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N. Burns (DOB 1-7-14)

Materials Reviewed:

I have reviewed the provided medical imaging examinations as tabulated below along with the available radiology reports and medical records.

Imaging Findings:

Chest 3-18-14 (1335 hr.): low endotracheal tube (ETT) esophagogastric tube (EGT); chest leads; left pulmonary opacities, atelectasis; Rad Report: ETT tip orifice right mainstem bronchus; left lung atelectasis.

Right leg 3-18-14 (1339 hr.): proximal tibial intraosseous catheter (IOC); osteopenia – poorly defined zones of provisional calcification (ZPC); Rad Report: IOC; otherwise unremarkable.

Abdomen 3-18-14 (1345 hr.): EGT, feeding tube; leads; osteopenia – poor ZPCs; Rad Report: NG tube; enteric feeding tube; nonobstructive bowel gas pattern; urinary bladder catheter.

Brain MR 3-18-14 (1717 hr.): axial, coronal DWI / DTI / ADC / eADC – no brain abnormality; bilateral large double-comparment extracerebral collections, T1 / FLAIR isohypointense, T2 / T2* hyperintense; smaller bilateral extracerebral collections T1/FLAIR isohyperintense, T2/T2* isohypointense about convexities, tentorium, falx, dural venous sinuses, veins, membranes, septations; additional smaller T2 hypointensities high convexities, vertex along veins (3-plane gadolinium T1 images); Rad Report (Prelim?): 2m/o PICU septic shock, dehydration, bulging fontanelle; no restricted diffusion for ischemia; no hydrocephalus; no abnormal enhancement; prominent subarachnoid spaces, can be normal within first year of life; Rad Report (final, addendum): benign external hydrocephalus; small extracerebellar, posterior extra-axial T1 iso-high, T2 high intensity early hemorrhage;

Chest 3-19-14 (0542 hr.): ETT higher; feeding tube; leads; right pulmonary densities, atelectasis; improved left lung; Rad Report: ETT slightly withdrawn, at carina; feeding tube, duodenum; gastric tube; improved left lung aeration; interval atelectasis right upper lobe.

Chest 3-21-14 (1756 hr.): ETT, EGT out; leads; improved lungs; Rad Report: now normal. (resolved bilateral atelectasis).

Chest 3-24-14 (2234 hr.): Rad Report: no acute; normal.

Cranial US 3-24-14 (2305 hr.): Rad Report (prelim): no visible intracranial hemorrhage; normal perfusion with color doppler; mildly prominent extra-axial fluid, unchanged; Rad Report (final): c/w MRI 3-18-14, normal.

US Pyloris 3-25-14 (2223 hr.): Rad Report (prelim): recurrent emesis; mild attenuation, spasm? Rad Report (final): normal pyloris, liver, spleen.

Brain MR 3-27-14 (1039 hr.): no restricted diffusion intensity abnormalities of brain; increased extracerebral collections; evolution of smaller extracerebral intensity abnormalities (T1 high intensities), including left high convexity, vertex veins; Rad Report: mild interval increase SDHs with mild mass effect; blood products more focally along left > right frontal, parietal lobes near vertex, increased slightly.

Skeletal Survey 3-27-14 (1408 hr.): osteopenia; poorly defined ZPCs; anterior rib flares; wide, poorly defined cranial sutures; Rad Report: no acute or healing fracture; proximal right tibial lucency, previous IOC.

Cranial US 3-30-14 (2129 hr.): Rad Report (prelim): H/O NAT, SDH on MRI, AMS change, CUS 3-24-14 WNL; Rad Report (final): bilateral extra-axial fluid collections consistent with subdural seen on MRI, interval increase right subarach. fluid, decreased right subdural collections.

General Discussion (CNS Injury):

The US and MRI findings are not specific as to causative pattern of injury and timing, and a differential diagnosis is required, including for an infant acute life threatening event (ALTE). Imaging cannot distinguish nonaccidental injury (NAI) from accidental injury (AI), or from predisposing or complicating medical conditions. According to the evidence-based medical literature, there is nothing about the imaging findings in this case, including in the presence or absence of retinal hemorrhage, that is specific for, or characteristic of NAI. Although shaking NAI may be a theoretical consideration, the current literature indicates that shaking alone (i.e. without impact) is unlikely to produce intracranial injury in the absence of requisite injury to the spinal cord, spinal column, or neck. Furthermore, the medical literature indicates that intracranial injury, as in this case, can result from short-distance impact scenarios (e.g. AI), or from medical conditions, including following a "lucid interval".

