Near-Drowning: Epidemiology, Pathophysiology and Imaging Findings

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Abstract

Although occasionly preventable, drowning is a major cause of accidental death worldwide, with the highest rates among children. A new definition by WHO classifies drowning as the process of experiencing respiratory impairment from submersion/immersion in liquid, which can lead to fatal or nonfatal drowning. Hypoxemia seems to be the most severe pathophysiologic consequence of nonfatal drowning. Victims may sustain severe organ damage, mainly to the brain. It is difficult to predict an accurate neurological prognosis from the initial clinical presentation, laboratory and radiological examinations. Imaging plays an important role in the diagnosis and management of near-drowning victims. Chest radiograph is commonly obtained as the first imaging modality, which usually shows perihilar bilateral pulmonary opacities; yet 20% to 30% of near-drowning patients may have normal initial chest radiographs. Brain hypoxia manifest on CT by diffuse loss of gray-white matter differentiation, and on MRI diffusion weighted sequence with high signal in the injured regions. This review presents the epidemiology and pathophysiology of nonfatal drowning and a detail description of the most common multisystem imaging findings.

INTRODUCTION

Worldwide, some half a million people die each year from drowning, and for each death, there are one to four drowning incidents serious enough to warrant hospitalization [1]. Organs such as the brain, lungs and kidneys are mainly affected by drowning accidents. However, the greatest permanent harm in drowning accidents is to the brain, which has negligible metabolic substrate reserves to subsist upon in the absence of continuous delivery of oxygenated blood. Much of the literature on near-drowning has concentrated on the respiratory effects of aspiration and on the management of both early and late respiratory complications such as aspiration pneumonia and adult respiratory distress syndrome. Thoracic imaging findings have been reported [2-4], but there is limited description of extra thoracic imaging manifestations seen in affected individuals. The aim of this article is to review the imaging findings and role of the different imaging techniques in the evaluation of the victim of nonfatal drowning.

EPIDEMIOLOGY

Drowning is a leading cause of unintentional injury death, accounting for 0.7% of deaths worldwide [1]. CDC analyses from National Vital Statistics System and National Electronic Injury Surveillance System for 2005-2009, indicated that each year an average of 3,880 persons were victims of fatal drowning and 34% of all fatalities were persons under 20 years old and almost four times more common in males. During the same period, there were on average 5,789 persons treated annually in U.S. emergency departments (ED) for nonfatal drowning cases, which included those classified as having a precipitated or immediate cause of “drowning/near-drowning,” and a diagnosis of “submersion”. Children under 4 years old accounted for 52.8% of the ED visits, and children aged 5-14 years accounted for 17.5%. Among children aged ≤4 years, 50.1% of fatal incidents and 64.6% of nonfatal incidents occurred in swimming pools. Drowning in natural water settings increases with increasing age group [5].

Given the variety and in consistency of definitions, particularly with terms like dry drowning and near-drowning, the WHO established in 2002 a new definition of drowning as, “the process of experiencing respiratory impairment from submersion/immersion in liquid”[6]. The drowning process begins with respiratory impairment as the person’s airway goes below the
surface of the liquid (submersion) or water splashes over the face (immersion). If the person is rescued at any time, the process of drowning is interrupted, which is termed a non-fatal drowning. If the person dies at any time because of drowning, this is termed a fatal drowning. Any submersion or immersion incident without evidence of respiratory impairment should be considered a water rescue and not a drowning [7].

**PATHOPHYSIOLOGY**

The process of drowning begins with the airway under water, leading to breath-holding; soon the person can no longer keep his or her airway clear, water entering the mouth is voluntarily spat out or swallowed. When the inspiratory drive is too high to resist, some amount of water is aspirated into the airways, and coughing occurs as a reflex response. Sometimes laryngospasm occurs, but in such cases, it is rapidly terminated by the onset of brain hypoxia.

As hypoxia, hypercapnia and acidosis ensue, the victim loses consciousness. This leads to larger fluid aspiration into the lungs, worsening respiratory and metabolic acidosis. The initial cardiovascular response to the process of drowning is tachycardia and hypertension, though the worsening hypoxia and acidosis leads to bradycardia, pulmonary hypertension and decreased cardiac output. Atrial fibrillation and other cardiac arrhythmias may occur, progressing finally to aystole [8-11]. The process from submersion of the victim to cardiac arrest usually occurs in seconds to few minutes, although in very unusual cases reported in ice water, this process can last up to one hour [12,13].

