A LETHAL SYNDROME IN THE AXOLOTL: ANEMIA WITH ENLARGEMENT OF THE LIVER AND SPLEEN

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For some 18 months beginning in the fall of 1977 the axolotls at Indiana University were affected by a syndrome that caused the death of most afflicted animals. The only external signs were enlargement of the abdomen and failure to feed, except, in certain cases, after the most persistent efforts of the feeder over a period of weeks. The animals developed ascites and died one to ten weeks after the first signs were noticed. On postmortem, in addition to ascites, secondary infection was sometimes found, but typically the only changes were anemia (pink or clear blood in normal amounts) and enlargement of the liver and spleen. Either or both of these organs were twice or more the usual size, and were permeated with whitish nodular inclusions, between which the tissue was a mottled gray shade.

No effective treatment was ever found. Animals were injected with a variety of antibiotics up to 20 times, as well as with B-complex vitamins to stimulate their appetites. An animal might regain its health if it could be enticed into regular feeding, but no systematic pattern to this was seen. (Live food - tadpoles and fish - and smelt were tried, as well as the usual meats; and force-feedings were regurgitated.)

We made intensive efforts to recover bacteria or to detect parasites, without success. Infrequently, bacteria were grown from axolotls' viscera, but when these were injected into healthy controls no illness resulted. Transferring water from the bowls of affected animals to controls also produced no effect.

Tissue specimens and live, diseased animals were inspected by veterinarians and pathologists at three institutions. The findings of Herbert L. Thacker, D.V.M., of the Animal Disease Diagnosis Laboratory at Purdue University were typical:

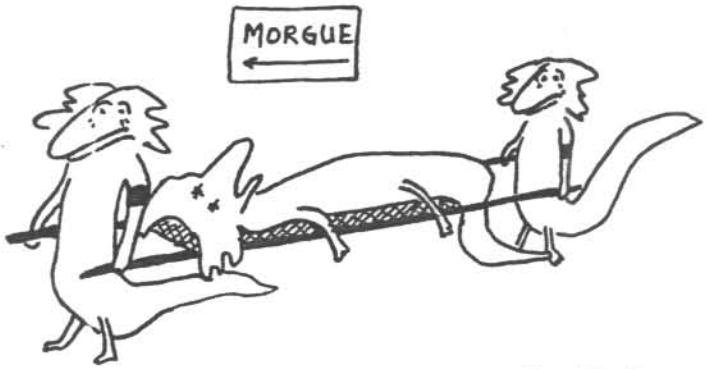
Histopathologic alterations in tissues examined were primarily limited to liver and spleen sections. Hepatic changes were of multifocal acute to subacute necrosis with early fibrosis, biliary hyperplasia and disseminated mixed cell population inflammation. Spleen changes were of disseminated lymphoid necrosis and congestion. The lesions observed were judged most likely to reflect non-specific toxic damage.
Diagnosis: Severe necrotizing hepatitis and splenic lymphoid necrosis of probable toxic etiology.

(No evidence of microbiological infection was ever noted by those studying the specimens.)

A search for toxicological agents was also negative, though it could not be conclusively negative since agents not specifically sought cannot be ruled out. Water samples were free of mercury and other heavy metals; also, changing water sources did not alleviate the problem. Changing the food source from liver to beef heart also had no effect.

The syndrome gradually became less frequent after several hundred animals of all genotypes and strains had died. It is now seldom seen at Indiana, though Dr. Larry Lemanski of the University of Wisconsin, Madison has noted the same problem (and has been advised similarly of evidence of chronic toxins by independent sources), as well as Dr. Jerry Justus of Arizona State University.

We would welcome any additional data or suggestions for investigation that could be supplied by interested parties.



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