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Case Report

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Vicarious Excretion of Intravenous Contrast in Gallbladder with Intact Kidney Function: A Case Report

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Abstract

Although the kidneys excrete most water-soluble contrast agents, their biliary excretion in patients with non-renal pathology is not well recognized. The definition of the contrast material in the gallbladder, a process also known as vicarious excretion, is a rarely reported event; it usually accompanies obstructive uropathy and kidney injuries. There are, however, case reports in literature of gallbladder enhancement and vicarious excretion in patients with normal blood creatinine levels, and no remarkable existing hepatobiliary problems. In the current case, a 43- year-old female patient with a suspected bowel obstruction received a diagnostic CT-imaging scan showing incidentally an abnormal contrast accumulation in the gallbladder. She had a general unremarkable medical history and did not develop signs of kidney injury while staying in the hospital. Thus, besides describing these radiological findings, we hope to give possible physiological explanations so that this incidental discovery could be better understood if and when found in any other case.

Keywords: Vicarious Excretion; Gallbladder Enhancement; Water-Soluble Contrast Agents; Biliary Excretion

Introduction

Water-soluble contrast media are primarily excreted in urine and little is known about biliary excretion in patients without impaired renal function. Vicarious excretion in the gallbladder is rare and usually found in patients with asymmetrically severe obstructive uropathy and renal failure. However, in very few case reports gadobenate produced gallbladder enhancement and vicarious excretion in the rest of the body in patients without renal failure or impaired hepatobiliary excretion [1,2].

Best of our knowledge, such findings have been recorded mainly by isolated case reports. To illustrate the context, we report cases of a 43-year-old woman referred for bowel obstruction in whom a diagnostic CT scan demonstrated unexpected pathology from accumulated contrast in the gallbladder. Here, we report these findings and suggest possible physiologic reasons as an incidental finding of normal variation, to contribute to the existing literature on the phenomenon.

Case Presentation

The 43-year-old female patient had no previous illnesses. She arrived at the facility with an acute onset of abdominal pain, nausea, and vomiting. Physical fares revealed tachycardia, tachypnea, dry mucosa, abdominal distension, and generalized tenderness. Small bowel obstruction is the prominent thought.

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An abdominopelvic CT with oral and IV contrast was ordered. The small bowel obstruction was confirmed by CT. Hydroelectrolytic therapy and conservative treatment were indicated with sequential CT scans; however, the patient had persistent small bowel obstruction in the second contrast CT scan performed within 24 hours of admission.

Decompressive surgery was therefore decided. Interestingly, the subsequent CT scan unexpectedly revealed the accumulation of contrast material within the gallbladder, a rare occurrence as shown in (figure 1,2). It is important to note that intravenous contrast agents are primarily excreted by the kidneys (90-99%), with a smaller amount eliminated through the hepato-biliary pathways, which may contribute to the formation of gallbladder stones.

To monitor the elimination of contrast from the gallbladder, additional examinations were conducted over one month, during which no traces of contrast were identified. Remarkably, the presence of intravenous contrast in the gallbladder appears to be purely coincidental and unrelated to the initial diagnosis of small bowel obstruction**.**

Discussion

Water-soluble contrast agents are commonly employed in radiology to improve the visualization of organs and structures. Although the kidneys serve as the primary excretory pathway for these agents, there are instances where alternative routes, such as the biliary tract, intestine, and stomach, may be involved in their elimination, a phenomenon known as "vicarious excretion".[3,5] When the biliary tract is involved in the excretion process, the contrast material can accumulate in the gallbladder and ultimately be released into the bile, resulting in enhanced visualization of the gallbladder. While rare, there have been reported cases of gallbladder excretion of water-soluble contrast agents in patients with normal renal function and unremarkable hepatobiliary evaluations [3,6-9]. Biliary excretion of contrast agents is an uncommon finding that is typically associated with obstructive uropathy and kidney injuries [10-12].

Heterotopic, vicarious biliary excretion of radiocontrast media is mostly determined by a variety of factors that include impaired renal function, longer recirculation time of radiocontrast media, high doses of iodinated contrast used in urography, the presence of gallbladder stasis, and increased protein binding in the presence of uremic acidosis.[13, 14] On CT imaging, gallbladder opacification (GBO) can obscure certain pathological or emergent gallbladder conditions such as neoplasms, stones, and hemorrhagic cholecystitis. This can result in diagnostic errors when vicarious renal excretion is mistaken for another disease that warrants unnecessary medical procedures.[15] A documented diagnostic error attributed to the misinterpretation of vicarious excretion of rectal iodinated contrast as a recto-vesical fistula therefore implies the existence of such a condition based on clinical presentation that must be kept at high index of suspicion by clinicians.[16] Moreover, GBO assessed by CT imaging has been reported as potentially able to predict patients with an increased risk for developing hypertransaminasemia, especially when the administration of contrast medium exceeds four days. Of importance, a positive correlation of delayed GBO with liver hypertransaminasemia has been reported.