Hypoxia appears to be the single most important abnormality in death resultant from submersion in water, while acidosis and hypercarbia may contribute secondarily [11,13,14]. Hypoxemia is initially due to apnea but once fluid aspiration occurs, acute lung injury develops secondary to the effects of surfactant dysfunction and washout, increased capillary permeability, alveolar collapse, atelectasis, ventilation-perfusion mismatch and intrapulmonary shunting [7,11,15-17].

The greatest permanent harm in submersion accidents is to the brain. The brain is sensitive to the timing, duration, and intensity of hypoxia. Irreversible injury develops in the hippocampus, basal ganglia, and cerebral cortex within 4–10 min. Resuscitation at this stage may manifest in memory, movement, and coordination disorders. Only a few additional minutes of hypoxia may result in persistent coma or death [17-19] (Table 1).

**IMAGING FINDINGS**

**Chest**

Initial imaging evaluation of near-drowning victims is typically performed with conventional chest radiograph. The chest radiograph remains a rapid, simple and cost-effective method of initial evaluation of these patients. From the earliest reports on this subject the similarity between imaging manifestations of near drowning and pulmonary edema have been recognized. The classic papers by Rosenbaum et al and later by Hunter et al., identify three basic imaging patterns: a) normal radiograph with no obvious abnormality, b) perihilar pulmonary edema, and c) generalized pulmonary edema with more diffuse involvement throughout the lung bilateral fields [4,20]. The most common radiographic finding is that of diffuse, hazy ground-glass and alveolar opacities throughout the bilateral lungs. These tend to coalesce in the perihilar and medial lung zones with sparing of the most lateral portions of the lung including the bases and the apices (Figure 1a). The distribution tends to be bilateral, but may also be asymmetrical with more extensive involvement of the right or left lung (Figure 1b). This imaging presentation is macro and microscopically indistinguishable from that of pulmonary edema, with alveolar hemorrhage, interstitial edema and dilation of alveolar spaces found on histopathological examination [21,22]. Air bronchogram is appreciated in those areas with denser air-space disease. In the more severe cases, consolidation may extend to the entire parenchyma of both lungs, with little or no areas of sparing. Parenchymal opacities tend to worsen in the initial 24 to 48 hours before starting to resolve (Figure 1c and 1d). In most cases there is complete clearing of the lungs between 3-5 days. Persistent infiltrates after the first week may represent superimposed bacterial pneumonia or adult respiratory distress syndrome [23].Diffuse areas of ground-glass attenuation characterize high resolution CT, which may be associated with interlobular septal thickening configuring a crazy-paving appearance (Figure 2). Nodular and patchy areas of denser air-space consolidation can also be appreciated, randomly distributed but predominantly in a central distribution [24]. In a multidetector CT study of 28 drowned subjects, Levy et al., reported ground-glass opacities with septal lines (89%), with

![Figure 1](image.png)

**Figure 1** Chest radiographs of three different patients with non-fatal drowning. Figure 1a: 25-year-old female with non-fatal drowning episode (swamp water). Bilateral airspace disease and consolidation is noted, denser in the central and parahilar regions. Figure 1b: 4-year-old female with non-fatal drowning (swimming pool). AP view of the chest demonstrates diffuse bilateral parenchymal opacities in the bilateral lungs, more confluent in the right side. Figure 1c and Figure 1d: 26-year-old male with non-fatal drowning. Initial chest radiograph shows ground-glass opacities in the bilateral lungs. Follow-up radiograph 24 hours later shows progressive bilateral airspace consolidation.
a predominant apical and parahilar distribution and severe dense consolidation in one third of cases [25]. Pneumothorax, pneumomediastinum and pneumopericardium are additional findings seen in some patients (Figure 3). Kim et al., detected interstitial pulmonary emphysema and pneumomediastinum on thin-section CT in 2 of 6 patients, while Wunderlich et al., observed bilateral pneumothoraces in 1 of10 near-drowned children [2,24,26]. Small bilateral pleural effusions have been reported in 10% of drowned subjects, but are less common in near-drowning patients [25].

