



Isolated primary Hodgkin's disease of rectum

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A 67-year old man arrived at our department with a one-year history of rectal tenesmus, bleeding and weight loss. A rectoscopy showed a large ulcerative lesion involving the posterior distal portion of the rectal ampulla and the upper third of the anal canal. The ulcer was infiltrating with necrotic tissue in the central area extending through the entire rectal wall; the margins were vegetating (Figure 1A). Tissue samples were taken. The histologic examination demonstrated a dense polymorphic infiltrate pushing apart the crypt epithelium with mucosal erosion. The infiltrate showed an irregular lower margin with infiltration of the muscularis propria. At high-power magnification the polymorphic

infiltrate appeared to be mainly constituted by small lymphocytes, eosinophils, plasma cells and several large blasts, mono or bi-nucleate with prominent nucleoli (Figure 2A). Immunohistochemistry for cytomegalovirus was negative, while blast cells showed the CD30⁺, CD15⁺, CD45⁺, EMA⁻ diagnostic profile required for diagnosis of Hodgkin's disease (HD) (Figure 2B). A diagnosis of rectal localization of HD was made. CT scanning was performed to investigate the possibility of lesions at other sites, but was negative. Chemical and hematologic parameters were within normal ranges and bilateral bone marrow biopsies were negative for infiltration.

The patient started chemotherapy with the MOPP

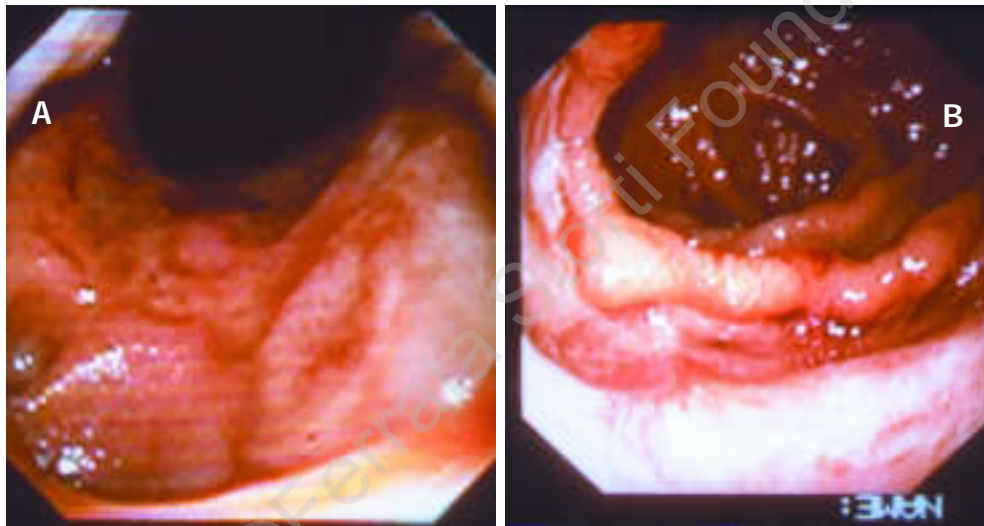


Figure 1. A. Pre-treatment endoscopic imaging. B. Post-treatment endoscopic imaging.

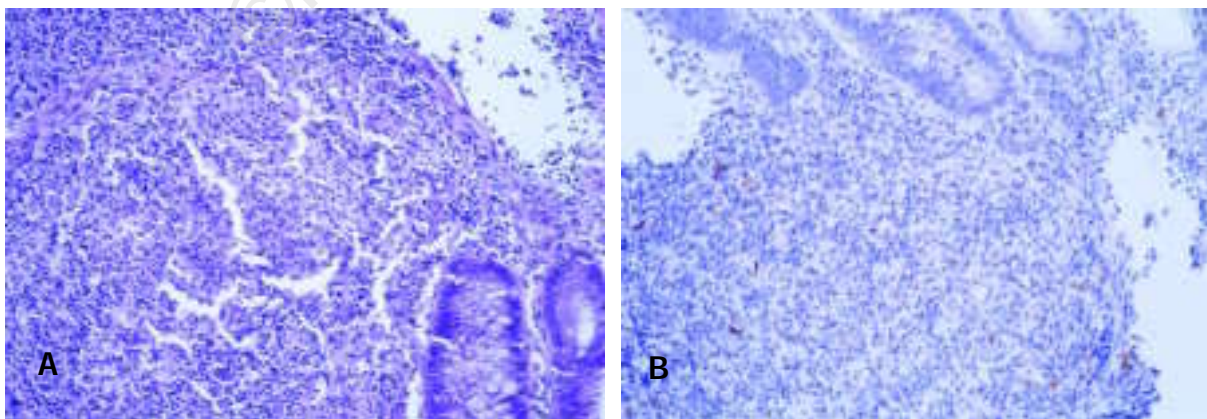


Figure 2. A. Submucosal polymorphic infiltrate mainly constituted by small lymphocytes, histiocytes, plasma cells, several eosinophils and several mono- or bi-nucleate blasts, with prominent nucleoli and abundant cytoplasm (hematoxylin and eosin; x250). B. CD30 immunostaining showing several Reed-Sternberg cells with characteristic membranous and cytoplasmic staining (avidin-biotin peroxidase method; x250).

protocol. Surgical debridement of the infiltrative lesion was excluded because the lesion was too near to the anal sphincter. After the first 2 courses of chemotherapy rectoscopy did not demonstrate any improvement in the lesion. The patient was considered unresponsive so radiation treatment was planned. He received pelvic irradiation with 3,600 cGy. Another rectoscopy, done after the end of radiotherapy, showed complete resolution of the lesion. Indeed, the ulcer had disappeared, and normal rectal mucosa covered the previously necrotic area (Figure 1B). A biopsy was not taken because of recently administered radiotherapy. CT scan confirmed the complete disappearance of neoplastic tissue in the anorectal wall. At present the patient is alive without any signs of local relapse or active HD.

This is a rare case of an isolated rectal localization of HD. As for the other cases reported in literature,¹⁻⁴ diagnosis was very difficult because of the atypical presentation of the malignancy, without other lymphadenopathy. An isolated rectal lesion usually raises the suspicion of other pathologies (e.g. isolated rec-

tal ulcer, carcinoma, rectocolitis), and only histologic study was helpful for the correct diagnosis. Our patient recovered after radiotherapy, as did the other cases reported in literature. This suggests that radiotherapy is the treatment of choice for this localization of HD, probably because of the difficulty of chemotherapeutic agents reaching peak therapeutic concentrations in rectal mucosae.

References

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