JOURNAL OF FORENSIC SCIENCES

J Forensic Sci, November 2012, Vol. 57, No. 6
doi: 10.1111/j.1556-4029.2012.02214.x
Available online at: onlinelibrary.wiley.com

CASE REPORT

PATHOLOGY/BIOLOGY

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Diagnosis of Hemorrhagic Stroke in an Exhumed Brain After Three Years of Burial in a Deep Grave

ABSTRACT: We present the forensic neuropathologic analysis of an exhumed decomposed brain following long-term interment in a 50-year-old white woman, who had been buried for 34 months. Next of kin authorized exhumation of the body for an autopsy to determine the cause of death. The embalmed body was anatomically intact and revealed decompositional changes with mold colonies. Internal viscera showed intact histomorphology. The brain revealed diffuse congestive swelling and extracellular edema with dissecting parenchymal hemorrhage and hematoma originating from the left putamen and thalamus and extending to the left lateral ventricle. Excitotoxic neuronal injury as well as penumbric parenchymal changes was noted. Cause of death was determined to be a hypertensive cerebral parenchymal hemorrhage. This case and our previously reported case are sentinel cases, which should encourage and guide the forensic neuropathologic work-up and investigation of causes of death in spite of long-term burial in deep graves.

KEYWORDS: forensic science, forensic neuropathology, deep grave, interment, exhumation, autopsy, brain, stroke

We have previously diagnosed Alzheimer's disease (AD) in the exhumed decomposed brain of an 81-year-old white man, who had been buried in a deep grave for 20 months (1). We now present the second case of forensic neuropathologic diagnosis following long-term interment in a deep grave. In this second case, we diagnosed hemorrhagic stroke following exhumation after a longer period of interment in a deep grave for approximately 3 years, compared to 20 months of interment in the first case. These two cases are sentinel cases, which should encourage and guide the forensic neuropathologic work-up and investigation of causes of death in spite of long-term burial in deep graves.

The decedent was a 50-year-old white woman who had died suddenly and unexpectedly without an attending physician. She was found dead in bed at her home and had complained of a severe headache on the same day she was found dead. The local coroner did not authorize an autopsy to be performed by a forensic pathologist, presumed that she died from natural causes, and certified the cause of death to be "presumed natural disease." The decedent was embalmed and buried in a standard six-feet-deep grave in a local cemetery approximately 1 week after her death.

The body was exhumed after 34 months of interment. Exhumation was authorized by the County Court of Common Pleas following a petition by the next of kin. The objective of the exhumation was for a complete autopsy to be performed with toxicologic analysis, in order to determine a definitive cause of death and exculpate a suspicion that she may have died from poisoning or drug toxicity.

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Received 28 June 2011; and in revised form 6 Oct. 2011; accepted 22 Oct. 2011.

Materials and Methods

Exhumation and Autopsy

Disinterment, exhumation, autopsy, and reinterment were performed on the same day within 12 h. The body was received in a sealed brown wooden funeral casket, accompanied by approximately 100 cc of brown fluid found at the bottom of the vault. The exterior of the casket was dry, structurally intact, and revealed diffuse thin layers of white mold colonies. The interior of the casket was moist, structurally intact, and lined by white velvety and cushioned upholstery and pillow, which revealed multifocal white mold colonies.

The body was completely covered with a white velvety coverlet, which showed multifocal gray–white mold colonies. The body was moist and laid supine in funeral repose. The body was removed from the casket, washed, and cleaned. A complete autopsy was performed by the combined technique of Virchow and Rokitansky (2). Representative sections of all organs were taken and submitted for routine tissue histochemical analysis by hematoxylin and eosin histochemical staining.

Representative samples of scalp hair were frozen and saved for toxicologic analyses. A red-top specimen bottle of fluid collected from the interment vault was submitted for toxicologic analyses. All fingernails were removed and saved for possible toxicologic analyses if the need arose.

Results

Forensic Pathologic and Neuropathologic Findings

The body was adequately nourished, weighed approximately 130 pounds, measured 65 inches, and appeared to be older than the stated age of 50 years old. The body was clad in funeral attire comprising multiple articles of clothing, which were torn and

decaying, and diffusely soiled by gray–white and brown-tan waxy adipocere-like material and gray–white mold colonies. Two articles of jewelry were found around the neck, which were soiled by waxy adipocere-like material.

