Pseudo-subarachnoid Hemorrhage: a Rare Neuroimaging Pitfall

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ABSTRACT: ***Objective:*** We report an unusual case of the CT appearance of diffuse subarachnoid hemorrhage in a patient with anoxic encephalopathy, a situation which neurosurgeons, neurologists, and neuroradiologists should be aware of. ***Clinical presentation:*** A young man collapsed unconscious in jail after abusing an unknown quantity and variety of drugs. CT scan showed a picture compatible with diffuse subarachnoid hemorrhage. ***Intervention:*** As the patient had a Glasgow Coma Score of 3 no heroic intervention was undertaken. An autopsy performed 40 hours after the initial ictus and 24 hours after death revealed no evidence of subarachnoid hemorrhage but gross and microscopic evidence of anoxic encephalopathy. ***Conclusion:*** Anoxic encephalopathy can mimic diffuse subarachnoid hemorrhage on CT.

RÉSUMÉ: Pseudo-hemorragie sousarachnoïdienne: une embûche rare à la neuro-imagerie. ***But:*** Nous rapportons un cas inusité d'apparition à la tomodensitométrie d'une hémorragie sousarachnoïdienne chez un patient présentant une encéphalopathie anoxique, une situation que les neurochirurgiens, les neurologues et les neuroradiologues devraient connaître. ***Présentation clinique:*** Un jeune prisonnier est devenu inconscient après avoir utilisé une quantité et une variété inconnues de drogues. La tomodensitométrie a montré une image compatible avec une hémorragie sousarachnoïdienne diffuse. ***Intervention:*** Comme le patient avait un score de 3 à l'échelle de coma de Glasgow, aucune intervention héroïque n'a été tentée. Une autopsie faite 40 heures après l'ictus initial et 24 heures après le décès n'a révélé aucune évidence d'hémorragie sousarachnoïdienne, mais des manifestations macroscopiques et microscopiques d'encéphalopathie anoxique. ***Conclusion:*** Une encéphalopathie anoxique peut simuler une hémorragie sousarachnoïdienne à la tomodensitométrie.

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The only condition we are aware of which can mimic the appearance of diffuse subarachnoid hemorrhage (SAH) on unenhanced computed tomography (CT) is pyogenic meningitis.[1] However, in most of these cases the clinical progression would distinguish it from subarachnoid hemorrhage. We report a case of sudden collapse with CT appearance compatible with diffuse SAH in a patient who in fact suffered from anoxic encephalopathy due to respiratory arrest secondary to drug overdose.

Case Report

A 35-year-old man with a known history of substance abuse was arrested and taken to a detention centre following a domestic dispute. He was drowsy and had consumed an unknown quantity of alcohol and drugs. While in jail he suddenly became comatose and was taken to a local hospital where he was intubated and resuscitated. Glasgow Coma Score (GCS) was 3. Plain CT of the head was interpreted by a neurologist and a general radiologist as demonstrating subarachnoid hemorrhage in the basal cisterns, convexity, and interhemispheric fissure (Figures 1, 2, 3). The patient was transferred to our neurosurgical centre where the CT was again interpreted as showing SAH by neurosurgery residents and staff, and neuroradiologists. As his GCS was 3, further therapy was discontinued and the patient expired several hours later.

A coroner's autopsy was performed by a forensic neuropathologist about 40 hours after the patient's collapse. The general autopsy was normal except for congestion in the lungs. The brain was swollen with generalized flattening of the gyri, effacement of the sulci, and bilateral herniation of the hippocampal unci. There was absolutely no evidence of subarachnoid blood. There were no gross lesions on sectioning of the brain and microscopic examination revealed evidence of acute hypoxic ischemic changes consisting of selective neuronal necrosis, patchy infarction, and cortical edema. Pre-mortem toxicology studies revealed toxic levels of alcohol, codeine, morphine, alprazolam, and acetaminophen.

The cause of death was attributed to anoxic encephalopthy due to respiratory arrest secondary to central nervous system depression induced by a drug overdose.

