic mechanisms of poisoning and to expose potential causative aquatic toxins.

METHODS

Search engines including PubMed, MEDLINE, OLDMEDLINE, Ovid, Google, and Google Scholar were queried for

Table 1. Demographic and Epidemiologic Features of Haff Disease Cases, United States vs China, 1984-2014

Demographic or Epidemiologic Feature	United States Cases n=27[a]	Chinese Cases n=54[a]	*P* Value
Male	33%	67%	0.035
Female	67%	33%	0.035
Age range, years	26-87	4-74	Not applicable
Mean age ± SD, years	55 ± 19	42 ± 20	0.028
Median incubation period, hours	8	7	0.062
Median incubation period range, hours	3-21	0.10-41	Not applicable
Case fatality rate	3.7%	0	1.000

Statistically significant difference is $P<0.05$.
[a]Complete demographic and epidemiologic data were only available for 27 of 29 cases of Haff disease in the United States and for 54 of 60 cases of Haff disease in China during the reporting period. Cases with incomplete demographic and epidemiologic data (United States=2, China=6) were excluded from this descriptive comparison.

the period 1946-2014 with the keywords Haff disease, seafood poisoning, and rhabdomyolysis to identify all scientific articles on Haff disease and/or rhabdomyolysis after cooked seafood consumption. The CDC definition was applied in this investigation and excluded all other causes of rhabdomyolysis.

Of the 23 references identified, 11 were selected for a comparative analysis of Haff disease cases in the United States (8 articles)[8-15] and in China (3 articles).[16-18] The data extracted from these articles were stratified initially by the 2 reporting nations, the United States and China. Additional stratified data included implicated seafood vectors in the 2 reporting nations, confirmed cases meeting the CDC disease definition, demographic and epidemiologic features, presenting clinical manifestations, and laboratory biomarkers of rhabdomyolysis. Because this investigation was an analysis of previously reported cases, institutional review board approval was not required.

Continuous variables are reported as means with standard deviations, and categorical values are reported as counts or proportions (%). Statistically significant differences, defined by P values <0.05, were determined using unpaired two-tailed t tests for analytically comparing continuous variables and two-tailed Fisher exact tests for analytically comparing categorical variables. The unpaired t tests were accompanied by effect size calculations reported as standardized mean differences bracketed by 95% confidence intervals. Effect size calculations are not applied to Fisher exact tests because of small sample sizes of 0-5 observations per cell.

RESULTS

Most Common Seafood Vectors

More than 1,000 cases of Haff disease were initially described in Eastern Europe and Sweden during the period 1924-1984 after the consumption of several species of cooked freshwater fish including burbot (*Lota lota*), pike (*Esox* spp), freshwater eel (*Anguilla anguilla*), and whitefish (*Coregonus* spp).[1-7] These cases were characterized by muscle pain and tenderness and evidence of rhabdomyolysis with reddish-brown or black urine (myoglobinuria).[1-7] The first cases of Haff disease in the United States were reported in Texas in 1984 following the consumption of cooked freshwater bigmouth buffalo fish (*Ictiobus cyprinellus*).[8,9] In 2001, two more cases of Haff disease were confirmed in North Carolina following the consumption of baked Atlantic salmon (*Salmo salar*).[10] Huang et al reported 54 confirmed cases of Haff disease following the consumption of freshwater pomfret in China.[16] In addition to cases following cooked fish consumption, Haff disease clusters have been reported following the consumption of boiled crayfish (*Procambarus clarkii)* in Louisiana (2001) and China (2011-2013).[11-15,17-19]

Twenty-nine cases of Haff disease have been reported in the United States, and 60 cases have been reported in China during 1984-2014.[8-19] Most of the US (n=27) and all of the Chinese (n=60) cases met the CDC case definition of Haff disease and followed the consumption of bigmouth buffalo fish (*I. cyprinellus*, n=16; 2 cases reported from 1984 were described and not confirmed) in the United States, crayfish (*P. clarkii*) in the United States (n=9) and China (n=6), freshwater pomfret in China (n=54), and Atlantic salmon (*S. salar*, n=2) in the United States.

United States vs China Comparisons

Table 1 compares the demographic and epidemiologic features reported for Haff disease cases following fish and crayfish consumption in the United States (n=27) and fish consumption China (n=54) during the reporting period, 1984-2014. Significantly more females were identified among US cases and more males among Chinese cases. US patients were significantly older than Chinese patients. No Chinese fatalities were reported. However, one US patient died from acute intracranial hemorrhage, an elderly female who consumed cooked buffalo fish and rapidly developed rhabdomyolysis with acute renal failure (case fatality rate=3.7%).[13] No statistically significant difference was seen in the median incubation periods reported (range, 7-8 hours) between the US and Chinese cases following toxic fish and crayfish ingestion.