Fluid from different types of aspirated content is commonly seen within the trachea and main bronchi in drowned individuals [27,28] (Figure 4). On post-mortem CT study in drowned subjects, central airway fluid was highly prevalent (93%) which in as much as 50% contained high attenuation sediment. This high attenuation sediment may also affect second order bronchi [25]. Sand aspiration during drowning and near drowning may occur; which can be identified on chest radiograph and chest CT as radiodense material within the affected bronchi (“sand bronchogram”) [3,26].

Between 20% and 30% of near-drowning patients may have normal initial chest radiographs [2,27]. This generally represents less severe cases, with good prognosis, but this is not always the case, since abnormal parenchymal opacities may develop later. Patients with obvious pulmonary symptoms with normal initial radiographs have been documented.
Associations between “Takotsubo” or stress cardiomyopathy and near-drowning syndrome have been suggested. A stressful situation like a near-drowning episode may result in significant sympathetic nerve activation and massive catecholamine release that may induce left ventricular wall dysfunction and chest pain, mimicking an acute myocardial infarction. Echocardiogram in such patients reveals left ventricular enlargement and abnormal wall motion, with an apical ballooning pattern as well as electrocardiogram changes typical for Takotsubo cardiomyopathy [29-31]. Experimental animal models have demonstrated massive release of catecholamine in drowning, associated with severe myocardial necrosis [32].

Paranasal sinuses and mastoid air cells

Fluid in the paranasal sinuses and bilateral mastoid cells is reported in all fatal drowning subjects. In some of them high attenuation material has been noted layering in the dependent portion of the maxillary and sphenoid sinuses on CT, consistent with inhaled high-density sediment [3,25] (Figure 5). In our experience, this is also a common finding on CT of the head in non-fatal drowning victims, with air-fluid levels detected within the different paranasal cavities, particularly in the sphenoid and maxillary sinuses.

Abdomen

Probably the most common abdominal finding in drowning and near-drowning victims is increased amount of gastric fluid with high attenuation content and distended stomach (89% of cases) [25] (Figure 6a). Occasionally, high density within the gastric lumen, reflecting aspirated sand or sediment can also be appreciated on plain films [3]. In patients with extensive pneumomediastinum and pneumothorax, free air may be seen extending into the peritoneal cavity [26].

Abdominal visceral involvement in near drowning victims is rare, but cases of acute kidney injury with acute tubular necrosis and multisystem organ failure have been documented [33]. In a series of 30 adult patients with near-drowning episodes, Spicer et al reported 50% developing acute kidney injury [34,35]. Fluid overload during resuscitation, as well as acute heart failure and acute kidney injury may be responsible mechanisms for the presence of periportal edema identified on the abdominal CT in some patients (Figure 6b and 6c).

Brain

There are complex mechanisms involved in causing the brain damage from drowning with hypoxia and ischemia being the primary contributory factors. Hypoxic-ischemic insult cause different injury patterns in infants, older children and adults. Imaging appearance also depends on the severity of the insult.

Severe asphyxia in infants causes involvement of basal ganglia, thalami, brainstem and perirolandic cortex. Relative sparing of thalami and perirolandic cortex is seen in children between 1 and 2 years of age with severe asphyxia [36].
Severe insult in older children and adults causes involvement of basal ganglia, thalami, sensorimotor cortex, visual cortex, cerebellum and hippocampi [37,38].

Mild to moderate insults cause involvement of the watershed zones (regions between the anterior cerebral and middle cerebral arterial territory and between middle cerebral and posterior cerebral arterial territory). Both cortex and white matter are involved [39].

CT shows diffuse loss of gray-white differentiation, effacement of the cerebral sulci and decreased density of basal ganglia and other structures (Figure 7). Up to 80% of initial CTs are normal [40] (Figure 8). Occasionally basal ganglia hemorrhagic infarction may occur, but usually several days after the acute event. A “reversal sign” can be seen within 24 hours, which appears, as increased attenuation of white matter in comparison to the gray matter. The possible explanation of this sign is partial venous outflow obstruction from increased intracranial pressure [41].