Discussion

The clinical presentation in this case was compatible with spontaneous SAH and the CT appearance was interpreted as showing diffuse SAH by every doctor who encountered the case pre-mortem and by numerous others who were presented the case at post-mortem conference. In this case the "mis-diagnosis" had no negative clinical implications for the management of the patient whose treatment was appropriate given his neurological condition. The incidence of the erroneous diagnosis of an apparent SAH is indeterminate as many cases like the one described

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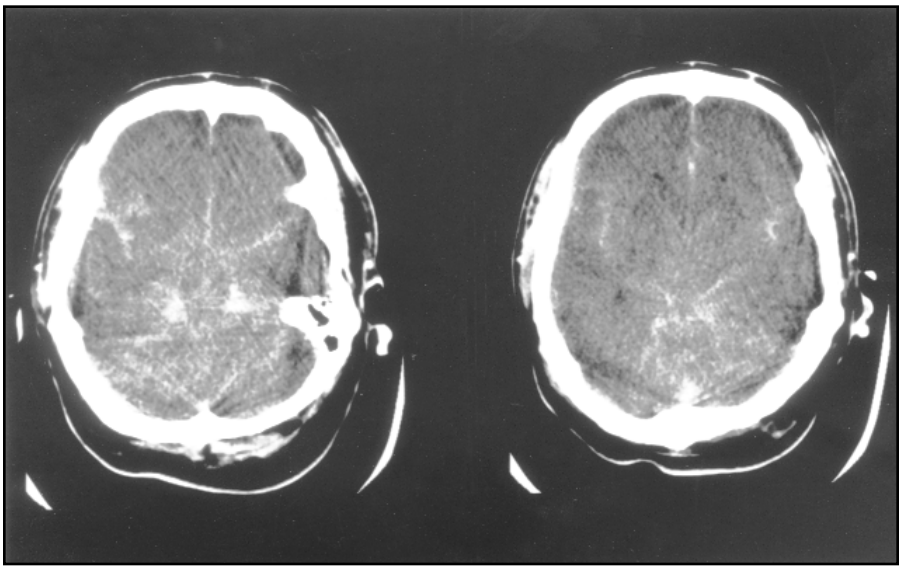

Figure 1: *Axial unenhanced CT slices at the level of the basal cisterns showing the appearance of hyperdensity involving the subarachnoid spaces, and loss of grey-white differentiation.*

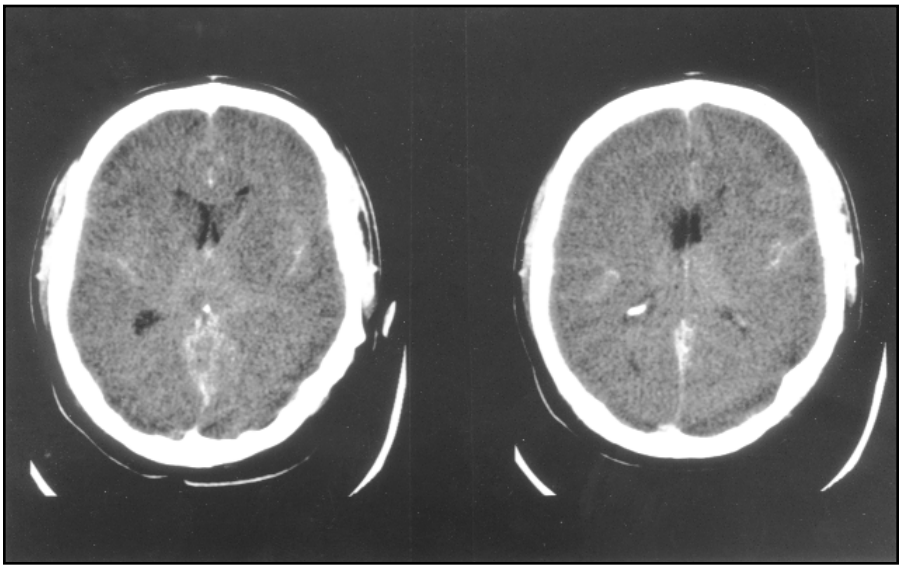

Figure 2: *Axial unenhanced CT slices demonstrating hyperdensity in the sylvian fissures bilaterally and loss of grey-white differentiation.*

