

Received: 2020.12.11

Accepted: 2021.04.27

Available online: 2021.05.20

Published: 2021.06.28

Intra-Abdominal Hypertension-Induced Gastroesophageal Intussusception: A Rare Complication of Transurethral Resection of a Bladder Tumor

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Study Design A

Data Collection B

Statistical Analysis C

Data Interpretation D

Manuscript Preparation E

Literature Search F

Funds Collection G

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Conflict of interest: None declared

Patient: Male, 81-year-old
Final Diagnosis: Gastroesophageal intussusception
Symptoms: Diffuse tenderness and distension of the abdomen
Medication: —
Clinical Procedure: —
Specialty: Critical Care Medicine

Objective: Rare disease

Background: Gastroesophageal intussusception (GEI) generally develops in patients with risk factors. However, intra-abdominal hypertension (IAH) rarely causes sudden GEI in patients without known risk factors. Endoscopic or surgical intervention is generally performed to reduce GEI. However, when GEI is induced by IAH, intra-abdominal pressure (IAP) decompression can contribute to GEI reduction.

Case Report: An 81-year-old man who underwent transurethral resection of bladder tumor (TURBT) for hematuria from a bladder tumor located at the left lateral wall had a deteriorated general status and bladder perforation during surgery in February 2020. The perforated portion was coagulated and treated conservatively using a urinary tract catheter. He was admitted to our Intensive Care Unit (ICU) following surgery after undergoing computed tomography (CT). CT revealed free air, ascites, and intra/retroperitoneal edema due to perfusion fluid leakage, and a new GEI was documented. The GEI required reduction; however, since his IAP increased to 21 mmHg, IAH-induced GEI was diagnosed; ascites drainage for IAP decompression was performed. IAP decreased to 12 mmHg after drainage; on subsequent gastrointestinal endoscopy, the GEI had reduced. His condition improved with no recurrence of GEI, and he was discharged from the ICU on day 8. Since cystography findings on day 26 showed no leakage of the bladder, he was discharged from our hospital on day 31.

Conclusions: We report a case of IAH-induced GEI as a complication of perfusion fluid leakage during TURBT. GEI was reduced by IAP decompression by ascites drainage without endoscopic or surgical intervention.

Keywords: Case Reports • Cystoscopy • Esophageal Diseases • Intra-Abdominal Hypertension • Intraoperative Complications • Intussusception • Urinary Bladder Neoplasms

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/930426>

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Background

Gastroesophageal intussusception (GEI) is a disease that is likely to develop in patients with risk factors such as anatomical abnormalities or digestive disorders [1-3]. The development of GEI in patients without risk factors is rare [4]. GEI can cause serious complications, such as esophageal perforation [1]; therefore, an immediate reduction is required. However, while the treatment for GEI reduction generally requires endoscopic or surgical intervention [3,4], which is sometimes invasive, a less invasive procedure is desirable, when appropriate.

Intra-abdominal hypertension (IAH) develops when the intra-abdominal pressure (IAP) is increased. The sudden progression of fluid collection/edema in the abdominal cavity or retroperitoneum can cause IAH [5]. Transurethral resection of bladder tumor (TURBT) is an endoscopic surgical procedure that requires the reflux of perfusion fluid to secure the operative field. Since TURBT excises the bladder wall, it can complicate bladder perforation and perfusion fluid leakage [6]. A previous case report described a patient who had perfusion fluid leakage complicated by bladder perforation due to TURBT and developed IAH and associated serious conditions [5]. Drainage of the leaked fluid and subsequent surgery for the postoperative hemorrhage contributed to improved IAH, which had been associated with the patient's serious conditions. If the postoperative hemorrhage could have been controlled without surgical management, the IAH and associated conditions would have been effectively improved with only a drainage procedure [5].

We describe a rare case of sudden GEI in a patient without known risk factors. GEI was induced by IAH that was complicated by bladder perforation due to TURBT. Although our patient initially had serious conditions, including GEI, the problems were improved after performing only ascites drainage and without endoscopic or surgical intervention.

