Medical Examiner · County N York



CASE SUMMARY REPORT

Case Number: 20-00902 Pathologist: Nadia A. Granger, MD Pronounced: Mar 30 2020 8:22PM ME Case County/Origin: Monroe Co.

Name:

Daniel T. Prude

Manner of Death: Homicide

Date of Birth: 09/20/1978

Cause of Death: Complications of asphyxia in the setting of physical restraint Acute phencyclidine intoxication

Age: 41 Years

FINAL FINDINGS

- Complications of asphyxia in the setting of physical restraint:
 - a. Bilateral organizing bronchopulmonary pneumonia.

 - c. Severe respiratory acidosis (clinical history).
 - d. Profound global hypoxic ischemic injury (clinical history).
- ii. Subfalcine and transtentorial herniation. II. Excited delirium:
 - e. History of physical restraint in prone position (incident report).
- b. Agitation and combative behavior (clinical history).
- Suicidal ideation and possible auditory hallucinations and paranola (clinical history). IV. Status post donor after cardiac death organ (liver and left kidney) procurement.
- III. Acute phencyclidine intoxication (toxicology studies).

AND. Granger, M.D. tia / 2020 Examiner

Office of the Medical Examiner Monroe County New York



AUTOPSY REPORT Case Number: 20-00902 Pathologist: Nadia A. Granger, MD ME Case County/Origin: Monroe Co.

EVIDENCE OF RECENT MEDICAL/SURGICAL INTERVENTION: Evidence of medical intervention includes a triplelumen catheter in the lateral right neck (secured with clear adhesive); five electrocardiograph patches attached to leads on the bilateral and mid back; intravenous catheters in the bilateral antecubital fossae (secured with clear adhesive); ventral right forearm (secured with clear adhesive) and the ventral right wrist (secured with clear adhesive and tubing secured by a brace to the right hand and dorsal right upper arm); and a pulse oximeter lead on the left index finger tip.

On internal examination, there is hemorrhage into the right neck muscles surrounding the intravenous catheter and hemorrhage into the oropharyngeal mucosa, consistent with intubation.

EVIDENCE OF INJURY/RECENT TRAUMA:

DESCRIPTION OF BLUNT FORCE INJURIES:

HEAD AND NECK: On the forehead are multiple scabbed abrasions, up to 2 % inches. There is a ½ inch diameter black scabbed abrasion on the right cheek. On the left cheek is a 1 ½ x 1 inch scabbed abrasion.

On internal examination, there is patchy subgaleal and subscalpular hemorrhage lining the frontal scalp.

THORAX AND ABDOMEN: On the left upper back is a 1 ¼ inch scabbed abrasion. There is a ½ inch L-shaped red abrasion of the left lower back.

UPPER EXTREMITIES: On the dorsal right forearm is a ½ inch red-yellow abrasion. Scattered scabbed abrasions, up to 1 inch, are present on the dorsal right forearm. On the dorsal left upper arm are scattered scabbed red-yellow to black abrasions, up to 3 % inches in greatest dimension.

LOWER EXTREMITIES: On the anterior surfaces of the bilateral legs are scattered scabbed abrasions, up to 1 ¼ inches in greatest dimension. On the medial left heel is a 2 ¼ x 1 ¾ inch green contusion.

INTERNAL EXAMINATION:

BODY CAVITIES: The body is opened with a standard Y-shaped incision. No adhesions or abnormal collections of fluid are in any of the body cavities. Except for previously described organ procurement related findings, all body organs are present in normal and anatomic position with the exception of the right kidney (located in the pelvis) and the appendix (remotely surgically absent). The serous surfaces are smooth and glistening.

CENTRAL NERVOUS SYSTEM: The scalp is without laceration. The skull is intact. The brain weighs 1510 grams. The dura mater and falx cerebri are intact and not adherent to the surface of the brain. The leptomeninges are thin and delicate. There is no epidural, subdural, or subarachnoid hemorrhage. The cerebral hemispheres appear edematous with flattening of the gyri and effacement of the sulci. The structures at the base of the brain, including cranial nerves and blood vessels, arise normally and are free of abnormality. Sections through the cerebral hemispheres reveal slight left-sided deviation of the corpus callosum and right cingulate gyrus, consistent with early subfalcine herniation. There is diffuse dulling of the gray-white junction. The cerebral ventricles are of normal caliber. Sections through the brainstern and cerebellum show hemorrhage into the brainstern, consistent with transtentorial herniation.

NECK: Except as described in the "EVIDENCE OF RECENT MEDICAL/SURGICAL INTERVENTION" section, examination of the soft tissues of the neck, including strap muscles and large vessels, reveals no abnormalities. The hyoid bone and larynx are intact. Special examination of the tongue is unremarkable.





Subfalcine Herniation

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Subfalcine herniation is the most common type of cerebral herniation. In this image, the left brain has herniated under the free edge of the falx cerebri (arrow) because hemorrhage from an infarct in the middle cerebral artery has increased intracerebral pressure.

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Transtentorial herniation

The medial temporal lobe is squeezed by a unilateral mass across and under the tentlike tentorium that supports the temporal lobe. The herniating lobe compresses the following structures:

- Ipsilateral 3rd cranial nerve (often first) and posterior cerebral artery
- As herniation progresses, the ipsilateral cerebral peduncle
- In about 5% of patients, the contralateral 3rd cranial nerve and cerebral peduncle
- Eventually, the upper brain stem and the area in or around the thalamus

Subfalcine herniation

The cingulate gyrus is pushed under the falx cerebri by an expanding mass high in a cerebral hemisphere. In this process, one or both anterior cerebral arteries become trapped, causing infarction of the paramedian cortex. As the infarcted area expands, patients are at risk of transtentorial herniation, central herniation, or both.

Central herniation

Both temporal lobes herniate through the tentorial notch because of bilateral mass effects or diffuse brain edema. Ultimately, brain death occurs.

Upward transtentorial herniation

Upward transtentorial herniation can occur when an infratentorial mass (eg, tumor in the posterior fossa, cerebellar hemorrhage) compresses the brain stem, kinking it and causing patchy brain stem ischemia. The posterior 3rd ventricle becomes compressed. Upward herniation also distorts the mesencephalon vasculature, compresses the veins of Galen and Rosenthal, and causes superior cerebellar infarction due to occlusion of the superior cerebellar arteries.

Tonsillar herniation

Usually, tonsillar herniation is caused by an expanding infratentorial mass (eg, cerebellar hemorrhage). The cerebellar tonsils, forced through the foramen magnum, compress the brain stem and obstruct cerebrospinal fluid (CSF) flow.

Etiology

Brain herniation is a complication of a disorder that causes increased intracranial pressure (ICP). Increased intracranial pressure may be caused by

- Space-occupying lesions (eg, brain tumor, edema, or abscess; contusions; hematomas)
- Generalized swelling or edema of the brain (eg, due to acute liver failure or hypertensive encephalopathy)

- Increased venous pressure (eg, due to heart failure, obstruction of superior mediastinal or jugular veins, or venous sinus thrombosis)
- Obstruction of the CSF flow (eg, due to hydrocephalus or extensive meningeal disease)