The differential diagnosis must also include predisposing or complicating conditions such as perinatal and birth-related issues; craniocerebral disproportion; developmental disorders; coagulopathy or vascular disease; metabolic, toxic, connective tissue, and nutritional disorders; infectious or post-infectious conditions; hypoxia-ischemia (e.g. airway, respiratory, cardiac, or circulatory compromise); seizures; and, multifactorial (e.g. synergistic cascade phenomena). Clinical deterioration due to a predisposing condition (e.g. coagulopathy or metabolic disorder) may be "triggered" by an infectious or post-infectious condition (e.g. recent vaccination), by hypoxia-ischemia (e.g. dysphagic choking or cardiorespiratory arrest and resuscitation), or "trivial" trauma (e.g. AI). Dysphagic choking or other forms of apnea may be associated with gastroesophageal reflux. Clinical deterioration, or death, occurring within the context of a recent vaccination should be reported to VAERS (Vaccine Adverse Event Reporting System of the CDC and FDA). In any of these scenarios, clinical deterioration may occur following a "lucid" interval. A complete and thorough medical workup is required including current and remote past medical history (e.g. perinatal, neonatal, and pediatric records), appropriate laboratory testing (extensive testing for coagulopathy, metabolic disorder, and vascular / connective tissue disorder), appropriate pathology testing (e.g. biopsy), and an assessment of the clinical management of the child, including medical or surgical interventions (e.g. spinal puncture or tap). Otherwise, with loss of life, the gold standard is a thorough and complete autopsy including neuropathology and extensive postmortem testing for the above differential diagnoses, as well as for timing of the injuries if possible. If the issue is AI vs. NAI, a biomechanical evaluation is indicated.

Case Analysis and Discussion:

According to the evidence-based medical literature, the US or CT findings of extracerebral collections cannot be precisely timed. Extracerebral collections may be subarachnoid, subdural, or both, and chronic rather than acute or subacute. This includes benign extracerebral collections and chronic subdural hematomas or hygromas that may be weeks to months old and can date back to, or before, birth. Such findings may be seen with craniocerebral disproportion (e.g. macrocephaly, cerebral underdevelopment), and head circumference measurements are important. Superimposed US-hyperechogenicity (or CT-hyperdensities) may represent more recent hemorrhages, re-hemorrhages, or thromboses. Infants with benign extracerebral collections and chronic subdural hematomas / hygromas are predisposed to hemorrhage or re-hemorrhage (e.g. along membranes or neomembranes) which may be spontaneous, associated with normal handling or trivial trauma, or triggered by acute-subacute medical conditions (e.g. venous thrombosis with perivenous hemorrhage). Only an MRI may provide more precise information regarding pattern of injury and timing.

With regard to the MRI findings in this case, the intensity characteristics indicate that the large T1/FLAIR isohypointense plus T2/T2* hyperintense extracerebral collections are likely weeks to months old and can date back to birth, or before birth (e.g. benign extracerebral collections or chronic subdural hematomas or hygromas). Some of the hemorrhages, re-hemorrhages, or thromboses are at least 2-3 days old (range 3-7 days, e.g. T1 hyperintensity with matching T2/T2* hypointensities), and others may be older (> 7-14 days, e.g. T1 and T2 hyperintense). The extracerebal "hemorrhagic foci" may be considered by some observers to represent acute-subacute traumatic injury. In fact, such imaging findings can represent venous thromboses with perivenous hemorrhage. Venous thrombosis may be related to thrombophilia, including vitamin D deficiency (vitamin D is antithrombogenic), and triggered by hypoxia-ischemia, trauma, infection, or fluid-electrolyte disorder (e.g. vomiting, dehydration, etc.). A CT venogram or a gadolinium-enhanced vascular MRI is often more reliable than a MR venogram in the definitive delineation of venous thrombosis

(e.g. dural venous sinus or cerebral venous involvement), especially when compared with other CT or MRI images. In this case, the MRI findings are entirely consistent with cerebral venous thrombosis superimposed upon chronic extracerebral collections with acute-subacute hemorrhages or re-hemorrhages.

The imaging abnormalities in this case indicate the necessity for a thorough hematology / coagulopathy and vascular workup beyond the simple "screening tests". This includes the hemophilic vs. thrombophilic states, as well as vascular anomalies and vascular fragility disorders (i.e. collagenopathy - e.g. vitamin C deficiency or depletion, osteogenesis imperfecta, Ehlers-Danlos syndrome) known to be associated with hemorrhages of this type.

Also, the findings on the followup imaging (as above described) likely indicate an increase in size of the older isohypodense collections due to the additional development of "subdural effusions" or some additional communicating hydrocephalus. With regard to the brain findings, no edema, swelling, or definitive brain injury pattern is seen on initial or followup imaging examinations. The absence of brain injury often indicates a favorable neurodevelopmental outcome.

Summary & Conclusions:

Medical imaging often cannot distinguish nonaccidental injury (NAI) from accidental injury (AI), or from predisposing or complicating medical conditions. There is nothing about the imaging findings in this case, including in the presence or absence of retinal hemorrhage, that is specific for, or characteristic of NAI.

In the context of these imaging findings, along with the widely acknowledged "lucid interval" phenomenon, whether post-trauma (AI or NAI) or associated with an evolving medical illness, it cannot be assumed that a given caretaker is responsible for such injury, particularly in the context of sibling or other child peer or pet factors. The is particularly important when the psychosocial evaluation of the caretaker(s) reveals no at-risk features for abuse. How complete was the medical evaluation in this case? Furthermore, in the absence of a specific medical diagnosis, NAI should not be the "default" diagnosis.

My opinions in this case are held to a reasonable degree of medical certainty and based on my clinical, teaching, and research experience in Pediatric Radiology and Neuroradiology over the past 35 years (CV, chronology of testimony, and references provided upon request). As more information becomes available, I reserve the right to submit an updated report. Please notify me at any time if I may be of further assistance.

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