The body was embalmed, anatomically intact, and diffusely covered by gray–white mold colonies on the face, neck, shoulders, and upper chest. All the anatomic regions of the head, neck, trunk, and extremities were wholly present. The body revealed early diffuse mummification and leatherization of the skin of the shoulders, pelvis, and upper and lower extremities, accompanied by diffuse variegated brown-gray-green-black cutaneous discoloration. There was a diffuse early cutaneous adipocere formation over all the surfaces of the body. There was patchy skin slippage of the face, scalp, trunk, and extremities. The eyeballs were collapsed and contained no vitreous fluid. The corneae and sclerae were opacified and cloudy.

There was no evidence of trauma, fractures, muscular atrophy, or edema. There was approximately 50 cc of serosanguineous fluid in each pleural cavity and approximately 75 cc of serosanguineous fluid in the peritoneal cavity. The organs of the pleural and peritoneal cavities were in the normal anatomic positions in relation to one another in situ, embalmed, intact, and revealed multiple perforating embalming trocar defects. The parenchymal histomorphology was intact with minimal early autolysis.

The heart weighed 310 g and revealed no ventricular wall hypertrophy or atrioventricular dilatation. The coronary arteries revealed severe segmental and eccentric atherosclerosis of the left anterior descending coronary artery and oblique branches with 75–90% multifocal intraluminal narrowing. The left coronary artery main stem, the right coronary artery, and the left circumflex coronary artery showed mild segmental and eccentric atherosclerosis with less than 30% multifocal intraluminal narrowing. The myocardium in the left ventricle revealed multifocal myofibrillar contraction band degeneration, myofibrillar cytoplasmic hypereosinophilia, and focal myocardial interstitial fibrosis. There was moderate atherosclerosis of the aorta.

The lungs revealed severe acute pulmonary edema and congestion with patchy bilateral irregular pulmonary emphysematous changes, many intra-alveolar pigment-laden histiocytes, and patchy pulmonary interstitial anthracosis. There was sparse nonspecific infiltration of the hepatic portal triads by lymphocytes without fibrosis. All other organs did not reveal any significant gross or histopathologic changes except the brain.

The scalp did not reveal any hemorrhages. The skull did not reveal any fractures. There were no epidural or subdural hemorrhages, membranes, or xanthochromia. The brain weighed 960 g and was embalmed. The cerebral hemispheres revealed a normal pattern of gyral and sulcal convolutions. There was severe diffuse global expansion of gyri and compression of sulci, accompanied by bilateral symmetrical grooving of the unci and cerebellar tonsils without necrosis. The arachnoid and pia mater revealed patchy acute subarachnoidal hemorrhages over the left cerebral hemisphere accentuated basally (Fig. 1). The vessels of the circle of Willis revealed no anomalies, significant atherosclerosis, or aneurysms.

The neocortical gray ribbon revealed no contusional necrosis or laminar necrosis. The gray–white matter demarcation was blurred. There was a left hemispheric hemorrhagic cavity containing large amounts of extravasated liquid and clotted blood (Fig. 2). This cavity obliterated and replaced the left posterior thalamus and putamen, extended across the wall of the left lateral ventricle into the cavity of the left ventricle, which contained blood, and formed one contiguous cavity with the hemorrhagic cavity. There was

