

# PEDIATRIC SURGERY Update\* Volume 62 No. 01 JANUARY 2024

## **Contrast-induce Nephropathy**

Contrast-induced nephropathy (CIN) is characterized by an increase in serum creatinine or a decline in renal function, presenting within the initial 24 hours following intravenous contrast exposure for imaging, with peak effects observed up to five days later. The typical rise in creatinine is around 25-30% above baseline in CIN, occurring within 1-7 days and generally returning to baseline at 7-14 days. Permanent kidney damage is rare. In children, the incidence of CIN after intravenous urographic studies is approximately 15%, while in adults, CIN ranks as the third most common cause of hospital-acquired renal failure in 11% of cases.

The contrast used for radiological procedures relies on iodine for radio-opacity and can be either ionic or non-ionic. Ionic contrast mediums create high osmolality in blood, leading to red blood cell deformation, systemic vasodilation, intrarenal vasoconstriction, and direct kidney tubular toxicity. The pathophysiology of CIN involves renal medullary hypoxia, preglomerular vasoconstriction, and cytotoxic effects of the contrast material itself. Renal vasoconstrictors released by contrast, such as adenosine and endothelin, result in reduced medullary blood flow, leading to medullary hypoxia and tubular cell death. The degree of cytotoxicity is related to the length of exposure, high urinary flow rates, and is crucial after contrast procedures.

Known risk factors for CIN include hyperglycemia (diabetes), nephrotoxic drugs, chronic or intrinsic kidney disease, concurrent acute kidney injury, hypotension, volume depletion, repeated exposure to iodinated material, and pediatric kidney transplant recipients. Kidney injury after CIN occurs within minutes of exposure, with a rise in creatinine occurring a day or two later. Most cases are nonoliguric and mild, with a decline in serum creatinine within 3 to 7 days. Other manifestations of CIN include hyperkalemia, acidosis, and hyperphosphatemia, and fortunately, most cases are self-limited. Management is conservative, with dialysis needed in 1% of cases unless there is underlying renal impairment.

Mortality rates of 20% have been reported in adults, with an associated increase in hospital stay. The risk of CIN in hospitalized children with stable mildly diminished renal function is low. It is crucial to assess renal function before contrast material administration in children, considering factors like serum creatinine and estimated GFR. In emergencies, a thorough history should rule out risk factors such as diabetes, volume depletion, and concurrent nephrotoxic drug use. Prevention strategies involve determining the contrast volume based on the child's size, with intravenous fluids (hydration), typically isotonic saline, remaining

the cornerstone for CIN prevention. No approved drugs for CIN prevention exist, and the additional benefits of drugs like N-acetylcysteine, statins, ACE inhibitors, angiotensin-II receptor blockers, and vitamin C remain inconclusive. Iodixanol is the least nephrotoxic low osmolar agent available. In dialysis patients, iso-osmolar contrast agents are preferred to reduce the chances of volume overload. Major contraindications for iodinated contrast agents include a history of allergy, impaired renal function, and thyrotoxicosis.

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## **Perioperative Respiratory Adverse Events**

Perioperative respiratory adverse events (PRAE) constitute a significant source of morbidity and mortality among children undergoing anesthesia and surgery. These events pose a considerable safety concern due to their association with an elevated risk of cardiac arrest, prolonged hospital stays, and increased long-term mortality. Several independent risk factors, including age, weight, anesthesia duration, ASA class, pain score, and surgical season, contribute to the development of PRAE. Children classified as ASA grade II and III exhibit an augmented risk of experiencing PRAE, and a comprehensive history of risk factors obtained from patients and families serves as a reliable predictor for adverse respiratory events.

Immunologic markers of allergic sensitization do not reliably predict adverse respiratory events. Common colds and upper respiratory infections (URI) are prevalent in children, exhibiting seasonal variations and heightened incidence during colder months. URI stands out as the most frequent reason for surgery cancellation in children, with rhinovirus being the predominant cause. Respiratory syncytial virus infection, a more severe form of respiratory illness, warrants careful risk-benefit analysis before proceeding with anesthesia and surgery, given its potential to induce airway inflammation, increased secretions, airway susceptibility, and bronchial hyperreactivity.

Performing anesthesia in children with URI elevates the risk of PRAE by a factor of 30%, particularly for complications such as laryngospasm, bronchospasm, desaturation, and breath-holding, with greater susceptibility in infants and premature children. Studies indicate that endotracheal tubes pose a higher risk of PRAE compared to less invasive

airways like laryngeal masks or face masks. Propofol, with its effective airway reflex blunting properties, is considered an ideal agent for induction in children at an increased risk of PRAE. Total intravenous anesthesia with propofol demonstrates lower PRAE rates compared to inhalation anesthesia with sevoflurane in healthy children following the removal of the laryngeal mask during an awake state.