Table 2 compares the initially reported presenting signs and symptoms of Haff disease cases following cooked fish consumption in the United States (n=20) and China (n=54). Significantly more myalgia, stomach pain, diarrhea, dizziness, and dry mouth were reported by Chinese patients,

Table 2. Initial Presenting Signs and Symptoms of Haff Disease Following Cooked Fish Consumption in the United States and China, 1984-2014

Sign or Symptom	United States Cases n=20[a]	Chinese Cases n=54[a]	P Value
Myalgia	9	46	0.010
Nausea ± vomiting	14	34	0.299
Stomach pain	0	34	<0.0001
Diarrhea	0	18	0.031
Dizziness	0	18	0.031
Headache	0	12	0.166
Dry mouth	0	25	0.002
Chest pain	4	5	0.220
Dyspnea	0	1	0.445
Muscle stiffness	6	1	0.001
Pain on light touch (allodynia)	6	5	0.026
Back and body pain	5	5	0.112
Brown urine/hematuria	2	0	0.156
Thigh and body numbness	3	0	0.048[b]
Diaphoresis	0	0	

Statistically significant difference is $P<0.05$.
[a]Complete clinical data were available for 20 cases of Haff disease in the United States following cooked fish consumption (buffalo fish consumption=18, Atlantic salmon consumption=2). Complete clinical data were available for 54 cases of Haff disease in China following cooked fish consumption (freshwater pomfret=54).
[b]Barely reached significance in a small sample.

Table 3. Initial Presenting Signs and Symptoms of Haff Disease Following Boiled Crayfish Consumption in the United States and China, 1984-2014

Sign or Symptom	United States Cases n=9	Chinese Cases n=6	P Value
Myalgia	9	6	1.000
Nausea ± vomiting	9	0	0.004
Stomach pain	9	6	1.000
Chest pain	9	5	1.000
Dyspnea	9	1	0.008
Muscle stiffness	6	1	0.119
Pain on light touch (allodynia)	0	5	0.011
Diaphoresis	9	0	0.004
Back and body pain	9	5	1.000
Brown urine/hematuria	1	0	1.000
Thigh and body numbness	0	2	0.518
Diarrhea	0	0	
Dizziness	0	0	
Headache	0	0	
Dry mouth	0	0	

Statistically significant difference is $P<0.05$.

and significantly more muscle stiffness and allodynia were reported by US patients.

Table 3 compares the initial presenting signs and symptoms of Haff disease cases following the consumption of boiled crayfish of the same species (*P. clarkii*) in the United States (n=9) and in China (n=6). Despite the consumption of the same species of crayfish (*P. clarkii)* in the United States and China, patients with crayfish-transmitted Haff disease in the United States reported significantly more nausea with and without vomiting, chest pain, body and back pain, dyspnea, and diaphoresis than Chinese patients and were more frequently misdiagnosed as having myocardial infarctions.[11,19] Chinese patients with autochthonous crayfish-transmitted Haff disease reported significantly more allodynic pain on light touch than US patients.[17,18]

Table 4 compares the laboratory biomarkers of rhabdomyolysis of Haff disease cases following cooked fish and crayfish consumption in the United States (n=23) and following cooked freshwater pomfret consumption in China (n=54). Initial and overall mean CK levels were significantly higher in US patients than in Chinese patients, and the mean CK-MB fraction was significantly higher in Chinese patients and slightly exceeded the CDC case definition (6.0% ± 5.4%, $P=0.005$).

Comparison of Toxidromes

Following a Haff disease cluster in Louisiana in 2001, Krishna and Wood suggested that buffalo fish and crayfish consumed the roots of water hemlock (*Cicuta maculata*) plants growing in marshy areas along shorelines.[19] The water hemlock shrub contains the neurotoxin cicutoxin and is responsible for most fatal wild plant poisonings in the United States.[19-21] Ingesting water hemlock can cause status epilepticus with resulting rhabdomyolysis.[20,21] The mechanism of rhabdomyolysis in hemlock poisoning is via cicutoxin's antagonism of gamma-aminobutyric acid that leads to unopposed spasmodic contractions, tonic-clonic seizures, myocyte disruption, and rhabdomyolysis with myoglobinuria.[21]

Compared with other types of seafood poisoning toxidromes, the Haff disease toxidrome most closely resembles marine palytoxin poisoning.[22] Palytoxin, a heat-stable aquatic neurotoxin, is produced by marine red algal dinoflagellates (*Ostreopsis* spp) and can be bioaccumulated in trophic levels in several finfish species and in anemones, corals (zoanthid corals), and crustaceans (xanthid crabs).[22] In addition to myalgia and rhabdomyolysis, other unique clinical manifestations of palytoxin poisoning that further differentiate it from Haff disease include pruritic skin rashes after dermal exposures to

