PEDIATRIC SURGERY *Update* ©
Vol. 38 No. 05 MAY 2012

Extravasation Injury

Extravasation injury (EI) during intravenous therapy is a significant source of morbidity, mortality and liability in children and adults. The spectrum of injury goes from mild irritation without inflammation to tissue necrosis depending if the agent extravasated is a vesicant, irritant or flare producing. Vesicants can produce tissue necrosis by absorption from local tissue and DNA binding. They should be administered by central vein route. Patients at increased risk of EI include the very young or old with impaired venous circulation and lymphatic drainage, and the critically ill child. Device related factors include metal needles, large gauge catheters, poorly secured IV cannulas, IV placed in antecubital fossa, dorsum of hand or near joint area, and catheter dysfunction by separation, breakage or dislodgement. Patients should be informed of risk of EI and encourage to notify the nurse of any change in sensation, swelling, leakage, pain, machine flow malfunction or burning. Management consists of immediate discontinuance of infusion, aspirate residual extravasated fluid and removal of the peripheral catheter. Affected extremity should be elevated. Avoidance of the extremity for future cannulation or blood pressure recording. Prompt consultation with the wound nurse should follow. Serial photographs are useful to monitor wound progress. Conservative local care is given and plastic and physical therapy are consulted as needed.

References:

1- Garland JS, Dunne WM Jr, Havens P, Hintermeyer M, Bozzette MA, Wincek J, Bromberger T, Seavers M: Peripheral intravenous catheter complications in critically ill children: a prospective study. Pediatrics. 89(6 Pt 2):1145-50, 1992
2- Gault DT: Extravasation injuries. Br J Plast Surg. 46(2):91-6, 1993
3- Kumar RJ, Pegg SP, Kimble RM: Management of extravasation injuries. ANZ J Surg. 71(5):285-9, 2001
4- Tong R: Preventing extravasation injuries in neonates. Paediatr Nurs. 19(8):22-5, 2007
5- Rose RE, Felix R, Crawford-Sykes A, Venugopal R, Wharfe G, Arscott G: Extravasation injuries. West Indian Med J. 57(1):40-7, 2008
6- Amjad I, Murphy T, Nylander-Housholder L, Ranft A: A new approach to management of intravenous infiltration in pediatric patients: pathophysiology, classification, and treatment. J Infus Nurs. 34(4):242-9, 2011

Hürthle Cell Neoplasms

Oncocytic or Hürthle cell adenoma and carcinoma represent less than 5% of all thyroid neoplasms in children and adults. The distinction between Hürthle cell adenoma and carcinoma is histologically determined by the presence of vascular or capsular invasion

in the latter. Hürthle cells are large polygonal eosinophilic cells with pleomorphic hyperchromatic nuclei and fine granular cytoplasm containing an abundance of mitochondria commonly associated with Hashimoto thyroiditis, nodular goiter and well-differentiated thyroid cancer. Hürthle cell tumors contain increased number of mitochondria with structural abnormalities resembling patients with mitochondrial disease or myopathy. Adenomas are more common than carcinomas. Hürthle carcinoma is more aggressive, produces more metastasis, has a lower survival rate and low uptake for radioiodine. Hürthle adenomas can be managed with hemithyroidectomy, while the carcinoma variety will need total thyroidectomy. Since most Hürthle cell neoplasms secrete thyroglobulin it can be used to detect recurrent disease. Hürthle cell tumor size is predictive of malignancy with adenomas on the average smaller than carcinomas (2 cm vs. 4 cm). Hürthle carcinoma has a prognosis that is reliably predicted by degree of invasion, tumor size, extrathyroidal disease extension, and initial nodal or distant metastasis. Molecular expression of high Ki-67 phenotype proliferative index correlates with recurrence and tumor-related mortality among Hürthle cell tumors.

