TECHNICAL NOTE

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Pseudo-Subarachnoid Hemorrhage of the Head Diagnosed by Computerized Axial Tomography: A Postmortem Study of Ten Medical Examiner Cases

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ABSTRACT: In this report, we describe ten cases of pseudo-subarachnoid hemorrhage on computer axial tomography (CT) scan of the head. A pseudo-subarachnoid hemorrhage is a false positive finding by CT of the head in which the scan is interpreted as being positive for a subarachnoid hemorrhage not substantiated by subsequent neuropathologic findings. This study is a retrospective review of postmortem cases brought into the Office of the Chief Medical Examiner for the State of Maryland over a three-year period (from 1997 to 2000). We compared the clinician's impression of the CT scan with the postmortem neuropathology. The clinical diagnosis of subarachnoid hemorrhage was based on misinterpretation of non-contrast CT scans of the head. In six of the ten cases, the reading was performed by a radiologist and in four cases by nonradiologist physicians (emergency room physician, neurologist, or neurosurgeon).

All the patients survived between a few hours to a few days after being admitted to the hospital. For most of the cases (80%), the neuropathology showed hypoxic/ischemic encephalopathy. The most common cause of death (four out of ten cases) was narcotic intoxication. This report is submitted so that clinicians and pathologist become more familiar with this entity.

KEYWORDS: forensic science, subarachnoid hemorrhage, hypoxic/ischemic encephalopathy, computerized axial tomography

Introduction

Over the three-year time period of this study (1997 to 2000) the Office of the Chief Medical Examiner (OCME) for the State of Maryland performed approximately 9000 postmortem examinations of which there were ten cases in which premortem-computed tomography studies of the head were incorrectly interpreted by clinicians or radiologists as subarachnoid hemorrhages with cerebral edema. The patients expired shortly afterwards (from 8 h to 4 days postadmission), but the followup autopsy examination demonstrated the absence of subarachnoid hemorrhage. These cases fell into the category of what has been described as "pseudo-subarachnoid hemorrhage" (1). The OCME is a statewide medical examiner system; all cases of sudden, unexpected, violent, or suspicious deaths from around the state are referred to it. Eight different hospitals were involved (of these eight, three are designated regional trauma centers for the Maryland Emergency Medical Services System). Each year approximately 5000 brain injuries occur in Maryland (incident rate at 97 per 100 000); in the year 1998 there were 793 fatalities (death rate at 99 per 100 000) (personal communication from the Maryland Traumatic Brain Injury Demonstration Project).

Annually, approximately 500 autopsies are performed on narcotic/drug abuse deaths by the OCME (2).

Ten separate clinicians and at least nine different radiologists made the interpretations (in one case the radiology report did not specify which radiologist made the reading). The cases are summarized below and tabulated (Table 1).

Materials

Medical charts/hospital records, computed axial tomography radiology reports, postmortem autopsy, and neuropathology reports were reviewed and compared.

Methods

A total of ten cases with complete postmortem examinations and neuropathology examinations performed by forensic pathologists and a neuropathologist were retrospectively collected; medical charts and radiologist interpretations were reviewed by a panel of forensic pathologists.

Cases:

Case 1

A 49-year-old White female with a history of schizoaffective disorder was admitted for acute psychosis. She was later discovered unresponsive in a locked seclusion room, time of 10 to 15 min from last being seen conscious. Approximately 4 h after she was found unresponsive, a head CT, without contrast, was performed.

