JBR–BTR, 2011, 94: 348-349.

CORTISONE ASSOCIATED DIVERTICULAR PERFORATION

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Patients on glucocorticosteroid therapy are at increased risk of gastrointestinal perforation. The associated morbidity and mortality of perforations in this group is increased, compared with normal groups. This difference is due to the delay between onset of clinical symptoms and treatment. In the presence of steroids, gastrointestinal perforation is more difficult to diagnose clinically because signs and symptoms of perforation are masked by the anti-inflammatory effect of the steroids.

Key-word: Intstines, perforation.

Steroid associated diverticular perforation is a rare, potentially life-threatening complication of glucocorticosteroid therapy, associated with a high mortality and morbidity rate. Glucocorticosteroid (GCS) use induces both an increased risk for perforation, and a delay in diagnosis, secondary to the mitigated clinical presentation of peritonitis.

Case report

A 55-year-old man, who recently received high dose intravenous GCS therapy (3 x 500 mg of methylprednisolone) for an acute relapse of Multiple Sclerosis, presented himself at the emergency ward, suffering from dyspnea and abdominal discomfort. A chest X-ray demonstrated clear signs of a pneumoperitoneum with air under both diaphragms (Fig. 1A, B). The patient had no history of recent abdominal surgery nor blunt or penetrating trauma. C-reactive protein levels were elevated (67 mg/L, normal values < 5 mg/L) and there was a neutrophilic leukocytosis (16 x 10^3 white blood cells / mm^3, 76% neutrophils). An abdominal CT scan, performed after intravenous administration of iodinated contrast, confirmed the presence of free intraperitoneal air and demonstrated diverticulosis of the recto-sigmoid with overt signs of diverticulitis and a pericolic abscess collection due to a ruptured diverticulum (Fig. 2A, B) The patient was treated by emergency Hartmann surgery, confirming the perforated colon and abscess collection. Postoperative recovery and wound healing was without complications, despite the GCS therapy and the patient returned home 6 days after surgery with planned closure of the colostomy.

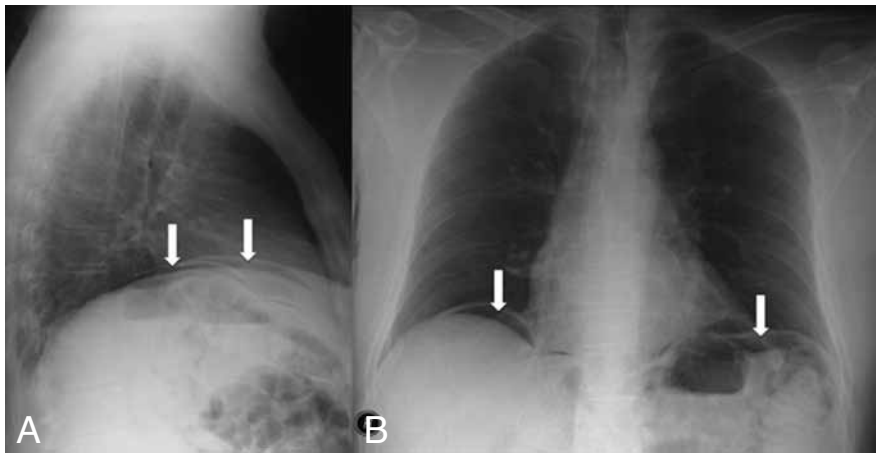

Fig. 1. — Chest X ray, demonstrating signs of free intra-abdominal air densities (white arrows), consistent with pneumoperitoneum on A: lateral X ray, B: frontal image.

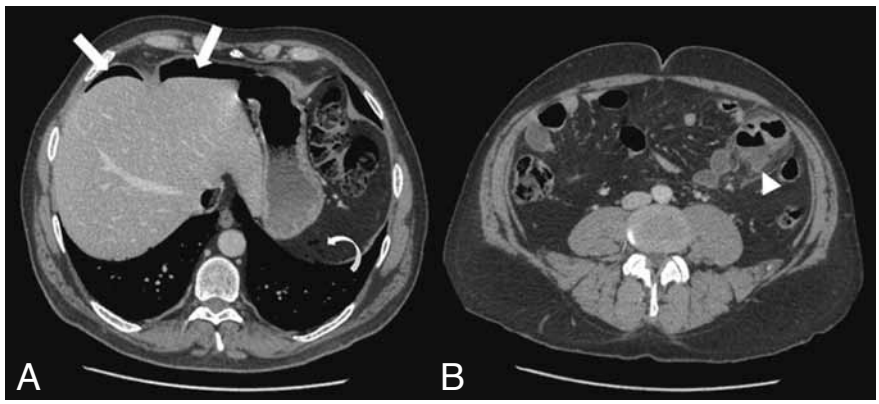

Fig. 2. — A. Confirmation of free air surrounding liver falciform ligament (white straight arrows) and in the intra-abdominal fat (curved white arrow). B: Peri-colic abscess collection (arrowhead).

Discussion

Although this case demonstrates evident radiological signs of a peridiverticular abscess with perforation, there was a vast radio-clinical discrepancy. Both the etiology of the intestinal perforation and this paucity of symptoms can be explained by the patient's history.

GCSs are known to be positively associated with an increased risk of

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perforated colonic diverticular disease (1, 2). The risk of steroid induced gastric and small bowel perforations was reported soon thereafter (3, 4). The positive relationship between colon diverticular perforation and GCS treatment is described to be more common in patients being treated with steroids for neurological disease, independent of steroid dosage (5). This increased risk, is explained by steroids causing intestinal atrophy, depletion of submucosal lymphoid patches and inhibition of the normal inflammatory reaction, leading to bacterial invasion, lack of containment and subsequent perforation (6, 7, 8). Others have suggested that steroids may cause direct colon mucosal injury (9).