Case Report

Written informed consent for publication was obtained from our patient. An 81-year-old man presented with gross hematuria in early January 2020. He had a medical history of ophthalmic disease (glaucoma and cataract), current smoking (nearly 1 pack of cigarettes per day), and social drinking and was occupationally unemployed at that time. Since the hematuria had not improved for a month, he consulted a nearby medical clinic, and prostatic hypertrophy associated with hematuria was diagnosed. However, even though medications were prescribed (tamsulosin hydrochloride, carbazochrome sodium sulfonate hydrate, and tranexamic acid), the gross hematuria persisted, and the patient's symptom of lightheadedness worsened. He was eventually transferred to our Emergency Department in February 2020.

In the Emergency Department, the patient presented with tachycardia (heart rate, 92 beats per min), tachypnea (respiratory rate, 22 breaths per min), and conjunctival pallor. Laboratory examination revealed severe anemia (hemoglobin level, 3.3 g/dL) without coagulation abnormality or thrombocytopenia (prothrombin international normalized ratio, 1.06; activated partial thromboplastin time, 25.1 s; platelet count, 270 000/ μ L). Other laboratory examinations indicated mild renal dysfunction (urea nitrogen level, 40.9 mg/dL; creatinine level, 1.51 mg/dL). Plain abdominal pelvic computed tomography (CT) showed a bladder tumor located around the left orifice of the ureter with an intra-bladder hematoma, but without hydronephrosis of the upper tract. The size of tumor was suspected to be approximately 5 cm; however, since the tumor and the hematoma were poorly marginated, we could not estimate the exact size. The tumor was suspected to be malignant, but there was no obvious metastasis (Figure 1). Based on the findings of severe anemia and intra-bladder hematoma and bladder tumor, the patient was suspected to have hemorrhaging from the bladder tumor. Therefore, he was admitted to our hospital with a plan to undergo a semi-emergency surgery after a blood transfusion. He underwent a red blood cell (RBC) transfusion on days 1 and 2 (560 mL per day). Thus, the patient's hemoglobin level increased to 6.8 mg/dL on the morning of day 3. He was additionally transfused with 280 mL of RBCs and underwent surgery (TURBT and hemostasis) on day 3.

Surgery was initiated under spinal anesthesia. At the start of the surgery, the operative findings showed that the bladder was filled with an organized hematoma. Because the hematoma was rigid and difficult to irrigate, it was broken down into pieces and removed. However, since the patient gradually exhibited agitation during the surgery, general anesthesia with intubation and ventilator support was administered. Subsequent blood gas analysis showed mixed acidosis, hyperchloremia, and hyperlactatemia (pH level of 7.196; base excess, -6.3 mmol/L; PaCO₂, 56.9 mmHg; chloride, 117 mEq/L; lactate, 4.6 mmol/L). After the hematoma removal, a bladder tumor sized 5 cm located behind the left ureteral orifice was found. The pathological result was urothelial carcinoma. After tumor removal and hemostasis around the surgical field was performed, a 3-cm perforation on the right side of the bladder was documented. The perforation was suspected to be complicated during hematoma evacuation. After performing hemostasis for the perforated portion and placement of a urinary tract catheter into the bladder, surgery was completed. However, an additional transfusion of 280 mL of RBC contributed to an increase in hemoglobin level (9.3 mg/dL), indicating that there was only a small amount of hemorrhage during the surgery, even with the perforation. After undergoing a CT scan for further evaluation, the patient was admitted to our Intensive Care Unit (ICU) for postoperative management.



Figure 1. Preoperative computed tomography findings. (A) A hematoma and bladder tumor was suspected (white arrow). (B) No apparent abnormality without hematoma and bladder tumor was documented.



Figure 2. Postoperative computed tomography findings. (A) Ascites (white arrow) and edema around the intra/retroperitoneum (black arrow). (B) Ascites in the rectovesical pouch (white arrow), retroperitoneal edema (black arrow), and free air (gray arrow). (C) Axial view of gastroesophageal intussusception. White arrowheads indicate the lateral wall of the stomach. Black arrowheads indicate the lateral wall of the esophagus. (D-F) Coronal view of gastroesophageal intussusception. White arrowheads indicate the lateral wall of the stomach. Black arrowheads indicate the lateral wall of the esophagus.