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TABLE 1—*Summary of radiology report.*

Case	Age/Race Gender	Cause of Death	Post Collapse Time Until CT Scan	Post Collapse Time of Survival in Hospital	Narcotic Drug Screen Positive	Hospital Regional Trauma Center
1	49-W-F	Dilated cardiomyopathy	4 h	25 h		+
2	48-B-F	ASCVD*	3 h	9 h		+
3	4-B-F	Acute bacterial leptomeningitis	at 1–2 h	21 h		+
4	42-W-F	Acute narcotic intoxication	2–2 1/2 h	8 h	+	
5	40-B-F	HCVD† associated with alcohol and drug use with complications	1 h	36 h	+	+
6	9 day-W-F	Hypoxic ischemic encephalopathy due to hypoglycemia	5 h	96 h		
7	44-W-F	Drug use with complications	1 3/4 h	12 h	+	+
8	45-B-F	Hypoxic encephalopathy due to narcotic intoxication	Unknown (>3 h)	72 h	+	
9	45-B-F	Pulmonary arterial thrombo-embolism	2 1/2 h	24 h		
10	45-B-M	Dilated cardiomyopathy and narcotic use	3 h	144 h	+	

* Atherosclerotic cardiovascular disease.
† Hypertensive cardiovascular disease.

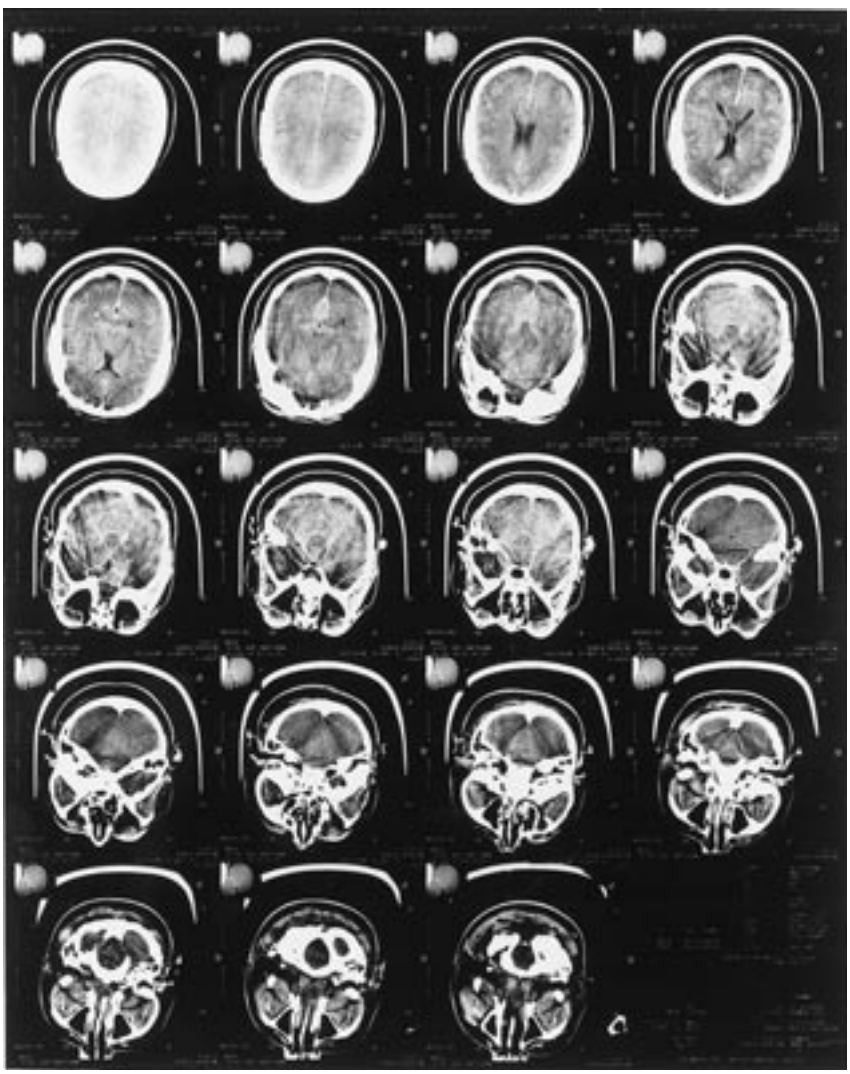

FIG. 1—(Case 3) *Radiologic interpretation of the CT scan of this brain: There is subarachnoid hemorrhage with blood seen within the suprasellar cisterns, paramesencephalic cistern, and interhemispheric fissure. A small amount of blood is also seen to outline the sulci.*