Other diseases associated with an increased risk for perforation of the GI system include peptic ulcer disease, necrotic or ulcerated malignancies, iatrogenic injuries, traumatic injuries, diverticulitis, ischemia, inflammatory bowel disease, stercoral perforation or NSAID treatment (10).

In GCS treated patients, the mortality rates of intestinal perforation are significantly higher, reported to reach up to 100%, compared with a non-GCS treated group (11). This increased mortality is not only related to the underlying condition for which GCS are given, but mainly to the masking effect of the GCS on the clinical presentation of the perforation, resulting in a longer delay between onset of clinical symptoms and diagnosis or treatment (11). This delay in diagnosis and treatment has been shown to increase as steroid dose increases (12).

GCS use can decrease the leukocyte count and cause hypothermia, as well stop the inflammatory process responsible for the clinical expression of peritonitis. This masking effect of GCS appears to work by decreasing the number and availability of immunoreactive participants, depressing the chemotactic homing mechanisms to an area of insult and minimizing the necessary interactions required for cellular immune defense (11).

Clinically, abdominal tenderness is the only symptom that is consistently present among GCS treated patients with intestinal perforation. With more pertinent signs and symptoms subdued, the diagnosis of a perforation is often made from a serendipitous roentgenographic finding, as was the case in our patient.

The diagnosis of a GI tract perforation is based on direct CT findings, such as discontinuity of the bowel wall and extraluminal air, or indirect findings such as bowel wall thickening, abnormal bowel wall enhancement, abscess and an inflammatory mass adjacent to the bowel (10).

If signs of pneumoperitoneum occur on standard chest X-rays, MDCT evaluation should be the next diagnostic step to pinpoint the site of perforation, since it is beneficial to localize the perforation site for planning the correct surgery. MDCT can accurately depict the site of gastrointestinal tract perforation in 86% of cases (13). Extraluminal air bubbles, segmental bowel thickening and focal defect of the bowel wall are strong predictors of the site of perforation (13). The amount and location of free air varies according to the perforation site. If free air is located around the liver and stomach, this most likely indicates a gastroduodenal perforation. Free air detected predominantly in the pelvis and supramesocolic and inframesocolic regions makes perforation of the colon or appendix more likely (14).

Conclusion

Aggressive diagnostic efforts are mandatory in front of any persisting abdominal pain in patients on systemic steroid therapy. The diagnosis of a diverticular perforation should be considered in any steroid treated patient with abdominal discomfort, fever of unknown origin or unexplained leukocytosis.

References

1. Morris C.R., Harvey I.M., Stebbings W.S., Speakman C.T., Kennedy H.J., Hart A.R.: Anti-inflammatory drugs, analgesics and the risk of perforated colonic diverticular disease. *Br J Surg*, 2003, 90: 1267-1272.
2. Venditti D., Valerio B., Ielpo B., Buonomo O., Petrella G.: Bowel perforations in a patient affected by Churg-Strauss syndrome under high-dose steroid treatment: will alternative drugs reduce risk of surgery? *Rheumatol Int*, 2009 Dec. 12.
3. Glenn F., Grafe W.R., Jr.: Surgical complications of adrenal steroid therapy. *Ann Surg*, 1967, 165: 1023-1032.
4. Markowitz A.M.: The less common perforations of the small bowel. *Ann Surg*, 1960, 152: 240-257.
5. Fadul C.E., Lemann W., Thaler H.T., Posner J.B.: Perforation of the gastro - intestinal tract in patients receiving steroids for neurologic disease. *Neurology*, 1988, 38: 348-352.
6. Rigotti P., Van Buren C.T., Payne W.D., Peters C., Kahan B.D.: Gastro - intestinal perforations in renal transplant patients immunocompromised with cyclosporine. *World J Surg*, 1986, 10: 137-144.
7. Sterioff S., Orringer M., Cameron J.L.: Colon perforation associated with steroid therapy. *Surgery*, 1974, 75: 56-58.
8. Faro R.S., Corry R.J.: Management of surgical gastrointestinal complications in renal transplant recipients. *Arch Surg*, 1976, 114: 310-312.
9. Warshaw A.L., Welch J.P., Ottinger L.W.: Acute perforation of the colon associated with chronic corticosteroid therapy. *Am J Surg*, 1976, 131: 442-446.
10. Kim S.H., Shin S.S., Jeong Y.Y., Heo S.H., Kim J.W., Kang H.K.: Gastrointestinal tract perforation: MDCT findings according to the perforation sites. *Korean J Radiol*, 2009, 10: 63-70.
11. ReMine S.G., McIlrath D.C.: Bowel perforation in steroid-treated patients. *Ann Surg*, 1980, 192: 581-586.
12. DiSesa V.J., Kirkman R.L., Tilney N.L., Mudge G.H., Collins J.J., Cohn L.H.: Management of general surgical complications following cardiac transplantation. *Arch Surg*, 1989, 124: 539-541.
13. Hainaux B., Agneessens E., Bertinotti R., De Maertelaer V., Rubesova E., Capelluto E., Moschopoulos C.: Accuracy of MDCT in predicting site of gastrointestinal tract perforation. *AJR*, 2006, 187: 1179-83.
14. Stoker J., van Randen A., Laméris W., Boermeester M.A.: Imaging patients with acute abdominal pain. *Radiology*, 2009, 253: 31-46.