

ary to an empty sella), which may cause erosion of the sellar floor, producing fistula into the sphenoid sinus.¹²

Cerebrospinal fluid rhinorrhea associated with an empty sella is an indication for prompt surgery. There are two common surgical approaches to the sella turcica.¹¹ A transfrontal approach is used in patients in whom the preoperative diagnostic tests do not show the existence of a transsellar fistula, for occasionally an empty sella can be associated with extrasellar fistulas.⁹ When preoperative diagnostic studies suggest that the fistula is through the sella, then the transsphenoidal approach is more direct

and safer than a craniotomy transfrontal approach.^{13,14}

References

1. Busch W: Die Morphologie der Sella Turcica und ihre Beziehungen zur Hypophyse. *Virchows Arch Pathol Anat* 320:437-458, 1951.
2. Lee W, Adams JE: The empty sella syndrome. *J Neurosurg* 28:351-356, 1968.
3. Ommaya AK, DiChiro G, Baldwin M, et al: Nontraumatic cerebrospinal fluid rhinorrhea. *J Neurol Neurosurg Psychiatry* 31:214-225, 1968.
4. Brisman R, Hughes J, Mount LA: Cerebrospinal fluid rhinorrhea and the empty sella. *J Neurosurg* 31:538-543, 1969.
5. Norsa L: Cerebrospinal rhinorrhea with pituitary tumors. *Neurology* 3:864-868, 1953.
6. Smith C, Walter L: Cerebrospinal fluid rhinorrhea with cyst of the pituitary body. *Arch Otolaryngol* 4:610-614, 1931.
7. Som ML, Kramer R: Cerebrospinal rhinorrhea pathological findings. *Laryngoscope*

50:1167-1177, 1940.

8. Kaufman B: The 'empty' sella turcica a manifestation of the intrasellar subarachnoid space. *Radiology* 90:931-941, 1968.

9. Jordon R, Kendal J, Kerber C: Primary empty sella syndrome. *Am J Med* 62:569-578, 1977.

10. Weisberg LA, Housepian EM, Saur DP: Empty sella syndrome as complication of benign intracranial hypertension. *J Neurosurg* 43:117-180, 1975.

11. Hooper AC: Sphenoidal defects: A possible cause of cerebrospinal fluid rhinorrhea. *J Neurol Neurosurg Psychiatry* 34:125-128, 1976.

12. Mortara R, Norrell H: Consequences of a deficient sellar diaphragm. *J Neurosurg* 32:565-573, 1970.

13. Garcia-Uria J, Carrillo R, Serrano P, et al: Empty sella and rhinorrhea: A report of eight treated cases. *J Neurosurg* 50:466-471, 1979.

14. Weiss MH, Kaufman B, Richards DE: Cerebrospinal fluid rhinorrhea from an empty sella: Transsphenoidal obliteration of the fistula: Technical note. *J Neurosurg* 39:674-676, 1973.

Deaths Related to Coffee Enemas

John W. Eisele, MD, Donald T. Reay, MD

NATUROPATHIC therapies are increasing in popularity in the United States, and many of them rely primarily on alterations in diet. Although diet and nutrition are recognized as important adjuncts in cancer therapy,¹ conventional medicine rejects the concept of cancer therapy relying solely on dietary changes, and unlicensed practitioners of such therapy have been discouraged or prevented from practicing in the United States. Although these therapies may be of questionable therapeutic value, they are usually considered harmless.

One regimen that relies primarily on a diet of natural foods combined with the ingestion of mixed potassium salts, Lugol's solution, thyroid extract, niacin, and pancreatin also includes coffee enemas administered as often as every two hours. This

therapy has been recommended not only to patients with cancer but also to those with chronic, degenerative diseases. In the last two years we have noted two deaths in which the common factor was the use of this therapy; autopsy examination and laboratory studies indicate that death was related to the coffee enemas.

Report of Cases

CASE 1.—A 46-year-old woman complained of intermittent, right upper-quadrant pain for 20 years. Her physician diagnosed this as colitis or cholecystitis; additional diagnoses were achlorhydria and degenerative arthritis. Five months before her death, at her request, diet therapy was started consisting of fresh fruit and vegetable juices with supplements of bile salts, calcium, phosphorus, lecithin, minerals, and two liver medications.