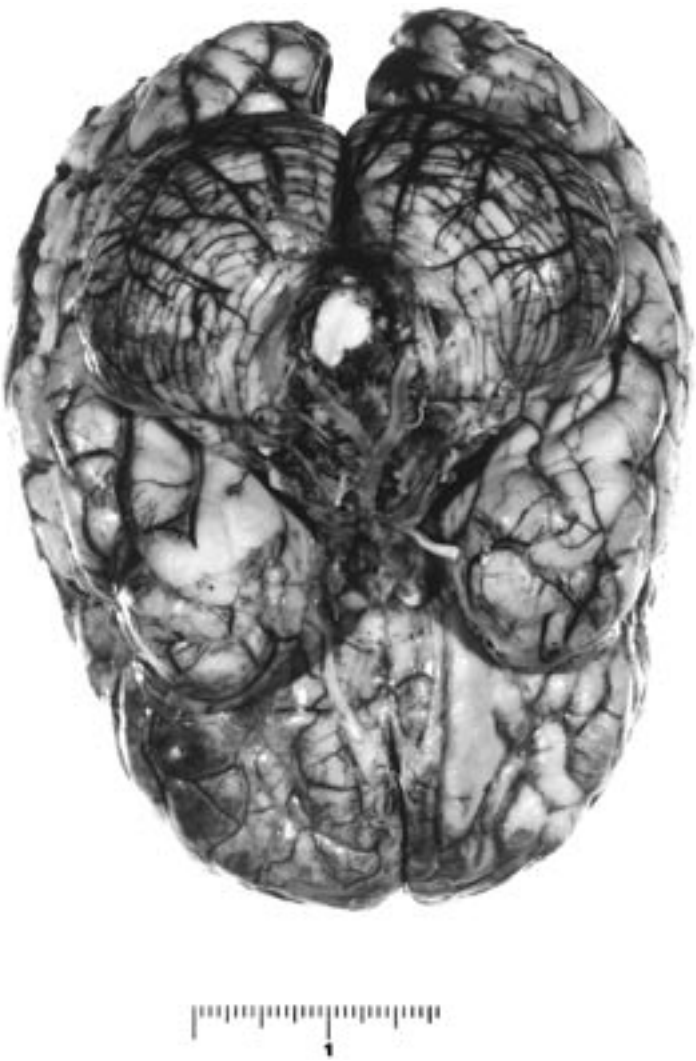

FIG. 1 *(continued)*

It was interpreted by a radiologist, neurosurgeon, and a critical care attending physician as showing diffuse subarachnoid hemorrhage with diffuse anoxic/ischemic changes. She was pronounced dead 25 h after she collapsed.

Autopsy showed an obese, 5 ft 6 in., 244 lb female with mild brain edema (brain weight 1330 g), possible anoxic/ischemic encephalopathy but no subarachnoid hemorrhage. She also had cardiomegaly and a dilated cardiomyopathy. The cause of death was certified as dilated cardiomyopathy, with schizo-affective disorder as a contributory condition.

Case 2

A 48-year-old Black female with a history of a recent fall, hypertension, anemia, chronic renal insufficiency, coagulopathy, and ethanol abuse complained of abdominal pain one morning before she collapsed unresponsive. She was brought to an emergency room in paroxysmal electrical activity but expired 9 h later.

A nonenhanced CT of the head, performed 3 h after her collapse, was interpreted by an emergency room physician and a neurosurgeon as showing subarachnoid hemorrhage and anoxic brain damage. Subsequently, a radiologist read the film as showing: "diffuse hypoxic/ischemic insult, with uncal tentorial herniation and right frontal intraventricular hemorrhage. All basilar cisterns are obliterated and 4th ventricle not visualized; may be some subarachnoid hemorrhage in the basilar system, but this is not definite."

