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Guam Seaweed Poisoning: Pathological Findings

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Abstract—Laboratory studies were not helpful in determining the agent responsible for an outbreak of illness associated with eating seaweed. Serum myoglobin levels, however, apparently had prognostic value and may be useful should similar cases occur in the future.

The seaweed poisoning cases came to my attention when the clinicians involved requested that I see what could be done to solve this puzzling problem. Because there was concern that contamination from an oil-fueled power plant near the seaweed harvest site might have had some influence on this incident, we requested analysis of blood serum samples for hydrocarbons as well as ciguatoxin.

Blood tests for the presence of ciguatoxin were negative for all five hospitalized patients. Sera from four patients (A.E., A.U., R.M., S.T.) were screened for 31 hydrocarbon compounds; none was detected. Analyses for plasma cholinesterase were within normal limits for two of the patients tested (R.M. and S.T., sera collected 72 and 120 hours respectively after eating seaweed) and slightly elevated for a third (A.U., 7.7 and 7.1 units/mL, specimens collected approximately 48 and 120 hours respectively after eating the seaweed, reference range 2.4–6.2 units/mL). There is no record of other toxicology studies being done, presumably due to confidence in the hypothesis that a marine biotoxin was involved.

I have reviewed the autopsy findings of Dr. Park and I have looked at the slides that he has prepared. One purpose of an autopsy is to try to define the cause and mechanisms of death with the hope that this information will contribute toward treatment or prevention of future cases. All of the changes observed at these autopsies were non-specific and I was unable to further define their etiology. I specifically looked at the gastrointestinal tract, thinking that if these deaths were due to an ingested toxin—we were considering palytoxin at this time as well as ciguatoxin—the effect on that organ would be greater than it was on other internal organs such as the heart. In fact muscles in the intestinal wall showed very marked degeneration and more hemorrhage than did the other organs. The problem, however, is that the normal intestine contains many enzymes and it is difficult to say for sure whether the observed changes were related to the presence of a toxin or to post-mortem decomposition.

It may be of importance to note that observation of serum myoglobin levels may assist in making a prognosis in this type of case as the patients with the three highest myoglobin levels were also the patients that died. Two of the fatalities also had fatty changes of the liver as well as pre-mortem elevations of the liver enzymes LDH, SGOT, and SGPT. The fatality which did not have fatty change had normal liver enzyme values so, as Dr. Park has suggested, the observed fatty changes may have been aggravated by activity of the toxins or they may be totally unrelated to this incident.

In summary, laboratory studies performed on specimens from these patients were not helpful in establishing a specific diagnosis although high serum myoglobin levels were associated with unfavorable outcome.