Obese children and adolescents, prone to various comorbidities, exhibit increased susceptibility to respiratory complications, airway obstruction, hypoxemia, and difficult mask ventilation. Respiratory comorbidities, including bronchial hyperreactivity and symptoms of severe asthma, heighten the risk of PRAE. The use of short-acting opioids, nonopioid analgesics, regional anesthesia, noninvasive airway management, protective mechanical ventilation, complete neuromuscular blockade reversal, and awake extubation are recommended strategies to reduce the incidence of PRAE in obese children. Postoperative care, continuous monitoring of oxygenation and ventilation, and diligent attention to recovery further contribute to preventing PRAE in this population.

Children with sleep-disordered breathing, encompassing conditions like sleep apnea syndrome, central sleep apnea, and nocturnal hypoventilation, also face an increased risk of PRAE. Notably, the majority of these cases are associated with tonsillectomy and adenoidectomy, suggesting the need for in-hospital overnight observation. While uncomplicated children with mild URI can usually proceed with elective anesthesia, guidelines recommend delaying elective anesthesia for moderate to severe URI by two to four weeks, and for respiratory syncytial viral or lower airway infections, the delay should be at least six weeks. The COLDS score serves as a predictive tool for assessing the risk of PRAE.

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## **Esophageal Anastomotic Stricture**

Anastomotic stricture (AS) is characterized by a narrowing at the esophageal anastomosis, as identified through barium contrast study and/or endoscopy. This condition is associated with significant functional impairment and symptoms. Among children who undergo

operative repair of esophageal atresia, AS is the most common complication, occurring in 20-50% of cases. While specific predictors exist, a substantial number of patients develop AS without apparent risk factors. Predisposing factors for AS include prematurity, low birth weight, VACTERL association, anastomotic leakage, anastomotic tension, increased esophageal gap length, type of suture material, traction technique repair, use of transanastomotic tube, and gastroesophageal reflux disease (GERD).

Vascular compromise affecting the lower esophagus, particularly in its segmental blood supply, contributes to AS. Excessive mobilization risks devascularization, and no correlation has been found between acid suppression duration and stricture formation. Prophylactic acid reflux therapy does not reduce the incidence of esophageal stricture post-esophageal atresia repair. Symptoms in children with AS include feeding difficulties, drooling, regurgitation, vomiting, coughing, choking during feeding, apneic spells, foreign body impaction, and poor weight gain. These symptoms may also be associated with other conditions such as esophageal dysmotility, tracheoesophageal fistula, tracheomalacia, gastroesophageal reflux, swallowing incoordination, laryngeal cleft, or vocal cord dysfunction.

The initial workup for suspected AS in symptomatic children involves a contrast esophagogram, followed by flexible upper endoscopy and rigid or flexible tracheoscopy as needed. Esophagograms are often performed between 5 and 10 days after esophageal atresia repair to rule out anastomotic leak. The Esophageal Anastomotic Stricture Index (EASI) utilizes fluoroscopic evaluation to assess the upper gastrointestinal tract postoperatively, offering a simple and reproducible tool for identifying children at risk of AS. Dilation is the preferred treatment for AS, performed in symptomatic patients with radiologic evidence of stricture. Prophylactic dilation has no preventive benefit. The primary goal of dilation is to relieve symptoms, facilitate age-appropriate oral nutrition, and reduce the risk of pulmonary aspiration.

Two types of dilators are used: fixed-diameter push-type dilators (bougie dilators) and radial expanding balloon dilators. Balloon dilators exert radial forces and are single-use, while bougies are reusable. Balloon dilatation, frequently performed under fluoroscopy or endoscopy, is the most commonly used method. Severe gastroesophageal reflux disease (GERD) is observed in severe AS cases, requiring proton pump inhibitor (PPI) treatment to address esophagitis and acid damage. Intralesional steroid injection and mitomycin C application can be considered adjunct therapies. Esophageal stenting is a promising tool for refractory or recalcitrant AS. Surgical intervention may be required for non-responsive cases, often involving resection and re-anastomosis. Long-segment strictures may necessitate staged approaches, such as segmental resection and traction procedures. Other causes of esophageal stricture include congenital esophageal stenosis, disk battery ingestion, peptic and eosinophilic esophagitis, actinic or neoplastic esophageal stricture, and epidermolysis bullosa.

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