Free air, ascites, and intraperitoneal and retroperitoneal edema due to perfusion fluid leakage were observed on CT, and a new GEI was documented (Figure 2). In addition, his physiological findings on ICU admission showed diffuse tenderness and distension of the abdomen, and the intra-abdominal pressure

(IAP), which was indirectly measured via the bladder with an instillation volume of 20 mL of saline, increased to 21 mmHg. Although we had concerns about the accuracy of the IAP level because of the bladder perforation, the patient was diagnosed with IAH owing to the leakage of perfusion fluid from

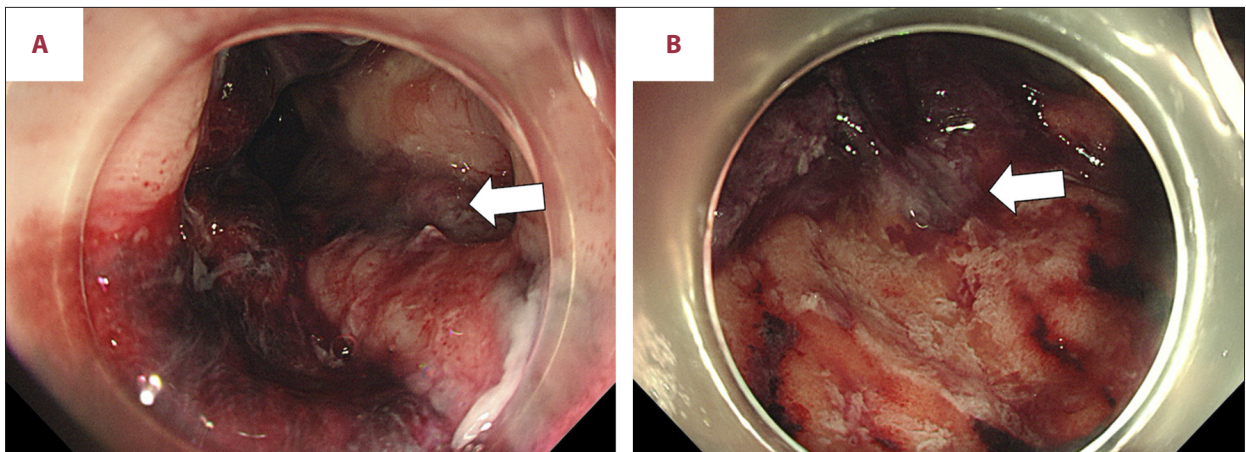


Figure 3. Endoscopy findings. (A) Erosions on the esophageal junction. (B) Erosions on the stomach fundus. White arrows indicate the erosions.

the perforated portion of the bladder that was used during TURBT. Anuria and retaining acidosis even after ICU admission were considered to be complications of IAH. Thus, the newly developed GEI required prompt reduction to avoid other complications. Therefore, we first performed ascites drainage for decompression of the increased IAP, and an improvement on IAH was expected. On draining 720 mL of ascites, the patient's IAP decreased to 12 mmHg. Next, for the GEI reduction, gastrointestinal endoscopy was performed. However, on gastrointestinal endoscopy findings, the GEI had reduced and erosion around the esophageal junction and stomach fundus was found (Figure 3). Thus, we concluded that the IAH-induced GEI had been reduced by the IAP decompression. To manage his anuria and acidosis, we initiated continuous hemodiafiltration after the ascites drainage. However, since the IAP decompression may also have contributed to the improvement in venous perfusion, the patient's urine output increased, and the acidosis improved within a short period. After these treatments, the patient's general status gradually improved. He was extubated on day 7 and was discharged from the ICU on day 8.

Although GEI-complicated erosion was documented, our patient started eating on day 10 and presented with no symptoms thereafter. The GEI did not recur or require any treatment throughout the patient's hospitalization. During the clinical course, the patient experienced a relapse of gross hematuria, and transurethral coagulation was required on day 11. However, while hemorrhaging from the wound from the tumor resection required electrocoagulation, granulation was observed around the perforated portion. Thus, the perforation was considered to be in the healing process after the conservative management approach. When cystography was performed on day 26, no bladder leakage was observed. The patient's overall condition improved, and he was discharged from our hospital on day 31.

For the patient's follow-up, we performed a contrast-enhanced CT scan 8 days after discharge. The CT findings revealed no obvious abnormalities, such as residual tumor and metastasis. In addition, there was no recurrence of GEI. Since the bladder was perforated during the TURBT and we were concerned about a risk of cancer cell dissemination, we planned to discuss the postoperative treatment plan, which included chemotherapy, with the patient. However, he did not come to our hospital on the next appointment day. Thus, he dropped out from the follow-up care.