The “white cerebellum sign” is another imaging feature described with severe hypoxic injury, which is seen as increased attenuation of brainstem and cerebellum relative to the cerebral hemispheres. Redistribution of blood to the posterior circulation is a possible explanation for this appearance [42].

In survivors, global parenchymal loss with ventriculomegaly and widened subarachnoid spaces is noted, usually after the second week [43]. CT has a strong predictive value for patient outcome and residual neurologic injury. Patients with an abnormal initial CT usually have worst prognosis, with higher mortality, permanent neurological damage or persistent vegetative state. Outcome is similarly poor for patients with a normal first CT who developed abnormalities on a later CT examination. In a study of 156 children with submersion injury, Rafaat et al., reported 100% mortality amongst patients with an abnormal initial CT, and 54% in those who developed abnormalities after a normal initial CT, with 42% presenting persistent vegetative state [40].

On MRI, diffusion weighted sequence shows the earliest findings with high signal in the injured regions and low signal on the ADC map in the same locations suggestive of diffusion restriction. High signal on the diffusion-weighted images peak at 3-5 days and then starts to become normal by the end of the first week which is known as “pseudonormalization”. T2-weighted and FLAIR sequences become positive after 24-48 hours and demonstrate increased signal. Cortical laminar necrosis is seen in the chronic stage with increased signal intensity on the T1-weighted images [44]. Imaging appearance of hypoxic-ischemic encephalopathy is different in preterm and term neonates; however drowning is a rare cause of hypoxic ischemic injury in this age group (Figure 9) [45].

MR spectroscopy (MRS) is another imaging modality for the detection of hypoxic-ischemic injury. MRS is more sensitive for injury than other MR imaging sequences in the first 24 hours [45]. Most common MRS findings include a decrease of N-Acetyl-Aspartate/Creatine ratio in 75.3% and presence of lactate in 65.5% [46]. In a series of 22 children with hypoxic encephalopathy after a near-drowning episode, generalized / occipital edema had the strongest correlation with poor outcome.

Additional indicators of poor outcome (death or persistent vegetative state) were high signal in the basal ganglia on T2-weighted images and abnormal signal intensity in the cortex on T2-weighted images (69% sensitivity, 100% specificity) and brainstem infarcts (25% sensitivity, 100% specificity). The best correlation with patient outcome seems to be in days 3 to 4, when an abnormal MR imaging exam has a positive predictive value of 100% for persistent vegetative state or death [47].

Cervical spine

Drowning and near-drowning victims may present associated traumatic injuries to the skull, facial bones, cervical spine, ribs, pelvis and extremities. In a series of 143 children involved in submersion episodes, Hwang et al reported 4.9% prevalence of traumatic injuries, all in the cervical spine. This type of injury
is particularly prevalent in near-drowning victims when the predominant mechanism of injury was diving. When considered across all ages the prevalence of cervical spine injury in drowning and near-drowning victims is low (less than 0.5%), but the incidence is significantly higher in adolescents (5%). This type of injury more commonly affects the lower cervical spine between C4 and C7 [48,49]. In non-fatal drowning victims, a complete work-up for the cervical spine injury should be obtained if there is concern for a diving accident (Figure 10). Lavelle et al reported a history or findings of abuse or neglect in 63% of pediatric bathtub near-drowning victims, including bruising, fractures and retinal hemorrhage [50].

**SUMMARY/CONCLUSION**

Imaging plays a paramount role in the initial evaluation as well as monitoring treatment response of near-drowning victims. A chest radiograph is commonly obtained as the first imaging modality, and typically reveals perihilar bilateral pulmonary opacities that tend to resolve in the first week. Brain hypoxia which more commonly affects the basal ganglia, thalami, brainstem, periorlancid and sensorimotor cortex manifest on CT by diffuse loss of gray-white matter differentiation, and by high signal intensity in the injured region on T2 weighted sequences, diffusion weighted MRI, and FLAIR, with some variation depending on patient's age. In those cases, in which the mechanism of injury includes diving, careful examination of the cervical spine should be part of the imaging protocol. Near drowning can present with varied imaging findings involving multiple organ systems, familiarity with imaging appearances is important to ensure prompt diagnosis and management.

**REFERENCES**