Seventeen days before death and five days before admission she experienced severe, right upper-quadrant pain. Her physician described her as alert and oriented with stable vital signs, but in severe pain. The following day her condition worsened, and she began frequent vomiting. That evening a diagnosis of cholelithiasis was made. The patient

refused hospitalization or surgery. During that night she received "ten or 12" coffee enemas, as frequently as "three or four" an hour. She also received "cheilonium and chenathus." Her pain decreased, and she reportedly passed several gallstones in her stool. The coffee enemas were, however, continued at a rate of one per hour until the evening before admission when she had a grand mal seizure. During the night she had two more seizures, and on the following morning a fourth seizure was followed by cardiorespiratory arrest. An aid car was summoned; resuscitation was successful but she remained apneic and comatose until her death 12 days later.

At the time of her admission, laboratory studies disclosed the following values: serum sodium, 113 mEq/L; potassium, 1.9 mEq/L; chloride, 69 mEq/L; bicarbonate, 25 mEq/L; pH, 7.52; P_aO₂, 81 mm Hg; and P_aCO₂, 19 mm Hg. A toxicological screen for barbiturates, phenothiazines, and glutethimide was negative. Xanthene levels were determined but were not considered toxic.

An autopsy was performed 23 hours after death. It was remarkable for bilateral confluent pneumonia and hypoxic encephalopathy. Grossly and microscopically, neither the gallbladder nor colon showed evidence of active inflammation; the gallbladder and bile ducts contained no stones and showed no stenosis or obstruction. Death was attributed to bronchopneumonia and cerebral hypoxia with hypokalemia as the proximate cause.

From the Office of the Medical Examiner, King County (Washington), and the Department of Pathology, University of Washington, Seattle.

Reprint requests to Office of the Medical Examiner, 325 Ninth Ave, Seattle, WA 98104 (Dr Eisele).

San Diego

CASE 2.—Thirteen months before her death, a 37-year-old woman had a right, modified radical mastectomy for a poorly differentiated, infiltrating ductal and lobular carcinoma with metastases to one of 20 axillary lymph nodes. She refused chemotherapy after surgery. Approximately six months later she noted nodules in the right side of the neck, right subclavian region, and mastectomy scar. Immunotherapy was started with dinitrochlorobenzene and BCG vaccine, and later with γ -globulin and tetanus toxoids. She responded poorly and two months later received chemotherapy with cyclophosphamide, fluorouracil, and methotrexate. Two months later her physicians recommended radiation therapy, which she refused. She also discontinued the immunotherapy and chemotherapy.

Eight weeks before death she attended a clinic where she remained for two weeks, and a special diet similar to that in the first case was started, with coffee enemas four times a day using 0.95 L of coffee for each enema.

She returned to Seattle and continued the treatments with weekly telephone consultations with the new clinic but without seeing a local physician. Two weeks before death the patient was weak, and the night before death she vomited a "bile-like substance." Similar material was expelled after the coffee enemas. Shortly before death she experienced chest and abdominal pain, dizziness, and dyspnea.

An autopsy was performed 24 hours after death. Anatomic examination was remarkable for metastatic or recurrent tumor in the mastectomy site, liver, and left breast, as well as the left axillary, right cervical, right supraclavicular, paratracheal, mediastinal, and hilar lymph nodes. There was no evidence of pericardial metastases. Although relatively widespread, the metastases were not thought to be extensive enough to explain her death. The only other pertinent findings were pleural effusions (450 mL in the right and 250 mL in the left), a pericardial effusion (505 mL), and ascites (100 mL). A toxicological screen on postmortem blood showed only a small amount of caffeine. Vitreous humor samples were drawn from the right eye 23 hours and 50 minutes after death and from the left eye 27 hours and ten minutes after death. Analysis for electrolytes in the left vitreous showed the following values: sodium, 113 mEq/L; potassium, 13.0 mEq/L; and chloride, 88 mEq/L. In the right eye the values were as follows: sodium, 114 mEq/L; potassium, 11.0 mEq/L; and chloride, 91 mEq/L. Extrapolation of the potassium values to the time of death yielded a value of -0.9 mEq/L. No anatomic or toxicological lesion adequate to cause death was found, and death was attributed to fluid and electrolyte imbalance.

