Stenting colonic neoplastic strictures is, nowadays, well recognized and largely used for preoperative decompression and palliation [1,2]. The main complications are immediate or delayed perforation, neoplastic or fecal obstruction, and migration. Ascites is not a contraindication to stent placement, rather the contrary, if the high risk of colostomy is taken into account [2]. To the best of my knowledge (and according to manufacturers and Pubmed), ascites infection, without perforation, has not yet been described.

A 77-year-old woman, in a good general state of health, suffered colonic obstruction that evolved over 3 days. Ultrasonography disclosed peritoneal cakes all over the peritoneum and moderate-volume ascites. Emergency colonoscopy attributed the occlusion to an obstructing, moderately differentiated carcinoma, located in the middle of the sigmoid loop. An enteral Wallflex stent, 20 mm in diameter and 90 mm in length, dilated the stenosis without difficulty. There was also a moderate, uncomplicated diverticulosis distal to the stenosis. Abundant fecal flow followed immediately after opening the stent.

The patient improved rapidly until the next morning, when diffuse abdominal pain contrasted with a normal transit. Ultrasonography failed to disclose any other abnormality, and particularly, any pneumoperitoneum. Urgent puncture obtained a liquid sample containing 16000 white cells per mm$^3$, with 20% polymorphonuclears (i.e. 3200/mm$^3$). Isolated cultures later showed rare Escherichia coli colonies that were sensitive to all the tested antibiotics. Blood counting was normal with 8800 white cells. C-reactive protein concentration was 220 mg/L. Despite rapid treatment with intravenous amoxicillin/clavulanic acid 1 g t.i.d., the patient’s condition worsened and she died 2 days later.

Correlation between stenting and infection seems very probable as the pain started the day after the procedure. Its mechanism is most probably translocation, taking into account the absence of pneumoperitoneum and the low number of colonies of a single species of bacteria. Wallflex stents are made of Nitinol, which expands more smoothly and slowly than earlier Wallstents made of stainless steel. The extremities are also soft and devoid of prickly wires.

In conclusion, the possibility of ascites infection without perforation should be taken into account, and preventive antibiotic treatment seems necessary when stenting a cancerous stenosis that is associated with ascites.

**References**


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