Discussion

GEI has been reported to likely develop in patients with anatomical abnormalities, such as esophageal hiatal hernia, or post-myotomy for esophageal achalasia [1-3]. These risk factors had not been previously identified in our patient. However, to the best of our knowledge, even though age has not been previously reported as a risk factor for GEI, older individuals are at a high risk for developing esophageal hiatal hernia [7]. Thus, the age of the patient (81 years) could have been associated with the GEI. In addition, previous endoscopy-based investigations in patients with GEI have suggested that eating disorders, alcohol abuse, sudden sustained exertion, small bowel obstruction, acid bile peptic disease, and pregnancy are risk factors for GEI [1]. Also, GEI can be associated with vomiting or sudden IAP elevation [8,9]. Since vomiting itself is associated with sudden IAP elevation [10], a sudden increase in IAP may cause GEI. This is in line with our case.

Persistent GEI can cause blood flow obstruction and subsequent complications such as Mallory-Weiss syndrome or esophageal perforation [1]. While there have been no relevant studies carried out on patients with GEI, a study of patients with bowel intussusception who required surgery for reduction

suggested that patients with intestinal necrosis have significantly longer durations before reduction compared with corresponding patients without intestinal necrosis [11]. Thus, an immediate reduction is essential. To reduce GEI, endoscopic or surgical interventions are generally performed [1-3]. However, our patient required only ascites drainage to reduce GEI, without endoscopic or surgical intervention. A previous report described a case in which GEI developed in a patient with massive pneumoperitoneum due to iatrogenic colonic perforation. Though IAP was not measured in that case, the GEI was reduced only by laparotomy degassing, without any additional surgical treatment [4]. Therefore, similar to our case, IAP decompression can contribute to GEI reduction. According to the previous report and the experience in our case, IAH was considered to be a causal factor of sudden GEI in patients without risk factors.

IAH is defined as an IAP greater than or equal to 12 mmHg, while abdominal compartment syndrome is defined as an IAP greater than 20 mmHg with organ dysfunction [12]. In these conditions, immediate IAP decompression is necessary to avoid subsequent complications. Similar to our case, a previous report described a patient who presented with a bladder perforation as a complication during TURBT. In addition, the patient had respiratory and circulatory failure with fluid collection in the abdominal cavity, which induced IAH [5]. In that case, ascites drainage immediately improved the patient's condition. However, a subsequently exacerbated intra-abdominal hematoma led to repeated IAH and deterioration in the patient's condition, necessitating a laparotomy for the evacuation of the hematoma. In the present case, ascites drainage contributed to IAP decompression, GEI reduction, and improvement in anuria and acidosis. Since our patient had experienced little postoperative bleeding, no additional interventions for GEI and IAH were required. While no previous case of GEI complicated with TURBT has been reported, from our experience, IAH-associated complications including GEI caused by fluid leakage, can be managed by ascites drainage. In addition, if subsequent exacerbation factors such as postoperative bleeding are not

secondary causes of deterioration in patients, the drainage can be enough to improve the patient's physical conditions.

We used intra-bladder pressure as a measurement of IAP in our case. While the bladder perforation may have led to the leakage of instilled saline and less remaining volume in the bladder, we confirmed that the IAP increased to 21 mmHg. In addition, considering a previous study reporting that a lower infusion volume (10 mL) into the bladder resulted in lower measured IAP compared with higher infusion volumes (50 or 100 mL) in patients at risk of IAH [13], the IAP levels in our case might have been underestimated even when the measured levels were high enough (21 mmHg and 12 mmHg). Thus, the increased IAP might have caused the GEI in our patient.

IAH-induced GEI as a complication of TURBT is rare, especially in patients without known risk factors. However, bladder perforation is a known complication of TURBT [6]. In the present case, the ascites drainage would have been an initial treatment if it had occurred with the perfusion leakage. Further investigations are needed to clarify the frequency of occurrence of this condition and the adequate treatment in these cases.

Conclusions

We report a case of IAH-induced GEI as a complication of perfusion fluid leakage during TURBT. This patient's GEI was reduced by an improvement in IAH following IAP decompression using ascites drainage, without endoscopic or surgical intervention.

Acknowledgments

We would like to thank Editage (www.editage.com) for the English-language editing of this manuscript.

Conflicts of Interest

None.

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