An autopsy, performed the next day on the 5 ft 8 in., 150 lb female, showed a 1220 g brain, with hypoxic/ischemic encephalopathy, acute hemorrhagic infarcts in the right frontal cortex, right temporal region (perivascular hemorrhages), and caudate nuclei and a small amount of hemorrhage in the 4th ventricle. The leptomeninges were translucent, without subarachnoid hemorrhage over the convexities or at the base. The heart showed triple vessel coronary atherosclerosis and the cause of death was attributed to her heart disease.

Case 3

A 4-year-old Black female complained of a headache and had congestion for two days prior to developing mental status changes (confusion). She was found unresponsive late one morning by her parent who called paramedics. On arrival in the emergency room her temperature was 103°F. and she had fixed and dilated pupils. One hour later she was brought for a CT scan of the head. The nonenhanced CT was read by a radiologist, a neurologist, and neurosurgeon as showing cerebral edema, subarachnoid hemorrhage with blood within the suprasellar and paramesencephalic cisterns and interhemispheric fissure; a small amount of blood was also seen to outline the sulci. Subsequent blood cultures were positive for *Streptococcus pneumonia.* An infectious disease specialist was consulted for the possibility of tissue/organ procurement. This clinician, aware of the head CT scan, considered the underlying etiology to be due to a viral encephalitis or less likely due to pneumococcal meningitis and recommended that organ procurement be deferred. The child expired 21 h postadmission.

An autopsy was performed the next day on the 3 ft 8 in. and 47 lb child. Neuropathology revealed a 1450 g brain, with acute bacterial leptomeningitis (thick diffuse greenish exudate), hypoxic/ischemic encephalopathy, and thromboses of the superior sagittal sinus and cortical veins. No subarachnoid hemorrhage was noted (Figs. 1*a* and 1*b*).

Case 4

This 42-year-old White female, with a past medical history of depression, ethanol/drug abuse, and remote neck and back surgery, was found unresponsive 30 min after last being seen by her family. She was brought to the emergency department and resuscitated. A urine drug screen was positive for opiates and cocaine. Unenhanced CT scan of the head performed 2 h after admission was interpreted by a radiologist as demonstrating extensive subarachnoid hemorrhage at the base of the brain, with effacement of subarachnoid spaces at the base of the brain and over the cerebral convexities, as well as effacement of the 3rd and 4th ventricles and severe brain edema. The patient expired about 8 h postadmission.

Autopsy performed on this 5 ft 6 in., 138 lb female showed the following neuropathology: translucent leptomeninges, except for the left inferior parietal cortex, where there was slight hemorrhagic discoloration. There was severe cerebral edema, bilateral uncal, and tonsilar herniation, a slightly adherent thrombus in the superior sagittal sinus, and acute hypoxic/ischemic encephalopa-

thy. The cause of death was attributed to an acute narcotic intoxication.

Case 5

A 40-year-old Black female, with a history of schizophrenia, ethanol and substance abuse, and diabetes mellitus, was found unresponsive by her daughter who last saw her conscious about 5 to 15 min earlier. When paramedics arrived she was in cardio-respiratory arrest, and she was brought to a local hospital. A nonenhanced head CT scan was obtained within ½ h of admission and interpreted by a neurosurgery consultant as showing diffuse swelling, loss of gray-white function, effaced basilar cisterns, bilateral basal ganglia infarcts, an open 4th and lateral ventricle, and questionable subarachnoid hemorrhage versus tentorium with compressed vessels. A neurology consultant interpreted the scan as showing diffuse edema. The radiologist read the scan as showing no evidence of hemorrhage. Toxicologic screen was positive for opiates and a blood alcohol of 0.13 g/dL. She was pronounced dead 36 h after admission.

