CASE REPORT

Cutaneous malakoplakia: case report and review*

Malacoplaquia cutânea: relato de caso com revisão da literatura

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Abstract: Malakoplakia is a rare acquired disease that can affect many systems but is more common in the urogenital tract. Cutaneous malakoplakia is even rarer. It is far more frequent in immunodeficient patients. We report a case of cutaneous malakoplakia in a kidney transplant patient who had recently stopped receiving immunosuppressive therapy to illustrate a review of the relevant recent literature.
Keywords: Kidney Transplantation; Malacoplakia; Review; Skin

Resumo: Malacoplaquia é uma doença adquirida rara que pode afetar diversos órgãos e sistemas, mas é mais comum no trato urogenital. O acometimento cutâneo é ainda menos frequente. Atinge principalmente imunodeficientes. Relatamos caso de malacoplaquia cutânea em um paciente transplantado renal que havia recentemente deixado de receber a terapia imunossupressora, a fim de ilustrar uma revisão da literatura recente relevante.
Palavras-chave: Malacoplasia; Pele; Revisão; Transplante de Rim

INTRODUCTION

Malakoplakia is a term derived from the Greek, meaning "soft plaque".[1,2] The disease was first described in 1902 by Michaelis and Gutman.[3] It describes a granulomatous process of infectious etiology triggered by bacteria that occurs preferentially in subjects affected by primary or secondary immunodeficiency.[4,5] The pathogenesis of malakoplakia remains poorly understood, and it is thought to represent an acquired bactericidal defect of macrophages associated with infection, immunosuppression, and/or immunosuppressive agentes.[4,6,7]

The most common site of occurrence is the urogenital tract, although the condition has also been found to affect the gastrointestinal and respiratory tracts, retroperitoneum, thyroid gland, lymph nodes, bones/joints, middle ear, eyes and brain.[4-9] The condition has been considered rare, and cutaneous malakoplakia is even rarer; the first case was reported by Leclerc and Bernier in 1972.[10]

We report a case of cutaneous malakoplakia in a kidney transplant recipient and proceed with a review of the topic.

CASE REPORT

A 51-year-old white man from Brazil, suffering from idiopathic chronic renal failure, presented with a 2-year history of asymptomatic cutaneous lesion on the left groin, noticed by his nephrologist during hospitalization due to sepsis caused by catheter infection. The patient was frequently catheterized at this site since an unsuccessful kidney transplantation 2 years before.

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The lesion was a yellow-erythematous-purple plaque measuring around 1 cm in diameter, on the left groin, near a femoral vein catheter (Figure 1).

The lesion was sampled for histopathologic and culture studies. The culture results revealed the growth of *Providentia spp* and *Candida albicans*. Histopathologic analysis revealed a chronic inflammatory process characterized by sheets of closely packed macrophages containing PAS-positive inclusions (von Hansemann cells) and calcospherites known as Michaelis-Gutmann bodies, as demonstrated by Von Kossa stain, which shows the homogeneous bodies in black (Figures 2A, 2B e 2C). Prussian blue staining demonstrated the presence of hemosiderin inside macrophages, which may explain the purple color of the lesion (Figure 2D).

The patient was treated with surgical excision in association with sulfametoxazol-trimetoprin antibiotic therapy. No evidence of recurrence was detected on 3-year follow-up, as shown in figure 3.

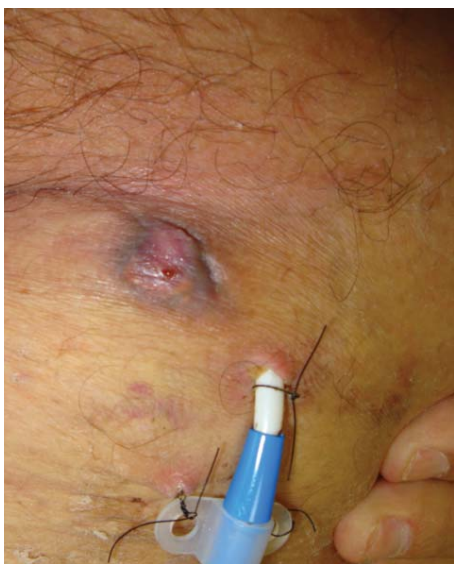

Figure 1: Malakoplakia: Picture of the yellow-erythematous-purple plaque measuring little more than 1 cm in diameter on the left groin of the patient near a femoral vein cateter