Table 4. Laboratory Biomarkers of Rhabdomyolysis in Haff Disease Cases in the United States and China, 1984-2014

Laboratory Biomarker	United States Cases n=23[a]	Chinese Cases n=54[a]	Comparative Analysis
Initial mean ± SD CK, IU/L (normal value <200 IU/L)	9,661 ± 8,277	5,270 ± 5,616	t=2.709 P=0.008 d=0.6745 95% CI=0.174, 0.175
Overall mean ± SD CK, IU/L	8,538 ± 7,654	2,813 ± 3,350	t=4.588 P<0.0001 d=1.1424 95% CI=0.6221, 1.6627
Range of CK, IU/L (normal range 25-200 IU/L)	8,670-160,000	92-11,690	Not applicable
Peak mean ± SD CK, IU/L (normal value <200 IU/L)	29,630 ± 5,068	Not reported	Not applicable
Mean ± SD CK-MB fraction, % (normal value <5%)	2.667 ± 1.705	6.0 ± 5.4	t=2.861 P=0.005 d=0.7124 95% CI=0.2115, 1.2132
Range of CK-MB fraction, % (normal range 0.1%-5%)	0.5-4.8	0.2-20.6	Not applicable

CI, confidence interval; CK, creatine kinase; CK-MB, creatine kinase-muscle/brain; d, standardized mean difference.
$P<0.05$ is statistically significant.
Complete laboratory biomarker data were only available for 23 US cases of Haff disease following consumption of cooked fish and crayfish. Complete laboratory biomarker data were available for 54 Chinese cases of Haff disease following consumption of freshwater pomfret.

anemones and corals and autonomic and peripheral nervous system toxicity including circumoral paresthesias, oral temperature reversal mimicking ciguatera poisoning, dysgeusia, and dysarthria.[22] The mechanisms of rhabdomyolysis in palytoxin poisoning include sodium channel opening effects that prolong depolarization, causing muscle spasms with myofilament damage and release of muscle enzymes.[22]

Compared to water hemlock and palytoxin poisoning, Haff disease has few neurotoxic manifestations other than limb and body numbness and paresthesias. In addition, Haff disease most commonly occurs after eating freshwater seafood, while palytoxin poisoning occurs exclusively after eating cooked saltwater species. Table 5 compares the most common toxidromes characterized by rhabdomyolysis and myoglobinuria.

DISCUSSION

The results of this descriptive analytical comparison demonstrate that all confirmed Haff disease cases in the United States and China were characterized by muscle pain and laboratory evidence of rhabdomyolysis, but the seafood vectors, with the exception of boiled crayfish, differed. Although the comparative analysis of cases of rhabdomyolysis after crayfish consumption in the United States and China was limited by its small study sample size (n=15), it was epidemiologically significant for the following reasons: (1) the same species of boiled crayfish (*P. clarkii*) is a popular seasonal dish in Louisiana and in China, and (2) Chinese crayfish are popular imports in the United States.

Samples of the suspected seafood in 21 of the US cases reviewed in this article were tested by the CDC or the US Food and Drug Administration and were negative for the following aquatic toxins: ciguatoxin, saxitoxin, brevetoxin, tetrodotoxin, palytoxin, domoic acid, okadaic acid, and two blue-green algal or cyanobacterial toxins, microcystin, and nodularin.[9] Subsequent government laboratory testing of toxic crayfish samples in China were also negative for marine toxins.[17-18] Although governmental and regional health laboratories in the United States and China have not identified the heat-stable myotoxin that causes Haff disease, mice that were fed samples of toxic buffalo fish developed histopathologic evidence of rhabdomyolysis with renal tubular myoglobin and myoglobinuria.[3,9]

In 2001, 9 cases of Haff disease were reported by the Louisiana Office of Public Health in patients who resided within a 30-mile radius of each other in south-central Louisiana.[11] All patients were hospitalized for suspected myocardial infarction with the onset of chest pain, dyspnea, diaphoresis, and nausea 3-16 hours (mean, 8 hours) after consuming boiled crayfish purchased from the same seafood vendor.[11] Although the differential diagnosis of Haff disease includes myocardial infarction and other

Table 5. Comparison of Three Ingestion-Specific Toxidromes Characterized by Rhabdomyolysis and Myoglobinuria