An autopsy on this 5 ft 8 in., 230 lb Black female revealed a poorly preserved brain weighing 1360 g with autolysis without subarachnoid hemorrhage. Microscopically, acute hypoxic/ischemic changes of the pons and cerebellum were seen. The heart showed concentric left ventricular hypertrophy. The cause of death was certified as hypertensive cardiovascular disease associated with alcohol and drug use with complications.

Case 6

A 9-day-old White female developed seizures after showing increasing lethargy and decreased appetite one day prior to admission. On admission, she was noted to be jaundiced and have jerking movements. Hypoglycemia (blood glucose = 18 md/dL) was found. She was subsequently resuscitated but remained comatose and expired on the fourth hospital day.

Five hours after admission a CT scan of the head, without contrast, was performed. The radiologist read this scan as showing: subarachnoid hemorrhage, bilaterally, particularly in the frontoparietal regions, and anterior and posterior interhemispheric fissures. There were symmetrical decreased attenuations of the bilateral middle cerebral and posterior cerebral artery territories consistent with infarcts. Sulcal effacement was seen; the 3rd and lateral ventricles were small.

Two days after admission a followup CT scan of the head, with and without contrast, was read by another radiologist as showing: decreased density to both cerebral hemispheres compatible with ischemic changes with sparing of cerebellum and basilar ganglia regions; there was loss of the quadrigeminal plate cisterns with a patent suprasellar cistern; the lateral and 3rd ventricles were compressed. A contrast enhanced CT study of the head showed normal opacification of the Circle of Willis and intracerebral vasculature; the superior sagittal sinus and internal cerebral vein were patent. No mention of subarachnoid hemorrhage was made in these latter reports.

Autopsy revealed a 19 ¾ in. (90th percentile) and 2500 g (50th percentile) infant. The brain weighed 680 g, with translucent leptomeninges and no subarachnoid hemorrhage. There was an extensive infarction of the occipital region and overall changes consistent with an acute metabolic injury to the brain, such as hypoxic/ischemia or hypoglycemia. The cause of death was determined to be hypoxic-ischemic encephalopathy due to hypoglycemia.

Case 7

A 44-year-old White female became unresponsive after a witness observed her snort heroin. Forty-five minutes later she was brought into an emergency room, intubated, and resuscitated. A CT of the head, without contrast, was performed 1 h postadmission to the emergency department. A neurosurgical consultant and the resident admitting the patient interpreted the film as showing edema and possibly subarachnoid hemorrhage in the supracellar cistern. A neurologist interpreted the scan as showing possibly a subarachnoid hemorrhage, generalized edema with loss of the gray-white junction. The following day the radiologist's interpretation was diffuse brain edema compatible with anoxic brain injury. Hyperdensity along the vessels of the Circle of Willis was thought to be probably due to slow flow in an anoxic brain rather than subarachnoid hemorrhage. The patient expired approximately 12 h postadmission.

Autopsy showed a 5 ft 5 in., 198 lb White female; the brain weighed 1490 g and was grossly normal, without subarachnoid hemorrhage. The cause of death was certified as due to drug intoxication with complications.

Case 8

A 45-year-old Black female was brought into an emergency room by a friend in cardio-respiratory arrest, where she was resuscitated. The friend did not indicate how long the patient had been unresponsive. A toxicology screen was positive for opiates.

A noncontrast CT scan of the head performed 3 h postadmission was read by a radiologist as demonstrating diminutive caliber to the entire subarachnoid space and the ventricular system. The 3rd and 4th ventricles were not visualized; there was increased density material within the subarachnoid cisterns and along the tentorium. The differential diagnosis of the abnormalities included diffuse subarachnoid hemorrhage, or in the absence of subarachnoid hemorrhage, diffuse cerebral infarction. The patient remained comatose and was pronounced dead 2 ½ days postadmission.