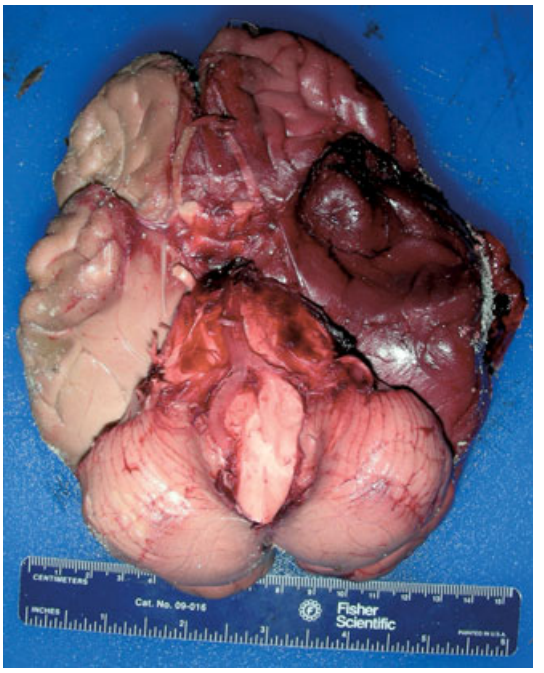

FIG. 1—*Gross autopsy photograph of the brain, showing the basal surfaces of the cerebral hemispheres, cerebellar hemispheres, and brainstem with acute subarachnoidal hemorrhages over the left cerebral hemisphere and brainstem with bilateral grooving of the cerebellar tonsils and compression of the brainstem and basal subarachnoidal cisterns.*

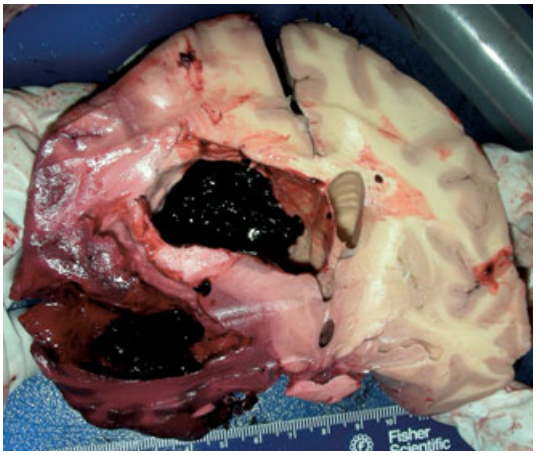

FIG. 2—*Gross autopsy photograph of the coronal section of the cerebral hemispheres at the level of the globus pallidus showing a left ganglionic parenchymal hemorrhage extending into the left lateral ventricle. There are large amounts of left intraventricular hemorrhage accompanied by diffuse cerebral parenchymal edema.*

penumbric gray-dusky parenchymal discoloration and softening of the left temporal lobe, left temporal pole, and left mesial temporal lobe. Representative samples of the cerebral hemorrhage were taken in gray-top sodium fluoride specimen bottles and saved for toxicologic analyses.

The right lateral ventricle and the third and fourth ventricles were compressed and contained small amounts of blood. There was no subcortical ganglionic or white matter atrophy including the hippocampus. The midbrain, pons, and medulla oblongata revealed no hemorrhage or necrosis. The cerebellar hemispheres were symmetrical and revealed no atrophy, necrosis, or hemorrhage. There

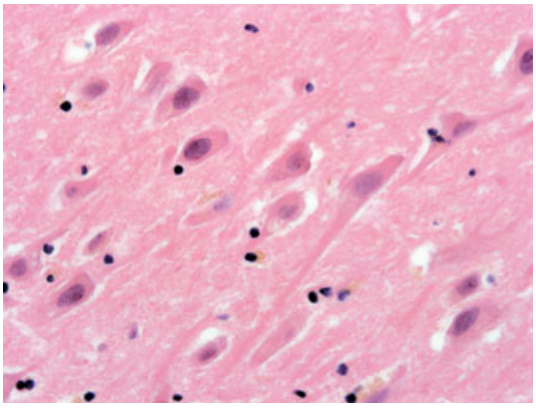

FIG. 3—*Photomicrograph of hematoxylin and eosin stained section of the stratum pyramidalis of the cornu ammonis showing eosinophilic and pyknotic neurons (×600 magnification).*

was diffuse autolysis of the internal granule cell layer. The pituitary gland was autolytic and showed no tumors, parenchymal necrosis, or hemorrhages. The spinal medulla showed no parenchymal or meningeal hemorrhage, exudate, or necrosis.