	Haff Disease	Palytoxin Poisoning	Water Hemlock Poisoning
Toxin	Unknown	Palytoxin	Cicutoxin
Ingestion histories	Buffalo fish, crayfish, Atlantic salmon, freshwater pomfret	Marine reef fish, bottom feeding fish, marine crabs, and anemones	Water hemlock (*Cicuta maculata*)
Incubation	4-10 hours	8-16 hours	15 minutes-10 hours
Mechanisms	Isolated striated muscle myotoxicity, rhabdomyolysis	α-agonist, sodium channel opener, sarcolemmal membrane disruption, free radical–induced myofilament damage, rhabdomyolysis	GABA antagonism, unabated depolarization, tonic-clonic seizures, striated muscle damage, rhabdomyolysis
Presenting symptoms (mostly GI)	Vomiting, no fever	Metallic taste, vomiting, abdominal cramps, diarrhea, no fever	Profuse salivation, vomiting followed by convulsions, no fever
Neurotoxicity	Allodynia, oral paresthesias, extremity numbness, severe muscle pain	Circumoral and extremity paresthesias, dysgeusia, oral temperature reversal, weakness, dysarthria, muscle spasms, tonic-clonic seizures	Mydriasis, apnea, status epilepticus
Miscellaneous toxicity	Diaphoresis $\pm$ chest pain	Chest pain, dyspnea, asthmatic bronchitis, conjunctivitis, pruritic skin rashes, hypertension, electrocardiogram changes of sodium channel blockade	Diaphoresis, flushing, increased bronchial secretions
Myotoxicity	Muscle stiffness, pain, weakness, brown urine (myoglobinuria)	Muscle weakness, spasms, painful tonic-clonic contractions, brown urine (myoglobinuria)	Severe tonic-clonic seizures, brown urine (myoglobinuria)
Laboratory findings	↑↑↑ CK, CK-MB $<$5%, ↑ LDH, ↑ transaminases, normal troponin	↑ K, ↑ P, ↑ CRP, ↑↑ CK, CK-MB $<$5%, ↑ LDH, ↑ transaminases, normal troponin	↑↑↑ CK, CK-MB $<$5%, normal troponin
Morbidity	Resolves in 2-3 days	Resolves in months	Hypoxic brain damage, CRF possible in survivors
Case fatality rate	$<$0.5%-1.0%	1%-10%	25%-50%

CK, creatine kinase; CK-MB, creatine kinase-muscle/brain fraction; CRF, chronic renal failure; CRP, C-reactive protein; GABA, gamma-aminobutyric acid; GI, gastrointestinal; K, potassium; LDH, lactate dehydrogenase; P, phosphorus.
↑↑↑, massive levels of elevation; ↑↑, significant levels of elevation; ↑, moderate levels of elevation.

causes of toxic rhabdomyolysis, no specific laboratory tests exist for Haff disease other than the biomarkers of rhabdomyolysis.

In 2013, Xie et al reported a case of Haff disease in a 26-year-old male who had consumed boiled crayfish within 24 hours and presented with severe myalgias that lasted for nearly 3 months.[18] His serum CK and lactic dehydrogenase were significantly elevated, and a biopsy of the left biceps brachii muscle demonstrated rhabdomyolysis histopathologically. A radioisotope (Tc-99)-labeled bone scan demonstrated increased radioisotope tracer uptake in nearly all muscle groups, especially in the proximal extremity musculature. The authors concluded that a presumptive diagnosis of Haff disease based on presenting clinical and laboratory features could be further supported by muscle biopsy and bone scan by precisely locating sites of rhabdomyolysis.

No antitoxin exists for Haff disease poisoning, and treatment is entirely supportive. The initial clinical presentation may mimic myocardial infarction that must be excluded by normal electrocardiograms, serum troponin (troponin I and T) levels, and serum CK-MB levels $<$5%. Intravenous fluid hydration is required to maintain urine output and prevent the intratubular accumulation of myoglobin. In addition to intravenous fluid therapy, the management of rhabdomyolysis includes correction of metabolic acidosis, intravenous infusion of sodium bicarbonate to promote urinary alkalinization, and the use of intravenous mannitol as an antioxidant and osmotic diuretic to further minimize the nephrotoxic effects of intratubular myoglobin.[23] If these supportive therapies fail, extracorporeal renal replacement techniques, including hemofiltration and hemodialysis, may be instituted.[23] Severe myalgias may require management with intravenous opioids for pain control, and muscular spasm and rigidity may require management with benzodiazepines. Following successful management, most patients will have complete recovery without residual effects.

CONCLUSION

Haff disease in the United States most commonly may follow the ingestion of buffalo fish, but it may also follow the ingestion of crayfish and Atlantic salmon. Clusters of Haff disease have followed the consumption of boiled crayfish of the same species in the United States and China. The early identification of the seafood vectors of Haff disease will control outbreaks and prevent additional cases when seasonal conditions cause seafood to become myotoxic. The bioaccumulation of a new, heat-stable freshwater and/or brackish/saltwater algal toxin in seafood, similar to palytoxin but primarily myotoxic and not neurotoxic, is suspected of causing Haff disease. Haff disease most closely resembles marine palytoxin poisoning without significant neurotoxicity. More toxicologic investigations in larger case clusters are recommended to identify the toxin responsible for Haff disease.

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