Autopsy showed a 5 ft 1 in., 107 lb Black female with a brain weight of 1500 g. There was hemorrhagic necrosis of the cerebral cortex, deep gray matter, brainstem, and cerebellum; focal fresh subarachnoid hemorrhage was noted in the left fronto-parietal region and in the right superior parietal region. The changes were thought to be consistent with global hypoxic-ischemic injury. The cause of death was hypoxic encephalopathy due to narcotic intoxication.

Case 9

A 45-year-old Black female collapsed, unwitnessed, on the street, while working as a census taker. Four years earlier she had an episode of respiratory arrest and was diagnosed with asthma. She was brought to an emergency room, intubated, and resuscitated after being down approximately 25 min. A CT scan of the head, without contrast, was performed 2 h postadmission, and it was read by a radiologist as showing diffuse cerebral edema, extensive subarachnoid hemorrhage debris in the basilar cisterns and in the inner hemispheric fissure, as well as distributed over the convexities (Fig. 2). The toxicology screen was negative. The patient ultimately had a negative brain flow study and neurology examination consistent with brain death. The cause of her collapse was assumed to be a subarachnoid hemorrhage probably due a ruptured aneurysm, although further premortem diagnostic studies were not done.

The family, approached by an organ procurement agency, granted consent for donation. The medical examiner's office was

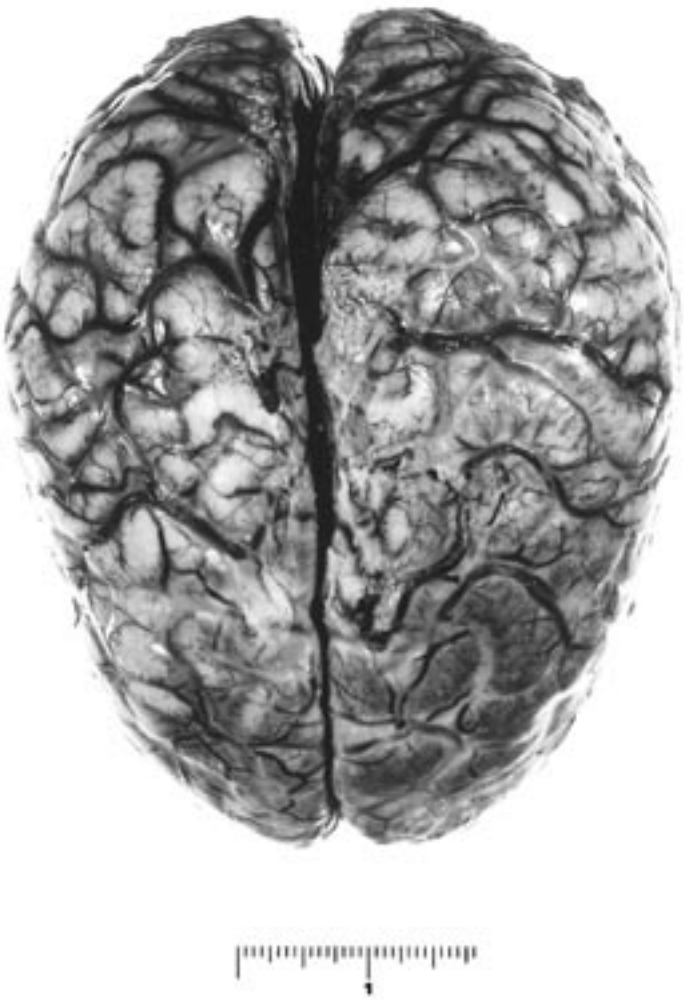

FIG. 2—(Case 9) *CT Scan of Head: Radiologic interpretation of this CT scan of the brain and posterior fossa: Examination discloses diffuse cerebral edema, and there is extensive subarachnoid hemorrhagic debris in the basilar cisterns and in the inner hemispheric fissure as well as distributed over the convexities.*

contacted and also granted permission for procurement to proceed. The heart and kidneys were taken for transplant. Consent for the lungs was also given but they were not taken because a bronchoscopy showed some purulent secretions in the left mainstem bronchus.

