News
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******Body chemical found to induce cholera-like disorder.

DALLAS--Studies by a Dallas gastroenterologist and his colleagues show that large amounts of a body chemical, known for its role as a regulatory peptide, create a condition mimicking cholera when given to healthy subjects.

A person can die from Asiatic cholera in a matter of a few hours because the disease causes the intestines to rapidly secrete large amounts of fluid and salt.

According to Dr. Guenter Krejs, associate professor of Internal Medicine at The University of Texas Health Science Center at Dallas, this same type of secretory diarrhea is found with abnormally high blood levels of vasoactive intestinal polypeptide, or VIP.

VIP makes blood vessels all over the body dilate and in the intestines causes secretion of fluid and salt resulting in diarrhea. Death can occur from dehydration and subsequent kidney failure, says Krejs.

A recent study providing evidence of this role for VIP was recently completed by Krejs and researchers Drs. Mary Kane, also of Dallas, and Thomas O'Dorisio of the Ohio State University Hospital, Columbus. Findings were printed in the December 15 issue of The New England Journal of Medicine.

The researchers found that when VIP was given intravenously over 10 hours to five healthy test subjects, it produced profound secretory diarrhea. By stopping the intravenous infusion the symptoms also stopped since VIP has a very short half-life in the blood circulation, he says.

Krejs was the first to get FDA permission for infusion of VIP into normal subjects.

VIP concentration in blood is known to increase when a person has a pancreatic islet cell tumor, called a "VIPoma." Although normal pancreatic islet cells do not produce VIP, pancreatic islet cancer cells do. The tumors grow slowly but ultimately metastasize to the liver. They secrete exorbitant amounts of VIP. This can lead to death if the tumor is not surgically removed or if chemotherapy is not effective once metastases are present.

"Previously, it was thought by some investigators that VIP was only an indicator of the disease but not the real cause. But now more and more scientists are seeing the peptide as the true cause of the disease," Krejs says.

"Normally an adult will absorb through the intestines about three gallons of fluid every day in food, drink, bile and so on. But in Asiatic cholera and VIPoma, the small bowel can fail to absorb and can secrete many gallons of fluid instead."

A.N. is a 48-year-old mechanic from Mabank, Texas. He was in good health until the summer of 1980 when he began to develop chronic watery diarrhea. Despite treatment, the diarrhea increased slowly and the patient became progressively weaker due to a loss of potassium. In December 1981 he became unable to move his extremities and was so paralyzed that he was unable to dial a telephone number. At that time he was brought to a Dallas hospital where a diagnosis of VIPoma was established. The patient was subsequently admitted to the General Clinical Research Center, a National Institutes of Health-sponsored facility at UTHSCD. There additional studies were performed by Krejs and other physicians.

A.N. is one of five VIPoma patients seen by Dallas researchers during the past nine years. He was the only such patient from Texas, says Krejs.

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"This disease is very rare, but it can act as a model for the pathophysiology of secretory diarrhea and help us understand other disease mechanisms where intestinal secretion is involved," he says.

The disorder was first identified in 1958 by Verner and Morrison, who described a patient with islet cell tumor who lost five or six liters of stool and died from fluid and salt depletion. In later years, it became known as "pancreatic cholera syndrome," since the diarrhea results from intestinal secretion just as in Asiatic cholera. However, this term fails to denote that some tumors occur outside the pancreas, Krejs says.

Dr. Sami Said, formerly a faculty member at the health science center, and Dr. Stephen Bloom from London were the first to find a high level of plasma VIP in such patients.

Krejs has collaborated with Said for several years to determine the functions of VIP in the intestinal tract. The present study closed the chain of evidence that established VIP as the true cause of this disease.

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