Intravenous and Oral Hydration

Approaches, Principles, and Differing Regimens

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KEYWORDS

• Contrast-induced nephropathy • Saline • Forced diuresis • Furosemide • Bicarbonate

KEY POINTS

- Correct volume depletion before administration of contrast in all patients.
- Encourage diuresis with oral ingestion of water before and after contrast administration in all patients.
- In high-risk patients, intravenous sodium chloride is recommended, starting 2 to 4 hours before contrast administration and continuing for at least 6 hours after contrast administration.
- In high-risk patients, the use of sodium bicarbonate may be justified when time does not permit administration of sodium chloride.

INTRODUCTION

The administration of oral and intravenous (IV) fluids before, during, and after exposure to iodinated contrast media (CM) for the prevention of acute kidney injury (contrast-induced nephropathy [CIN]) has a long history in clinical medicine. It is now part of the guidelines of the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Intervention,[1] and European Society of Urogenital Radiology.[2] However, these recommendations do not specify the amount, type, or route of administration of fluid. Recent surveys suggest that only about two-thirds of high-risk patients receive hydration per the guidelines.[3] In this review, the rationale for the administration of fluid is discussed and then the results of clinical trials are considered, comparing different amounts, types, and routes of fluid administration.

TERMS

Before discussing the details of fluid administration, common terms used in this review require comment. The term volume, as used in volume expansion, refers to isotonic fluid. Several isotonic fluids are approved for use in clinical medicine: normal or 0.9% sodium chloride, D5W (5% dextrose in water), Ringer lactate, and so forth. However, only fluids containing sodium as the major solute are isotonic in the body. Furthermore, isotonic sodium-containing solutions have a volume of distribution in the extracellular space, approximately 20% of total body weight. On the other hand, the glucose in D5W is metabolized, leaving only water behind. The volume of distribution for water is 60% of total body weight. Three times more water is required compared with isotonic sodium solutions to produce the same expansion of the extracellular space. Therefore,

The authors have nothing to disclose.

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Intervent Cardiol Clin 3 (2014) 393–404
http://dx.doi.org/10.1016/j.iccl.2014.03.009
2211-7458/14/$ – see front matter

in practical as well as clinical terms, volume (whether depletion or expansion) refers to isotonic sodium-containing fluid.

The term hydration is frequently used in the literature to mean volume. It more correctly refers to water surfeits and deficits, as in overhydration or dehydration, respectively. As noted earlier, the volume of distribution of water is 3 times that of sodium. Therefore, more water needs to be administered to provide comparable expansion of the extracellular (including the intravascular) space.

PATHOPHYSIOLOGY OF CM NEPHROTOXICITY

CM causes nephrotoxicity through direct effects on renal tubule cells.[4–6] The higher the urinary concentration of CM and the longer the CM remains in contact with tubule cells, the greater the likelihood of injury. CM is fully filtered at the glomerulus, and the initial urinary concentration is identical to that of plasma. However, as CM moves along the nephron, solute and water are reabsorbed without CM, resulting in an increase in the concentration of CM within the tubule lumen. The concentration can easily increase to 100-fold the plasma concentration. The degree to which CM are concentrated in the tubule lumen depends on the amount of solute and water reabsorbed.

CM also can upset the balance between O_2 use and O_2 delivery within the kidney. The medulla receives only ~5% of the total blood flow to the kidney[7] but is the location of the thick ascending limb of Henle (TAH) and S3 segments of the proximal tubule, where active transport of sodium requires high amounts of adenosine triphosphate and oxygen. Because of the unique vascular arrangement of the vasa recta that supply blood to this area, an oxygen diffusion gradient exists between the closely approximated descending (O_2 rich) and ascending (O_2 poor) vasa recta. This situation results in diffusion of O_2 from the descending to the ascending vasa recta, which further reduces the absolute amount of O_2 delivered into the medulla. The net result is that under resting conditions, O_2 tissue levels in the cortex are ~40 mm Hg, whereas in the medulla, levels ~15 to 20 mm Hg are found. An increase in solute delivery to the TAH (as might occur with osmotic diuresis) or inhibition of active transport by the TAH (as might occur with the use of a loop diuretic) can dramatically alter O_2 consumption.[8]

CM, with osmolalities higher than plasma, result in the delivery of more solute to the loop of Henle and distal tubule sites (osmotic diuresis). This situation could increase O_2 consumption in those nephron segments. The delivery of more solute to the macula densa would activate tubuloglomerular feedback mechanisms, resulting in a decrease in blood flow to the vasa recta (mediated by increased adenosine). In addition, as the vasa recta enter the hyperosmolar environment of the medulla, water (but not CM) moves freely across the endothelium,[9] and the concentration of CM increases (up to 4-fold). At these increased concentrations, all CM cause vasoconstriction in the descending vasa recta.[10] The resultant decrease in blood flow would further upset the balance between consumption and delivery and contribute to ischemic injury.

Both direct tubule toxicity and vasoconstriction lead to the generation of reactive oxygen species and a decrease in nitric oxide availability, which amplify the damage to the tubule cell.[10]

RATIONALE FOR FLUID ADMINISTRATION

There are several mechanisms by which fluid administration could be protective of the kidney after exposure to CM. These effects depend on the amount, type, and timing of the fluid administration. These effects include:

1. Decrease in urine concentration of CM
2. Decrease in urine viscosity
3. Decrease in renal vasoconstrictive factors
4. Increase in antioxidant mechanisms
5. Decrease in O_2 consumption
6. Decrease in intra vasa recta CM concentration

Decrease in Urine CM Concentration

As noted earlier, the concentration of CM within the tubule lumen depends on the amount of solute and water removed from the urine as it moves down the nephron. In states of volume depletion, more solute and water are reabsorbed as a result of the actions of angiotensin II, aldosterone, and vasopressin. The concentration of CM increases proportionately. On the other hand, with volume expansion and suppression of angiotensin II, aldosterone, and vasopressin, less solute and water are reabsorbed, and the concentration of CM within the tubule lumen increases to a lesser extent. Thus administration of fluid, particularly when it corrects volume depletion and expands intravascular volume, can be expected to decrease the concentration of CM in the tubule lumen. Because the concentration of CM is 1 factor contributing to the direct tubule cell toxicity, a reduction in the incidence of CIN with volume expansion and high urine flow rates would be expected. Even in the euvolemic state, administration of a sodium load decreases sodium