Enterobius vermicularis infestation masquerading as cervical carcinoma: A cytological diagnosis

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Abstract
Although prevalence of *Enterobius vermicularis* (EV) infestation in intestines ranges from 35% to 70%, its prevalence in female genital tract is not known despite several incidental findings. Acute inflammatory cells in the background of cervical Pap smear indicate infestation and should not be neglected as contamination. A 40-year-old woman presented with white vaginal discharge persistent for past 1 year. Local examination showed hypertrophied cervix with eversion of both lips and hard consistency of the anterior lip of cervix. A clinical diagnosis of cervical carcinoma was made. However, cervical Pap smear indicated EV eggs in an inflammatory background, treatment to which resulted in complete recovery.

Key words: Cervical smear, *Enterobius vermicularis*, pap smear

INTRODUCTION

*Enterobius vermicularis* (EV)/pin worm is a common helminthic parasite in intestines and is rarely observed in unusual sites.[1-3] Detection of EV in vagina or Pap smear is although unusual but is previously reported.[3]

*Enterobius vermicularis* infection is usually asymptomatic.[4] Pap test that is routinely used to detect epithelial abnormality can sometimes detect EV infections, evident by presence of characteristic cytomorphology of the eggs thus necessitating appropriate treatment.[5] The cytological/histopathological differential diagnosis are other parasitic ova, pollen grains, contaminated vegetable cells and fungal spores.[1,6]

CASE REPORT

A 40-year-old woman presented with white discharge per vagina since 1 year. Per-speculum examination showed hypertrophied cervix with eversion of both lips. Per-vaginal examination revealed hard consistency of the anterior lip of cervix. A provisional clinical diagnosis of cervical cancer was made. With the consent of the patient, conventional Pap smear was taken and stained with Pap stain. Pap smear was satisfactory for evaluation, showed endocervical cells as junctional component; clue cells were present and background showed neutrophils and coccobacilli. Two focal areas showed 2-3 eggs of EV that was oval/elongated in shape, flattened on one side, measured approximately $50 \mu \times 25 \mu$, had double contoured birefringent yellow to orange shell with coiled larvae within it [Figures 1 and 2]. Despite no significant history or family members having EV infestation and no signs/symptoms of oxyuriasis, cellophane tape test performed 5 times at different intervals did not show evidence of active parasitic disease. Patient was not immuno-compromised. Hence the diagnosis of the inflammatory smear with bacterial vaginitis and EV infestation was made. Patient responded well with antibiotics and anthelmintics and was symptom-free posttreatment for 1 year follow-up.

DISCUSSION

*Enterobius vermicularis* is the most prevalent human nematode worldwide.[1,3,7] The prevalence of intestinal enterobius ranges from 35% to 70%. However, the prevalence in female genital tract (FGT) is not known as only a few case reports are published. EV in FGT is uncommon and is
usually found as an incidental finding in routine Pap test, at surgery or at autopsy without signs/symptoms or evidence of active parasitization. In our case, it was an incidental finding in Pap smear.

The mode of infection is by direct ingestion of infected eggs by anus to mouth contact, contact with an infected person or contaminated objects. Indirect transmission occurs through airborne eggs dislodged from contaminated clothing or bed linen and inhaled with dust. The ingested eggs hatch and release larvae in small intestine and adult worm reside in caecum and colon. The adult female worm once fertilized descends into rectum and migrate to perianal and perineal region during the night usually in children. In female patients sometimes it enter vagina and lay eggs that mature in a few hours into fully developed infectious larvae. Self-infestation also occurs by retro-infection that is however not the usual route. The worm can migrate into cervix, endometrium, fallopian tube and may enter peritoneal cavity giving rise to vulvovaginitis, cervicitis, endometritis and salpingitis involving all organs of FGT. Peritoneal involvement has also been reported to happen by penetration of the gastrointestinal tract with preexisting disease especially in males. Other rare ectopic locations reported especially for female EV are prostate, urinary bladder, ureter, spleen, intestinal wall, liver, lungs, epididymis and conjunctival sac where diagnosis is possible only by histology.

Less than 30% of reported cases have clinical signs/symptoms such as perianal itching to severe life-threatening illness including fatalities in primates. The nongastrointestinal signs/symptoms are pruritus vulvae, urinary tract infection, vaginal discharge, postmenopausal bleeding, epididymitis, pelvic mass, tubo-ovarian abscess, salpingitis and generalized peritonitis by parasite/eggs.

