Perianal Skin Amebiases*

FIDEL RUIZ-MORENO, M.D.
Mexico City, Mexico

Nasse, in 1891, is credited with reporting, at the University of Berlin, the first case of skin amebiasis of the abdominal wall, which spread from an amebic abscess of the liver. Since then, numerous cases of cutaneous amebiasis have been reported throughout the world.

They have been divided into three clinical groups. The most common is the type in which the perianal skin becomes involved secondarily from amebic colitis. Another type is that in which there is invasion of the skin at operation or at sites of rupture with direct spread to the wound. Examples of this type occur in patients with amebic abscess of the liver, anorectal abscess, colostomy and appendectomy in a patient with amebic colitis. In the third type, an amebic cutaneous infection is blood borne and occurs at a distance from the liver and intestine.

Etiology

The causative organism of perianal skin amebic involvement is the Entamoeba histolytica. Only the trophozoite form is present in the skin disease. The cyst has never been found in extra-intestinal amebiasis.

Pathogenesis

In perianal skin amebiasis the following factors occur:

E. histolytica penetrates a dermal excoriation or fissure and extends into the cutaneous and subcutaneous tissues where the parasites multiply. Normal tissues do not admit invasion and multiplication of amebas. There must be a certain amount of moisture in the skin because the ameba disintegrates and dies in a dry environment. Incomplete or inefficient treatment of amebic colitis, poverty, malnutrition, chronic infections and uncleanliness are important predisposing factors.

To prevent amebic disease of the perianal skin, colonic surgery on an amebic patient should be preceded and followed by a thorough course of anti-amebic treatment.

Perianal skin amebiasis occurs in both sexes of all ages, and in all races. However, it is a rare complication of amebic colitis.

Pathology

Macroscopically, cutaneous amebiasis occurs as a fairly superficial ulcer with an irregular, reddish, granular base. Sometimes it is fungous, ulcerative and granulomatous, or it may occur as a mass. All types have a tendency to grow rapidly, deeply and superficially.

It is a necrotic lesion of the skin and subcutaneous tissues, with irregular, indurated or soft borders. The base is covered by a grayish-yellow sanguineous discharge and, when this is removed, a sensitive, bleeding, granulomatous area remains. Owing to secondary bacterial infection, the lesions are painful and foul-smelling.

Sometimes, when the lesion has been contaminated secondarily by bacteria, it becomes granulomatous, and a tumorlike mass may be formed which is covered by a purulent exudate. Microscopic examination reveals granulation tissue, great polymorphonuclear infiltration, microabscesses and numerous amebas in the trophozoite stage.

* Read at the meeting of the American Proctologic Society, Minneapolis, Minnesota, June 14 to 16, 1963.
Symptomatology

Usually pruritus ani precedes the appearance of small, confluent ulcers. The lesion is painful, discomfort increases with bowel movements, and the discharge is serous, semifluidulent or blood-stained. When the lesion becomes granulomatous and hypertrophic, a tender, fast-growing, soft, moist mass may appear.

Diagnosis

Skin amebiasis should be suspected when an anal or perianal postoperative wound fails to heal within a reasonable period of time in a patient who has had amebic colitis or in one whose stools contain the ameba or its cysts. Constipation may be the only clinical manifestation of amebic colitis. Microscopic examination of scrapings of the base or border of the ulcer will reveal amebae in the trophozoite form. Biopsy of the same sites, or of the perianal mass, will also reveal amebae in the same form. This is the only reliable diagnostic procedure.

Perianal skin amebiasis may be confused with other perianal pathologic changes such as those of lymphogranuloma venereum, syphilitic anal ulcer, tuberculous ulcer, condyloma, mycosis, leishmaniasis and basal cell carcinoma.

Treatment

Once the diagnosis has been made, we administer emetine, intramuscularly, as the treatment of choice. The dosage is 1 mg./kg. of body weight, using a maximum of 0.06 Gm. daily for 10 days. Electrocardiographic control during emetine treatment is advisable because this drug may harm the myocardium. Emetine should be followed by administration of antibiotic agents such as kanamycin, tetracycline, iodoxyquinoline, and arsenic acid derivatives to treat colitis. A combination of these agents will give the best results. It is imperative that the amebic colitis be treated vigilantly to prevent recurrence of skin amebiasis.

I have encountered seven patients with perianal skin amebiasis. The first was diagnosed on January 11, 1950, and the last on June 8, 1964 (Table 1; Fig. 1-10). During this period of 14½ years, I have seen

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Type of Lesion</th>
<th>Time Since Onset of Symptoms</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>28</td>
<td>M</td>
<td>Fungous, ulcerative</td>
<td>6 mo.</td>
<td>Lymphogranuloma venereum</td>
<td>Emetine</td>
<td>Cured, 9 da.</td>
</tr>
<tr>
<td>46</td>
<td>M</td>
<td>Ulcerative abscess</td>
<td>8 mo.</td>
<td>Cancer</td>
<td>Emetine</td>
<td>Died, 48 hr.</td>
</tr>
<tr>
<td>40</td>
<td>M</td>
<td>Ulcer</td>
<td>2 wk.</td>
<td>Anal ulcer</td>
<td>Emetine</td>
<td>Cured, 7 da.</td>
</tr>
<tr>
<td>43</td>
<td>M</td>
<td>Fungous, ulcerative</td>
<td>1 yr.</td>
<td>Lymphogranuloma venereum</td>
<td>Emetine</td>
<td>Cured, 10 da.</td>
</tr>
<tr>
<td>48</td>
<td>M</td>
<td>Ulcer</td>
<td>1 mo.</td>
<td>Postoperative anal ulcer</td>
<td>Emetine</td>
<td>Cured, 8 da.</td>
</tr>
</tbody>
</table>