Autopsy on this 5 ft 5 in., 216 lb female revealed several pulmonary thrombo-emboli in both lungs and thrombi in her deep calf veins, bilaterally. The brain weighed 1430 g and showed edema, herniation of the tonsils, and hypoxic-ischemic encephalopathy. There was no subarachnoid hemorrhage present. The cause of her death was attributed to pulmonary arterial thrombo-emboli due to deep vein thrombosis.

Case 10

This 45-year-old Black male, with a history of hypertension, went into cardio-respiratory arrest shortly after he was incarcerated by a sheriff's department for driving while intoxicated. Subsequent toxicology testing was positive for opiates. He was brought to an emergency department and resuscitated, but he was comatose with fixed, dilated pupils. A CT scan of the head, without contrast, was performed 3 h postadmission; a radiologist's interpretation was: extensive subarachnoid hemorrhage; the sulci were effaced, gray-white junction indistinct, consistent with generalized edema; effacement of ambien cisterns and 4th ventricle, increased density of posterior fossa most likely representing subdural hemorrhage along the tentorium. The patient's condition did not improve over the next few days. On his 3rd day of hospitalization a repeat head CT scan, without contrast, was performed. The radiologist's interpretation of this scan was: increasing subarachnoid hemorrhage involving the bilateral cerebral hemispheres; blood was seen on the cisterns, which were effaced. The temporal horns were effaced; the frontal horns were not as well demonstrated, probably due to increasing edema; blood was seen tracking along the tentorium without significant internal change. Neurological and neurosurgical consultations were obtained, but no invasive procedures were performed because of his poor prognosis and condition. He expired on Day 6 of his hospital stay.

An autopsy performed on the 5 ft 9 in., 205 lb male showed a 1440 g brain with marked discoloration of the convexities, with an exudate of the leptomeninges, bilaterally (leptomeningitis). There was extensive necrosis and autolysis of the base of the brain, with possible herniation of the temporal lobe unci; coronal sectioning of the brain revealed extensive hemorrhagic necrosis of the structures at the base of the cerebral hemispheres, diffuse infarct of the cortex, basal ganglia, hypothalamus, cerebellum, and edema of the brainstem and cerebellum. The cause of death was certified as leptomeningitis with cerebral cortical infarction; contributory conditions consisted of dilated cardiomyopathy and narcotic use.

Discussion

Review of records of individuals brought to the State of Maryland's Office of the Chief Medical Examiner between 1997 and 2000 revealed ten cases in which an apparent discrepancy existed between the clinical interpretation of premortem, nonenhanced head CT examinations and the postmortem neuropathology results. The age of the ten individuals at death ranged from nine days up to 49 years. Their treating institutions included eight different medical centers in Maryland. The ten premortem CT scans were interpreted by at least nine different radiologists. In six out of ten cases the radiologists read the films as showing subarachnoid hemorrhage. In three out of ten cases, the radiologists read the films as showing either subarachnoid hemorrhage or changes of edema/anoxic brain injury. This indicates that the nonenhanced CT scans cause a common problem related to the limitations of the scans resolution and the inability to distinguish true subarachnoid from pseudo-subarachnoid hemorrhages. Only one case had a contrast enhanced CT scan performed and that was done as a followup three days later. In this latter case, subarachnoid hemorrhage was not described and postmortem neuropathologic examination did not find a subarachnoid hemorrhage. Based upon only one case, it is not clear whether or not contrast enhancement would help to distinguish true from false-subarachnoid hemorrhage. It should be mentioned that none of our ten cases underwent magnetic resonance imaging (MRI) studies. Two of our cases involved a 9-day-old and a 4-year-old, indicating that this diagnostic problem is not limited to adult patients. In two cases of meningitis a misinterpretation of subarachnoid hemorrhage was made. This phenomenon has been reported previously (3). The high density noted on the head CT in the subarachnoid space was attributed to the elevated cerebrospinal fluid proteinaceous exudate. In our two cases of meningitis, premortem cerebral spinal taps were not performed.