herein do not undergo autopsy. This situation has been alluded to,[2] and increased density of the dura on CT mimicking subarachnoid hemorrhage has been reported in an autopsy study in patients who had increased intracranial pressure of non-SAH etiology.[3]

CT scans of normal patients show the cranial bone lined by a thin layer of dura mater which overlies the subarachnoid space filled with cerebrospinal fluid (CSF). The average width of this space varies, increasing with age. In the case of brain swelling due to acute hypoxic anoxic encephalopathy the cerebral cortex becomes displaced into areas normally occupied by CSF and veins become congested. The increased average tissue density immediately deep to the dura and superficially within the cerebral sulci results in hyperdensity on unenhanced CT, simulating the appearance of SAH. Another plausible explanation[2] is that with severe ischemia the density of the brain on CT decreases relative to the density in the congested veins in the superficial cortex again giving a relatively hyperdense appearance to superficial tissues. Yet another explanation incriminates impairment of the vascular circulation of the dura[3] although the latter could not account for the appearance of the sylvian fissure and cortical sulci seen in our case.

In spite of the frequency of anoxic encephalopathy in patients studied with CT, the incidence of the CT appearance of subarachnoid hemorrhage in these patients is either extremely rare or under-recognized and/or under-reported. We feel that this

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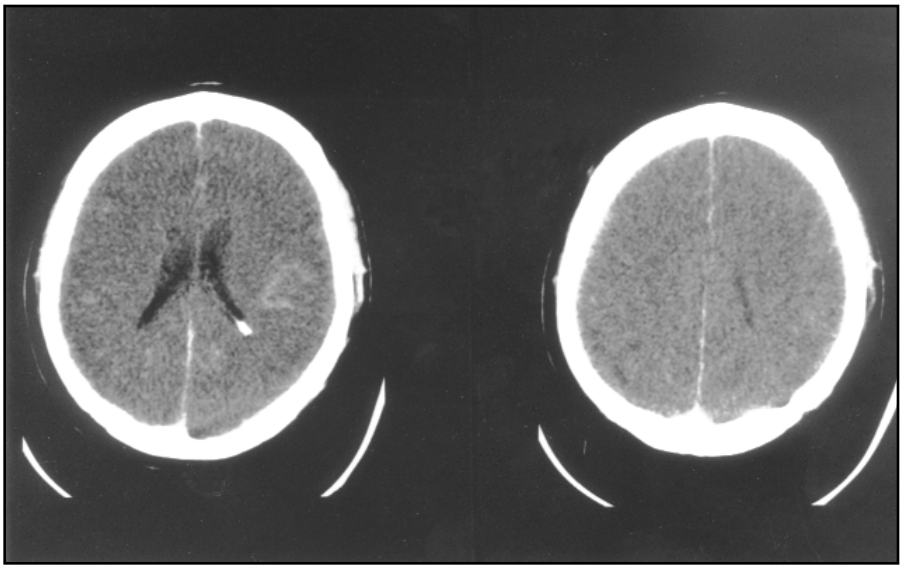

Figure 3: *Axial unenhanced CT slices showing hyperdensity in the left convexity subarachnoid space and loss of gray-white differentiation.*

rare imaging pitfall should be known to all neurosurgeons, neurologists, and neuroradiologists, as well as residents and emergency room physicians caring for patients with unexplained unconsciousness. It could have clinical implications if a patient with anoxic or other metabolic encephalopathy with GCS greater than 3 were inappropriately treated as a case of SAH. Furthermore, there could possibly be medico-legal implications of the misdiagnosis of SAH.

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