References:

1- Chen H, Nicol TL, Zeiger MA, Dooley WC, Ladenson PW, Cooper DS, Ringel M, Parkerson S, Allo M, Udelsman R: Hürthle cell neoplasms of the thyroid: are there factors predictive of malignancy? Ann Surg. 227(4):542-6, 1998

2- Hoos A, Stojadinovic A, Singh B, Dudas ME, Leung DH, Shaha AR, Shah JP, Brennan MF, Cordon-Cardo C, Ghossein R: Clinical significance of molecular expression profiles of Hürthle cell tumors of the thyroid gland analyzed via tissue microarrays. Am J Pathol. 160(1):175-83, 2002

3- Stojadinovic A, Hoos A, Ghossein RA, Urist MJ, Leung DH, Spiro RH, Shah JP, Brennan MF, Singh B, Shaha AR: Hürthle cell carcinoma: a 60-year experience. Ann Surg Oncol. 9(2):197-203, 2002

4- Maximo V, Soares P, Lima J, Cameselle-Teijeiro J, Sobrinho-Simaes M: Mitochondrial DNA somatic mutations (point mutations and large deletions) and mitochondrial DNA variants in human thyroid pathology: a study with emphasis on Hürthle cell tumors. Am J Pathol. 160(5):1857-65, 2002

5- Besic N, Hocevar M, Zgajnar J, Petric R, Pilko G: Aggressiveness of therapy and prognosis of patients with Hürthle cell papillary thyroid carcinoma. Thyroid. 16(1):67-72, 2006

6- Bremer AA, Feldman BJ, Iezza G, Clark OH, Rosenthal SM: Report of a Hürthle cell neoplasm in a peripubertal girl. Thyroid. 17(2):175-8, 2007

7- Sippel RS, Elaraj DM, Khanafshar E, Zarnegar R, Kebebew E, Duh QY, Clark OH: Tumor size predicts malignant potential in Hürthle cell neoplasms of the thyroid. World J Surg. 32(5):702-7, 2008

Cloacogenic Polyps

Inflammatory cloacogenic polyps are very rarely found in the pediatric age. They arise from the transitional zone of the anal canal, but can extend proximally toward the sigmoid colon. Characterized histologically by marked hyperplasia of the muscularis mucosa with extension of smooth muscle and fibrous stroma into the lamina propria. The typical presentation of the patient is difficulty with defecation and passage of mucous and blood per rectum. The polyps can prolapse; this is due to the malfunction of the internal anal sphincter; and the smooth muscle that covers the rectum. During endoscopy they can appear polypoid with flat base. The polyps vary in size from 3-4 cm in diameter, and have a sessile appearance. Inflammatory cloacogenic polyp is related to solitary rectal ulcer syndrome and is most likely due to prolapse of the anorectal

transition zone. Cloacogenic polyps are not a neoplasm arising from a preexistent normal transitional epithelium but a nonspecific regenerative process. Management consists of endoscopic removal of the polyps. Those unable to be removed endoscopically or endorectal will need sigmoidectomy with low anterior resection.

References:

1- Ciriza de Los Rios C, Tomas Moro E, Garcia Duran F, et al: [Inflammatory cloacogenic polyps: a rare cause of rectal bleeding]. Gastroenterol Hepatol. 30(8):461-4, 2007

2- Calva-Rodriguez R, Gonzalez-Palafox MA, Rivera-Dominguez ME, et al: [Inflammatory cloacogenic polyp]. Rev Gastroenterol Mex. 72(4):371-5, 2007

3- Washington K, Rourk MH Jr, McDonagh D, Oldham KT: Inflammatory cloacogenic polyp in a child: part of the spectrum of solitary rectal ulcer syndrome. Pediatr Pathol. 13(4):409-14, 1993

4- Bass J, Soucy P, Walton M, Nizalik E: Inflammatory cloacogenic polyps in children. J Pediatr Surg. 30(4):585-8, 1995

5- Poon KK, Mills S, Booth IW, Murphy MS: Inflammatory cloacogenic polyp: an unrecognized cause of hematochezia and tenesmus in childhood. J Pediatr. 130(2):327-9, 1997

6- Siafakas C, Vottler TP, Andersen JM: Rectal prolapse in pediatrics. Clin Pediatr (Phila). 38(2):63-72, 1999

---

* Edited by: Humberto Lugo-Vicente, MD, FACS, FAAP
Professor of Pediatric Surgery, University of Puerto Rico - School of Medicine,
Rio Piedras, Puerto Rico. Director - Pediatric Surgery, San Jorge Childrens Hospital.
Address: P.O. Box 10426, Caparra Heights Station, San Juan, Puerto Rico USA 00922-0426.
Tel (787)-786-3495 Fax (787)-720-6103 E-mail: *titolugo@coqui.net*
Internet: http://home.coqui.net/titolugo

ISSN 1089-7739