Comment

In the first case the laboratory results are standard antemortem serum values and can be interpreted as such. In the second case death occurred before blood could be drawn, and we relied on vitreous humor for evaluation of electrolyte levels. Although this procedure is widely accepted by forensic pathologists, it may be unfamiliar to many clinicians. Coe² has performed extensive studies on this fluid and summarized its value in postmortem diagnosis. He indicates that sodium and chloride concentrations in vitreous humor are stable until putrefaction begins, although their normal ranges are somewhat wider than those in serum: for sodium, levels of 130 to 155 mEq/L and for chloride, levels of 105 to 135 mEq/L are considered normal. Potassium is more difficult to evaluate, since its concentration increases after death. Numerous studies cited by Coe, however, have shown that this increase is linear. Adjutantis and Coutselinis³ showed that by taking samples from each eye and extrapolating to a normal level, the time of death can be determined to within 1.1 hours during the first 12 hours. In our case we knew the time of death and extrapolated to determine an antemortem potassium concentration. The negative value obtained is obviously impossible; errors were magnified by the long delay before the initial sample and the short time interval between samples. Nevertheless, the results indicate that the antemortem potassium level was probably low.

A second question concerns the validity of the fluid and electrolyte abnormalities as mechanisms of death. In both cases complete autopsy and toxicological examination failed to reveal another cause of death. In the first case the survival interval permitted correction of the initial imbalance; it also permitted further diagnostic tests that indicated no other disease processes or injuries. In the second case the presence of pleural, pericardial, and peritoneal effusions supported the impression of fluid excess, and the pericardial effusion may have been enough to cause fatal cardiac tamponade.

The laboratory values obtained in both deaths would place them in Coe's² "low-salt pattern." In vitreous hu-

mor this pattern is characterized by low sodium level (less than 130 mEq/L) and chloride level (less than 105 mEq/L), and relatively low potassium level (less than 15 mEq/L). It is a pattern seen frequently in a medicolegal population, most commonly in chronic alcoholics. Recently Coe² reported a series of cases in which this pattern was associated with diuretic therapy and salt restriction.

Examination of coffee used in the enema therapy offers an explanation of the mechanism of these deaths. This material had a potassium concentration of 17.0 mEq/L, and an osmolality of 62 mOsm/kg. Sodium and chloride could not be detected. Using 0.95 L of this liquid three or four times an hour certainly could produce sodium and chloride depletion and fluid overload on the basis of osmotic and concentration gradients across the colonic mucosa. The decrease in serum potassium may represent the dominance of the osmotic effects over the concentration effects. Toxicological results in both cases indicate that not enough caffeine was absorbed to cause a substantial toxic effect.

We are unable to evaluate the prevalence of coffee enemas and are unaware of any other deaths attributed to this treatment. When the second case was publicized by the news media, we received telephone calls and letters from numerous individuals and groups who were using or prescribing coffee enemas. With the current wave of popularity of naturopathic medicine, one would expect an increase in this therapy and consequent morbidity and mortality.

George Rowley was responsible for the field investigation and medical histories of both patients.

References

1. Proceedings of the American Cancer Society and National Cancer Institute Conference on Nutrition in Cancer, June 29-July 1, 1978, Seattle, Washington. *Cancer* 43(suppl):1955-2162, 1979.
2. Coe JI: Postmortem chemistry of blood, cerebrospinal fluid, and vitreous humor, in Tedeschi CG, Eckert WG, Tedeschi LG (eds): *Forensic Medicine: A Study of Trauma and Environmental Hazards*. Philadelphia, WB Saunders Co, 1977, pp 1033-1060.
3. Adjutantis G, Coutselinis G: Estimation of the time of death by potassium levels in the vitreous humor. *Forensic Sci* 1:55-60, 1972.
4. Coe JI: *The Case of the Saltless Spinster*, forensic pathology check sample No. FP-104. Chicago, American Society of Clinical Pathologists, 1979.