The largest series of cases to examine the problem of brain

edema and false positive subarachnoid hemorrhage was reported by Avrahami et al (4). This group reviewed 100 comatose patients who had CT diagnoses of brain edema and subarachnoid hemorrhage. In their retrospective study, the authors concluded that these patients probably did not have subarachnoid hemorrhages. Their conclusions were based upon the following reasons: (1) the varying etiologies for the patients' comas made subarachnoid hemorrhage unlikely and (2) some subarachnoid hemorrhages could be excluded by spinal tap results. The authors hypothesized that it was a disturbance in the venous drainage through the lateral sinus, which produced engorged veins subsequently misinterpreted as subarachnoid hemorrhage by CT scan. The limitation of this study was the lack of postmortem neuropathology followup.

There have been two reports with postmortem comparison studies. One was by Spiegel et al. (5), who reviewed ten patients who underwent a nonenhanced head CT that showed brain swelling as a mass effect, with high density of the falx and tentorium, suggesting a subarachnoid hemorrhage. In this study the authors hypothesized that impaired vascular circulation of the dura may have been responsible for the increased density of the tentorium and falx simulating subarachnoid hemorrhage. In the other report (2), a 35-year-old drug abuser collapsed after an overdose. His head CT scan (nonenhanced) was misinterpreted as subarachnoid hemorrhage, but the subsequent autopsy showed only anoxic encephalopathy and no hemorrhage, similar to five of the cases in this report. Three explanations were proffered: (1) In anoxic brain injury the cerebral cortex, with its congested veins, is displaced into the area usually occupied by cerebrospinal fluid. The average tissue density adjacent to the dura is hyperdense, which appears as a subarachnoid hemorrhage. (2) With ischemia, brain density decreases relative to congested veins in the superficial cortex giving a relative hyperdense appearance of the superficial tissues. (3) As already described above, there is impairment of vascular (venous) circulation through the dura.

In one of our cases there was organizing thrombus in the superior sagittal sinus. Another case had some nonadherent clot and in one case the sinus was filled with slightly adherent thrombus. Therefore, impairment of vascular circulation via the dura sinuses does not appear to explain the pseudo-subarachnoid hemorrhage in every case.

For one of our ten cases, the medical examiner was contacted by a tissue/organ procurement agency and asked to permit donation to proceed based upon a clinical impression of brain death due to a subarachnoid hemorrhage (Case 9). Several organs and tissue were subsequently taken. However, the lungs were not recoverable because of possible bronchopneumonia/bronchitis noted on bronchoscopy. Ultimately, the postmortem examination showed that the clinical impression was incorrect and that there was evidence of pulmonary arterial thrombo-embolism in these lungs. The medical examiner faces a predicament, namely, whether or not he/she should grant consent for organ harvesting under circumstances where no definitive diagnosis has been made other than by a nonenhanced head CT in a patient with anoxic encephalopathy. We urge great caution be taken in these cases and for the pathologist to consider the potential problems that could ensue should the case be one of pseudo-subarachnoid hemorrhage. This may include an inability to render an accurate cause and manner of death following the organ donation and subsequent postmortem examination.

In some cases the initial impression of the treating clinicians and their consultants was based upon their misinterpretation of the head CT scans prior to the radiologist's reading. This usually occurred during the night when CT scans were obtained on an emergent basis and read by attending clinicians, fellows, and residents prior to the official reading by a radiologist the next day. Clinicians should be aware of this diagnostic problem as they develop their plans of care.

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