Intractable Postoperative Nausea and Vomiting (PONV) After Bilateral Simultaneous Percutaneous Nephrolithotomy on a Horseshoe Kidney: Case Report of a Unique Cause+

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Percutaneous nephrolithotomy (PNL) has proven to be a minimally invasive surgical technique for stone disease, including complex kidney stones. Bilateral stone disease and stones in horseshoe kidneys are considered as complex stones, and their coexistence is a significant challenging situation for surgery. Yet, PNL is a safe and effective treatment modality for both situations.1,2 We introduce a case of bilateral simultaneous PNL in a horseshoe kidney, who had intractable postoperative nausea and vomiting (PONV). The cause seems to be unique, since no literature on such condition exists.

Keywords: Percutaneous Nephrolithotomy, Horseshoe Kidney, Stone Disease, PONV

A 37 years-old male admitted with bilateral flank pain and dysuria. A roentgenogram revealed bilateral multiple kidney stones, with a total burden of 300 mm² at right and 650 mm² at left sides (Figure 1). Ultrasonography revealed horseshoe kidney and bilateral hydronephrosis. In intravenous pyelograms (IVP) lower pole fusion was not contrasted and no ureteric filling was observed (Figure 2). No vascular abberations were considered on either side. The patient was otherwise healthy and his medical history did not reveal any gastrointestinal (GIS) disease. He was offered simultaneous bilateral and separate sessions PNL, among which he chose first but gave consent for both. Under general anesthesia, bilateral retrograde pyelograms were obtained, revealing anterior pelvis on both sides, a stone located at right ureteropelvic junction (UPJ) and narrowing of UPJ at right. The stone was thought as the cause of narrowing as no true anatomic obstruction was considered so open surgery was not planned.3 Two 6Fr ureteric catheters were advanced to renal pelvices and were fixed to a urethral catheter. An uneventful PNL was conducted in 40 minutes on right and 70 minutes on left sides. Because right UPJ was edematous with an obstructing stone, no additional procedures for UPJ obstruction was
considered intraoperatively. At left side, two stones adjacent to fusion area inside the isthmic pole could not be reached anyhow. Foley catheters of 20Fr were placed as nephrostomy tubes at both sides. No morphine, atropine or acetylcholine was used during surgery. Cephazoline 1 gr was administered for prophylaxis. After uneventful awakening, the patient received 80 mg meperidine intramuscularly and had silent 4 hours at ward. Then he complained of an acute mild periumbilical pain and nausea followed by vomiting, neither projectile nor bloody. Thorough physical examination of abdomen, chest and neurological system was normal as were hemoglobin, creatinin and electrolytes. He had no bowel movements and minimal tenderness on epigastrium. Pain at bilateral flanks were mild and tolerable and no fever was observed. Both nephrostomies were draining properly without any excessive hematuria. Standing roentgenogram and ultrasonography did not reveal air and/or collection in abdomen or retroperitoneum. He received trimethobenzamid 200 mg intravenously (IV) and a repeat dose 1 hour later, but this did not resolve the symptoms. After consultation by general surgery and gastroenterology, an abdominal computed tomography (CT) was obtained and pantaprazol 40 mg IV with ondansetron 8 mg IV were prescribed. Nausea was relieved for 2 hours but vomiting course did not change. No cause of acute GIS symptoms was revealed on CT.

Since no mechanical or chemical cause was identified as ethiology, an upstream diagnostic workup on PONV was undertaken. This revealed an unnoticed abnormal positioning of the nephrostomy tube inside the left kidney. It was leaning to and pushing the isthmic pole and causing an isthmic-pelvic bulge anteriorly, just where the two residual stones were located (Figure 3). As soon as the left nephrostomy tube was withdrawn by 3-4 cm, all GIS symptoms disappeared and never repeated. Late arriving tests did not reveal blood in stool. Patient did well after violent 14 hours postoperatively. Left and right ureteric catheters were removed at days 1 and 2, respectively; both nephrostomies were withdrawn at day 3 following antegrade nephrostograms. The patient was discharged with pantaprazol daily. Three months followup was uneventful.

PONV is not uncommon in both open and endoscopic procedures. Thorough workup including observation of the vomiting (action and content), physical examination together with radiological and laboratory tests are essential in order not to overlook any complications of surgery. If, as in our case, this approach fails to establish a definitive cause, backstream workup on patophysiology of symptoms may exert diagnostic role. Vomiting center of medulla lies at the level of dorsal motor neuron of vagus and controls and integrates action of emezis. Three main mechanisms are involved in stimulation: vagal or sympathetic stimuli from viscera, direct stimulation of chemoreceptor trigger zone (CTZ) or provocation by higher cortical centers.
A stepwise evaluation of all these possible mechanisms may be beneficial to reveal the cause of intractable PONV: Visual, odorus or similar psychologic inputs act on third mechanism, which were not the case in our patient. Chemicals, including opioids, acting on CTZ can stimulate emesis. Morphine and other opioids may delay gastric emptying and promote vomiting by direct action on CTZ, but this is confined to ambulatory patients and those with less pain. In our patient, opioid was considered as an unlikely cause as the dose and route of administration were quite safe and besides, nausea began 4 hours after the injection. No other medication involving CTZ was given. Vagal or sympathetic stimulation is the most likely reason for the intractable symptoms. Since celiac ganglion receives input from both renal and gastric viscera, they either may cause PONV. In kidney, afferent stimulus for nausea and vomiting arises by the distension of renal capsule. Since our patient had wide nephrostomies and ureteric catheters in both kidneys, acute distension by liquids was unlikely. On the other hand, forceful insertion of the left nephrostomy tube caused distention (but not perforation) of the collecting system (Figure 3). We consider that this caused a direct effect on renal capsule, provoking afferent sympathetic stimulus for PONV.

Both mechanisms suggested above are mostly supported by the dramatic cessation of symptoms immediately after repositioning of the tube to a non-stretched condition. Ineffectiveness of antiemetics on vomiting further supports consideration of a mechanical cause. Vomiting (and to a less degree, nausea) stemmed from the chemical stimuli would respond to postoperative ondansetron properly.

**CONCLUSION**

Surgeons and radiologists that place nephrostomy tubes are advised not to distend or stretch the collecting system with forceful insertion. If their patients have similar intractable symptoms following nephrostomy tube placement, determination of the exact positioning of the tube and repositioning to an unstretched condition may help, definitely with a thorough workup for other causes.

**REFERENCES**


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