Dutz- Kohout

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DEPARTMENT OF VETERANS AFFAIRS
Hunter Holmes McGuire Medical Center
1201 Broad Rock Boulevard
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In Reply Refer To: 652/113

Professor Dr. Matthew Meselson
Harvard University
Dept. of Biochemistry and Molecular Biology
7 Divinity Avenue
Cambridge, MA 02138

Dear Prof. Meselson,

Thank you for the opportunity to read your very interesting paper.

In reference to your questions concerning our case of gastric anthrax, I would like to add that:

1. There was no perforation of the stomach.

2. There was lymphadenitis due to anthrax infection. Large number of bacilli were found in the lymphatics and occasional bacilli in the perigastric lymph nodes and in the more distant lymph nodes (gastrohepatic, gastrocolic, perihepatic lymph nodes). The lymph nodes showed massive intrasinusoidal and intrapulp hemorrhages. Occasional bacilli were found in liver sinusoids which also contained numerous neutrophils. There was approximately 10 liters of straw colored fluid in the abdomen indicating no gross massive contamination from gastric fluid.

3. The picture of the stomach lesion was that of a primary anthrax, numerous serpiginous ulcers were found and the gastric wall measured up to 4 cm in thickness.

This pathology could be better explained by transmigration of the anthrax bacilli and the elaboration of their specific toxins.

Other organs showed no pathology (kidney, brain, spleen and lungs).

Death occured due to hypovolemia although the patient received large amounts of fluids and antibiotics empirically.

Case 6 of our 1984 paper had anthrax and typhoid fever. (Peyer's plaques were enlarged there).

In your cases as you stated further examination of the gastrointestinal

tract may have contributed more to the understanding of your cases.

The lungs seem to have all the pathology and in the picture in your publication the intestinal mucosa is intact and covers the areas of hemorrhage, a point distinctly against a primary gastrointestinal anthrax. Yet lymph node involvement present would indicate that the primary lesions may have been present but could not be located. The primary anthrax lesion produces a defect in the gastric mucosa not necessarily a perforation.

If there was hemorrhagic lymphadenitis near the intestinal hemorrhages this would also point towards the possible presence of gastrointestinal lesions in your case. Or was lymphadenitis only present in the mediastinum and near the lung?

I understand the climate (dry summers) could help the propagation of spores. This is the case especially when there are many grazing animals in the area and there is a relatively sparse vegetation that could be associated with a lot of spore formation.

Most reports in animals of anthrax in the most recent literature follow ingestion of contaminated meat or are probably due to a combination of spore inhalation and environmental contamination as well as an ingestion of spores. Certainly primary intestinal or gastric anthrax is a rare event. Lung and skin anthrax is much more frequently observed. As to the etiology as you stated a multifactorial etiology has to be taken into consideration.

In summary I enjoyed your paper and I enjoyed your discussion and I hope I will hear from you when you have some more interesting findings in the cases you were able to study. May I also add that I cultured the anthrax bacilli. These bacilli are cultured in the soil in the area we worked in.

Thank you again.

Sincerely,

E. DUTZ-KOHOUT, M.D.
Chief, Clinical Pathology