Brain histomorphology comprised diffuse mild parenchymal autolysis with multifocal eosinophilic neuronal necrosis (Fig. 3) showing selective topographic vulnerability of neurons. There was mixed cytotoxic and vasogenic diffuse cerebral extracellular gray and white matter edema. There was hyaline arteriolosclerosis in many penetrating cerebral parenchymal blood vessels with sparse perivascular hemosiderin-laden histiocytes in few penetrating vessels. Sections of the left thalamus and putamen revealed acute dissecting parenchymal hemorrhages extending across the periventricular wall and ependymal lining into the left ventricular cavity.

Postexhumation Toxicologic Findings

Toxicologic analysis of the representative samples of the liquid and clotted cerebral hemorrhage revealed 1.05 mg/L of caffeine and 1.7 mcg/dL of lead. Toxicologic analysis of the vault fluid sample revealed 0.35 mg/L of caffeine. There was no acetaminophen, salicylic acid, ethyl alcohol, or other acidic, neutral, or basic drugs identified in the blood or vault fluid samples. Toxicologic analysis of the scalp hair samples revealed 0.46 mcg/g of lead. No arsenic, mercury, or other heavy metals were identified in the blood or hair sample. Ethylene glycol was not detected in the blood sample or vault fluid sample. There were no birefringent calcium oxalate crystals in histologic sections of the kidneys microscopically examined in a polarized light field.

The underlying cause of death of this 50-year-old white woman was determined to be hypertensive cerebral parenchymal hemorrhage (stroke), which originated from the left thalamus and putamen and extended into the left lateral ventricle. The manner of death was natural. There was no autopsy evidence of poisoning or drug toxicity.

Discussion

In this reported case, the neuropathologic findings correlated and confirmed the prevailing terminal forensic scenario of sudden death following complaints of severe headaches. Brain examination revealed a hemorrhagic stroke, which accounts for 10% of all strokes, and is associated with a 50% case fatality rate (3). Diminishing sensorium, headaches, vomiting, and hemiparesis are frequent presenting complaints of hemorrhagic stroke. Death frequently occurs within 1–3 h with large hemorrhages like in this case; however, smaller hemorrhages may be compatible with survival. As we have seen in this case, the putamen is the most common site for hypertensive hemorrhagic strokes. Hypertension causes the majority of these hemorrhages in middle-aged patients; however, hemorrhagic strokes sometimes occur in patients who are stressed by a variety of stress inducers (3). Toxicologic analyses, in this case, reasonably exculpated any proposition of drug poisoning or toxicity.

This case and our previously reported case (1) indicate a serial trend that forensic neuropathologic diagnosis can be made with a reasonable degree of medical certainty in exhumed embalmed human bodies that have been buried in deep graves for prolonged periods of time. Autopsies of putrefied bodies were thought to be useless until the end of the 19th century (4). In spite of increasing acceptance of exhumed body autopsies, there remains marked paucity of published data on the forensic neuropathologic examination of autopsied decomposed brains. Examination of decomposed brains may not have gained wider acceptance possibly because the brain is widely expected to decompose quickly following death because human tissue water content is greatest in the brain (about 90%, while total body water is about 60%) (5,6). However, this case and our previously reported case show that prolonged interment should not preclude forensic neuropathologic analysis of brains from exhumed bodies, especially if the body was embalmed prior to interment. Advanced postmortem changes should be no reason to refrain from tissue analysis, histochemistry, immunohistochemistry, and DNA analysis; however, there appears to be no linear relationship between postmortem interval and discernible autopsy findings (4).

Adequate embalmment of the body and brain prior to interment appears to be a common denominator in our cases and the case reported by Tintner et al. (7), whereby they diagnosed the characteristic spongiform changes of Creutzfeldt-Jakob disease in the basal ganglia and cerebellum of the embalmed brain of a 32-year-old man who had been buried for 7 months. Their brain was reported to be embalmed and exhibited tissue consistency and general appearance that were similar to the usual formalin-fixed brain without marked autolysis, similar to the exhumed brain we are reporting in this case report.

We strongly encourage forensic pathologists and neuropathologists to proceed with exhumations, autopsies, and tissue analysis for the determination of causes of death in cases involving long interment. Causes of death may be determined with reasonable degrees of medical certainty in about 78% of exhumed cases, and adequate preservation of anatomy for tissue diagnosis may be expected up to 10 years of interment, and even longer if the body was embalmed (8,9).

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