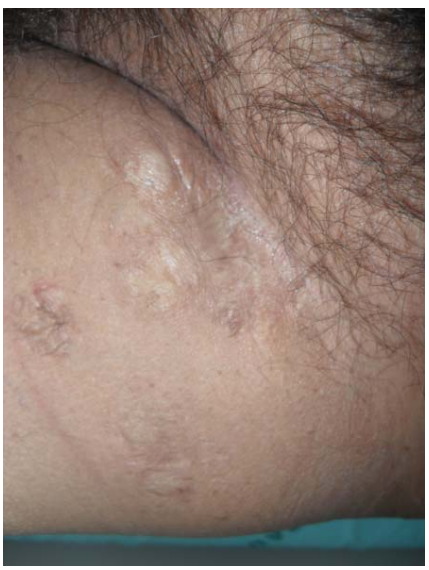

Figure 3: Malakoplakia: Left groin picture of the same patient after treatment and 3-year follow-up

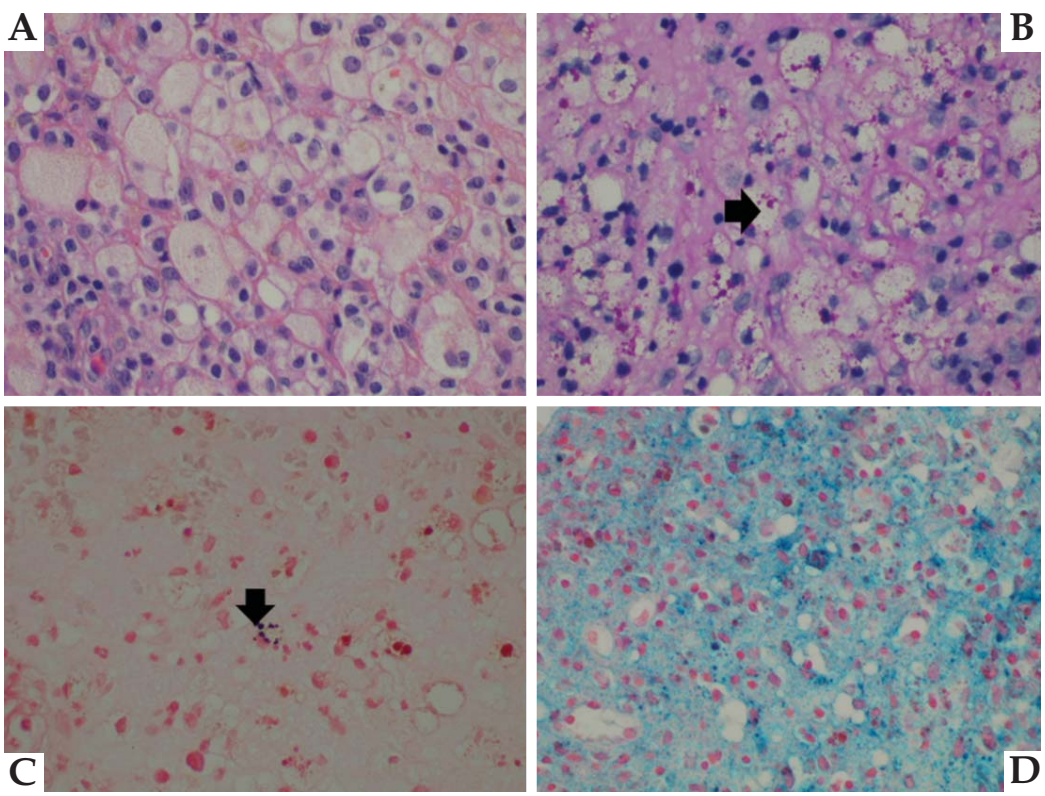

Figure 2: Malakoplakia: A-hematoxylin-eosin (400 X) stain showing the sheets of macrophages. B-von Hansemann cells in PAS stain (400 X)(black arrow). C-Michaelis-Gutmann bodies, shown in black after Von Kossa staining (400 X)(black arrow). D-Prussian blue demonstrates hemosiderin inside macrophages (400 X)