In the present case, the patient presented with white discharge per vagina for 1 year.

In Pap smear presence of eggs with acute inflammatory cells in the background indicate infestation and should not be neglected as sampling contamination. The EV is diagnosed cytologically by the characteristic morphology of eggs because adult parasites are usually degenerated. The size of eggs is 55-60 µ in length and 25-30 µ in width and are flattened on one side. It has thick double birefringent refractile sheath containing larvae that appear granular or as curved structure. Sometimes empty shadow eggs or wrinkled shell enclosing clumped granular material are also observed. The lesions of EV is usually superficial granulomas with variable necrosis and fibrosis along with diagnostic eggs, granulation tissue, acute inflammation and sometimes degenerated adult parasite. Ova are considerably more resistant than the worm. The parasite usually presents as surface lesion and does not invade normal tissue. However, it may be seen in ovarian parenchyma where parasitization occurs immediately following rupture of the ovarian follicle. Multiple histologic sections are required in case of older fibrous lesions as a result of the inflammatory response to dead adult female worm. Usually, the cytology/histopathology finding is incidental. The active disease can be evaluated by cellophane/sootch tape for 5 times on separate days. Peripheral blood smear may show eosinophilia. In this case report, the Pap smear showed EV eggs having characteristics features. Surprisingly there was no evidence of either active disease or blood eosinophilia.

The differential diagnoses are the endometritis, salpingitis, appendicitis and peritonitis due to various causes. The granuloma of different etiology in FGT forms differential diagnosis. If the lesion presents as a mass, the differential diagnosis is either neoplasm or metastatic deposits. Two case reports are published in which the lesion resembled metastatic deposits in ovary in a case of in situ squamous cell carcinoma of the cervix. The parasites which form a differential diagnosis are Entamoeba histolytica, Microfilaria, Strongyloides stercoralis, Schistosoma haematobium, Trichuris trichiura, Ascaris ova and Tenia eggs. Pollen grains, vegetables and fungal spores may also be intrinsic or extrinsic sample contaminations. Pollen grains microscopically have two layers with grains enclosed inside. Although enterobiasis is of low pathogenicity, complication due to microbial co-infection occur as bleeding, infertility, tubo-ovarian abscess and generalized peritonitis. In our case, the provisional clinical diagnosis was carcinoma cervix, probably because of tissue changes due to chronic EV infestation and associated bacterial infection.

To conclude, diagnosing unusual infection in Pap smear is challenging but can be valuable to initiate appropriate therapeutics. Clinical awareness and knowledge of cytomorphology of this unusual entity is important in certain cases of vaginitis/cervicitis for accurate diagnosis and initiate proper treatment.

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Development of hyperplastic polyps following argon plasma coagulation of gastric antral vascular ectasia

Abstract

The etiology of gastric antral vascular ectasia (GAVE) syndrome or gastric hyperplastic polyps (HPs) is not fully understood. We report a case of gastric HP arising in a patient treated with argon plasma coagulation (APC) for GAVE syndrome. Despite unclear etiologic progression, this and previously reported cases suggest a temporal relationship between the treatment of GAVE and HP.

A 68-year-old male with a history of coronary artery disease, congestive heart failure and diabetes type II who initially presented with symptomatic anemia 2 weeks after starting aspirin and clopidogrel therapy. Diagnostic esophagogastroduodenoscopy (EGD) demonstrated diffuse GAVE. He was treated with 5 APC treatments, at 6-week intervals, over a 30 weeks period. 16 months after the initial APC treatment, an EGD performed secondary to persistent anemia demonstrated innumerable, large, bleeding polyps in the gastric antrum. Biopsy performed at that time confirmed hyperplastic gastric polyps. It has been proposed that HPs are regenerative lesions that arise at sites of severe mucosal injury. Our patient's treatment of GAVE with APC created significant mucosal injury, resulting in HP. Technique and genetic factors may have promoted hyperplastic changes during the regeneration of mucosa, at sites previously treated with APC. This case highlights the potential progression of GAVE to HP in a patient with persistent anemia after APC therapy.

Key words: Argon plasma coagulation, gastric antral vascular ectasia, gastric polyps, hyperplastic polyps