In some cases, opacification of the gallbladder may not be noticed, and only enteric opacification may suggest vicarious excretion. Opacification of the gallbladder or colon after intravenous contrast although not specific usually implies impaired renal function. Evidence of vicarious excretion often requires delayed abdominal films or CT after intravenous urography or angiography and is detected more frequently by CT than by standard films because of its better contrast discrimination. The vicarious excretion thus seen should strongly suggest renal dysfunction.

We present the case of a 43-year-old lady who was admitted with complaints of abdominal discomfort, nausea, vomiting, bloating, and inability to pass flatus. Her physical examination revealed tachycardia, tachypnea, dry mucous membranes, abdominal distension, and generalized tenderness. Given the clinical background, small bowel obstruction was the most likely diagnosis. An abdominopelvic CT scan with oral and intravenous (IV) contrast confirmed the diagnosis.

Following the initial diagnosis, the patient was managed conservatively with hydroelectrolytic therapy. Despite treatment, a follow-up contrast CT scan within 24 hours showed persistent small bowel obstruction, leading to decompression surgery.

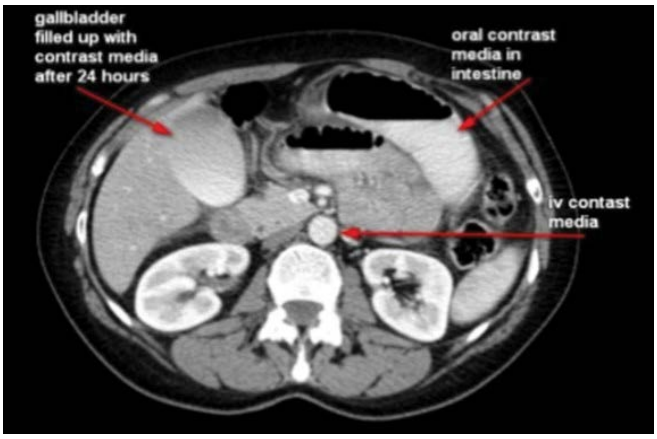

Figure 1: Gallbladder contrast CT, day 1.

Unexpectedly, a repeated CT scan revealed the presence of contrast material accumulated within the gallbladder. Typically, intravenous contrast agents are excreted primarily by the kidneys (90- 99%), with a smaller amount eliminated through the hepato-biliary pathways, which may contribute to

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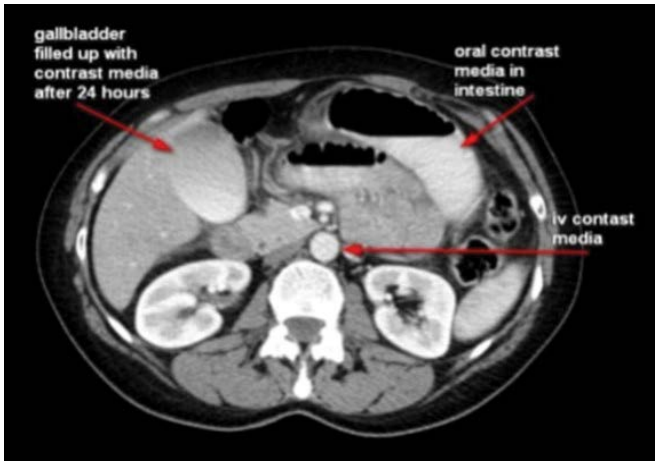

Figure 2: Gallbladder contrast CT, day 2.

gallbladder stone formation. A follow-up examination of the gallbladder showed no traces of contrast after one month. The presence of intravenous contrast in the gallbladder appears to be purely coincidental and unrelated to the initial diagnosis of small bowel obstruction.

Conclusion

This case report highlights the unusual finding of biliary enhancement on an abdominal CT scan in a patient with bowel obstruction and normal renal and hepatobiliary function. While the clinical significance of this finding remains unclear, further studies are necessary to elucidate its possible physiological mechanisms and determine its relevance in clinical practice.

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