DISCUSSION

Malakoplakia is a rare granulomatous disease in which a defect of the killing capacity of macrophages after endocytosis is considered to be the central event. Disturbed phagosome-lysosome fusion was suggested, but it is still not clear how and why this disorder happens, and the hypothesis is not fully accepted.[11-13] We reviewed published articles in indexed periodicals that appeared in a PubMed search performed using the term "cutaneous malakoplakia". Based on Kohl *et al.* 2008, we added cases of cutaneous malakoplakia published from January 2006 until January 2012, as demonstrated in chart 1.[14]

While the majority of subjects are immunodeficient patients, including HIV-infected patients, patients with neoplasia, transplanted patients and others, more recently cases involving previously healthy patients have been reported.[15-17] Almost all transplanted patient cases refer to kidney recipients, as the one in this article, but two were reported in heart transplant recipients.[18,19] There are few reports in which prevalence among women is higher (2:1).[20,21] The age peak occurs between the sixth and seventh decades, being even rarer in children.[17,22]

Approximately 90% of patients have coliform bacteria detected in urine, blood, or tissue, suggesting an infectious cause.[4] The most commonly found bacterium is *Escherichia coli*, but *Klebsiella*, *Proteus*, *Pseudomonas*, *Mycobacterium avium*, *Mycobacterium tuberculosis*, *Shigella*, *Staphylococcus aureus* and *Enterococcus* spp were also found.[23,24] *Rhodococcus equi* is the most commonly implicated microbe in HIV-infected patients.[25] In 75% of cases, the disease affects the genitourinary tract, but other systems have been implicated, including the skin.[26]

No typical clinical presentation is described, skin presentation varies from papules, plaques, nodules, abscesses with or without fluctuation, and fistula to ulcers, cystic and polypoid masses.[14] Therefore, the diagnosis is predominantly confirmed by anatomopathologic and culture studies. Vanbrabant *et al.2004* recently described the possibility of using 18-fluoro-deoxyglucose positron emission tomography for diagnosis and follow-up.[27]

Histopathologically, the pathognomonic finding of Michaelis-Gutmann bodies, which represent partially degraded bacterial organisms, can establish the diagnosis. Michaelis-Gutmann bodies are intracytoplasmic, round-ovoid, basophilic, concentric laminated inclusions in macrophages that are typically enlarged and display foamy cytoplasm and eccentric, hyperchromatic, round nuclei, denoted as Hansemann cells.

Differential diagnosis is possible with other infectious diseases or neoplastic and reactive/reparative processes. Infections to consider include tuberculosis, Whipple's disease, lepromatous leprosy, fungus (*Cryptococcus*), and parasites (leishmaniasis). Special stains for microorganisms and tissue culture are necessary. Reactive and neoplastic processes include Langerhans cell histiocytosis, fibrous histiocytoma, lymphoma, granular cell tumor, xanthoma, foreign-body granuloma, hemophagocytic syndromes, and sarcoidosis.[14] Although generally presenting benign self-limited evolution, a fatal outcome is possible, but none was described in cutaneous malakoplakia.[28] Pseudomalakoplakia was once described as a proliferation of histiocytes at a previous surgical site, but only as an abstract. No other publications on this theme are found.[29]

Our observation that the disease developed on a site of recognized trauma and contamination, in accordance with other related cases, highlights the importance of direct inoculation of bacteria in the pathophysiology, since the presence of immunosuppression is necessary, but not sufficient for its development.

There are no prospective comparative studies, probably due to the limited incidence, so approaches to management vary from surgical excision, with or without antibiotics, to the use of antibiotics alone.[8] Van der Voort *et al. 1996* compared treatments and concluded that surgical excision achieved the higher cure rate (90%), and that, when comparing antibiotics, quinolones seemed to be superior.[7] The discontinuation of immunosuppressives and treatment of HIV could also be helpful.[7,9] Sulfamethoxazole-trimethoprim is also cited as effective.[30] It was selected by us due to considerations such as cost, access and drug interaction.

