

**KENTUCKY REGIONAL POISON CENTER****of Kosair Children's Hospital**

P.O. Box 35070

Louisville, KY 40232-5070

Louisville (502) 589-8222 • Kentucky 1-800-722-5725 • Secretary 629-7264 • Administration 629-5326  
FAX 629-7277

January 12, 2001

Mayo Clinic  
Department of Pathology  
Attn: Dr. Eric Pfeifer  
Hilton 11, 200 First St SW  
Rochester, MN 55905

Dear Dr Pfeifer:

I have reviewed the records of Linda Adanalian provided to me, including: the Community Medical Center record from 2/11/2000, the post mortem tissue samples, the autopsy report and cardiac pathology reports, letters of Drs. Nellis Smith and Steven Chooljian and the hospitalization record of Linda Adanalian from May of 1999. It is my opinion that Linda Adanalian died as a result of acute ingestion of a large amount of selenium. I base this opinion on the following facts and review.

According to the records provided to me, Ms Adanalian suffered chest pain, syncope and shortness of breath just prior to her transport to the emergency department. Prior to that earlier in that day she had experienced vomiting and diarrhea. On arrival at Community Medical Center she was awake, hypotensive (78 systolic) and tachycardic. Her O2 saturation was 88% despite 15 liters of oxygen via non-rebreather mask. She rather rapidly deteriorated and required a full ACLS resuscitation. The EKG initially showed a prolonged QT, tachycardia and an AV block. A subsequent EKG, within one hour of the first EKG, showed evidence of bradycardia, frequent premature ventricular complexes, and ST changes suggestive of anterolateral infarct. Additional findings were pulmonary edema with intubation and suction of pink frothy material. Blood pressure became unsupportable and the EKG showed ventricular fibrillation, and ultimately asystole.

The clinical presentation of Ms. Adanalian on February 11, 2000 is fairly classic for acute selenium ingestion and is consistent with previous cases of acute selenium poisoning I have been involved with. Selenium poisoning is generally rare but the well-documented effects are cardiovascular collapse, pulmonary edema and GI effects of vomiting and diarrhea, all of which Ms. Adanalian exhibited. The patient's rapid cardiovascular collapse is consistent with selenium ingestion. Vascular resistance is decreased in selenium poisoning along with reduced cardiac functioning. Both combine to produce a profound hypotension. Ms. Adanalian was found by the initial EMS staff to be hypotensive and the hypotension continued to



Designated as a Regional Poison Control Center by the  
American Association of Poison Control Centers

 **NORTON**<sup>®</sup>  
HEALTHCARE

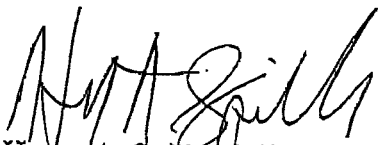
get progressively worse despite aggressive efforts by the ED physician. Specific EKG changes reported in Selenium poisoning have been ST changes suggestive of an acute MI, prolonged QT, premature ventricular contractions and Ventricular fibrillation, all of which were exhibited by Ms. Adanalian. Pulmonary edema is another prominent feature of acute selenium ingestion. The patient's initial complaints of shortness of breath along with reduced oxygen saturation measured in the ED are consistent with a progressing pulmonary edema. After intubation the patient's ET tube was suctioned of fluid, again consistent with pulmonary edema. The initial triage sheet at Community Medical Center has the chief complaint, taken when the patient was still conscious and able to give a history, of chest pain with frequent vomiting. This vomiting is again consistent with, perhaps even classic of, selenium poisoning. The failure to respond to heroic resuscitative measures has been reported frequently in selenium poisoning cases and is consistent with the fatal cases of selenium poisoning I have been involved in.

The autopsy report shows a number of features that are consistent with selenium poisoning. The first is bilateral pulmonary edema and congestion. The right hemothorax is also consistent with the typical presentation. The presence during autopsy of a green-brown cloudy fluid in the stomach has been reported in previous selenium ingestion deaths. No analysis of this fluid was done so nothing further can be said about this. The clinical picture of massive cardiovascular failure with EKG suggestion of a possible infarct is in contrast with the autopsy where no evidence of necrosis is found. The pathology report from Dr. Edwards supports the lack of anatomical evidence of a cardiac explanation for the sudden collapse. A second evaluation by the Stanford Pathology Consultants reports a similar finding of no morphological explanation for the patient's sudden and fatal collapse. This is consistent with other autopsy results in cases after selenium poisoning. Finally splenomegaly is consistent with previous fatal cases of acute selenium ingestion. This is probably secondary to the preferential uptake of selenium by the red blood cells.

Post Mortem blood samples were analyzed for a wide variety of drugs and the results were negative. Post mortem tissue samples (liver, kidney, spleen, and hair) were tested for cyanide, pesticides, drugs and metals. Significantly elevated selenium levels were found in the kidney and liver, the only tissues that were checked for selenium. Selenium is cleared via the kidneys and in acute ingestion I would expect elevated kidney levels. Tissues typically with the highest uptake are the red blood cells, kidney, liver, heart and lungs. Ms. Adanalian's levels in the kidneys were 11-12 times higher than would be expected in a normal kidney and are consistent with post mortem levels in known fatal cases of selenium poisoning. The liver levels are five to six times higher than would be expected and are again consistent with postmortem levels in known fatal cases of selenium poisoning. I believe these levels reflect an acute ingestion as opposed to a chronic elevation from a dietary source for several reasons. First, the hair sample records a low concentration of selenium. In a chronic exposure from a dietary source this level would be elevated in contrast to an acute exposure resulting in death where there would be no time to deposit selenium

in the hair. Second, the patient's physical appearance prior to this episode did not reflect that of a chronic dietary selenium exposure with the expected alopecia.

In conclusion, It is my opinion that Linda Adanalian died as a result of acute toxicity from selenium ingestion. This is based on the findings that 1) her clinical presentation is consistent with selenium poisoning, 2) her autopsy results are consistent with previous fatal cases of selenium poisoning and 3) her post mortem tissue samples are consistent with previous cases of fatal selenium poisoning.



Henry A. Spiller, MS, D.ABAT, D.ABFE  
Director, Kentucky Regional Poison Center