We illustrated this article reporting a case of cutaneous malakoplakia synchronous to previous immunosuppressive therapy, in a subject with no current immunosuppressive treatment. The option for surgical plus antibiotic treatment resulted in cure with no recurrence to date. We also reviewed the literature and counted the reported cases of cutaneous malakoplakia described on chart 1 (including this one).[31-66] ❑

CHART 1: Reported cases of cutaneous Malakoplakia

Reported Cases of Cutaneous Malakoplakia in the Literature*

Case No.	Reference No.	Age/Sex	Location	Gross	Medical History
1	5	51 y/M	Perianal, inguinal, scrotum	Nodules and ulcerations	Kidney Tx
2	5	67 y/M	Right temple	Nodule	Kidney Tx
3	33	69 y/F	Right axilla	Ulceration and mass	RA, breast carcinoma
4	34	40 y/F	Inguinal, broad ligament	Ulceration	N/A
5	10	64 y/M	Perianal	Indurated mass	RA
6	35	35 y/M	Left eyelid	Nodule	Kidney Tx
7	1	64 y/M	Perianal	Ulceration	Lymphoma
8	36	75 y/F	Vulva	Ulceration	RA
9	37	50 y/F	Abdominal wound	Polypoid mass	N/A
10	38	31 y/M	Right axilla	Mass	HIV
11	9	32 y/M	Abdomen	Abdomen Abscess	Kidney Tx
12	9	44 y/M	Perianal and left lung	Abscess	Kidney Tx
13	9	42 y/M	Right axilla	Chronic abscess	SLE
14	39	70 y/M	Buttock	Nodule	Chronic hepatitis C
15	40	75 y/M	Right hand and wrist	Abscess	N/A
16	41	41 y/M	Peritoneal, supraclavicular	Cystic mass and firm nodule	DM
17	42	74 y/M	Perianal	Nonhealing lesion	MPD
18	43	55 y/M	Gluteal cleft	Ulcers	HIV
19	44	67 y/M	Left neck	Mass	No significant PMH
20	45	81 y/F	Frontal mass	Irregular plaque	DM
21	46	56 y/M	Internal canthus of eye	Nodule	Sarcoidosis
22	32	44 y/F	Buttock	Nodule	Kidney Tx
23	47	60 y/F	Nasolabial sulcus	Ulceration	N/A
24	14	2 mo/M	Colorectal and perianal	Polypoid masses	Immunodeficiency
25	2	68 y/M	Left inguinal region	Papules	N/A
26	2	66 y/M	Right axilla	Nodule	RA, DM
27	48	53 y/F	Perineum	Papules	Kidney Tx
28	49	42 y/M	Inguinal region	Mass with ulceration	Lymphoma
29	50	41 y/M	Frontal scalp, right lung	Abscess, pulmonary lesions	HIV, Hepatitis B
30	51	64 y/F	Left neck mass	Mass with cavitation	Thyroidectomy
31	52	60 y/M	Gluteal fold	Cutaneous fistula	DM
32	53	62 y/M	Chest	Ulceration	N/A
33	54	65 y/M	Ureterocutaneous fistula	Abdominal fistula	N/A
34	19	51 y/M	Perianal	Nodule	Heart Tx
35	55	69 y/M	Left arm and flank	Ulceration, mass	Escherichia coli sepsis
36	56	55 y/F	Abdominal wall	Papules	N/A
37	57	22 y/F	Arm	Fluctuating mass	N/A
38	58	52 y/F	Inferior abdomen	Fistula with abscess	Kidney Tx
39	59	30 y/M	Perianal	Abscesses	Dermatomyositis
40	60	51 y/M	Left thigh	Mass with draining abscess	HIV, DM
41	16	60 y/F	Abdominal fold	Pink-yellow plaques	Healthy
42	17	23 y/M	Perianal	Pink nodules	Healthy
43	18	14 Y/M	Gluteal fold	Papule	Healthy
44	20	55 y/F	Right Labia	"Boil" (nodule + abscess)	Heart Tx
45	27	58 y/M	Perianal	Erosive plaque	Psoriasis
46	61	63 y/F	Abdominal wall	Fistula	Pulmonary sarcoidosis
47	62	83 y/F	Neck	Goma (nodule + fistula)	SLE, RA, Sjogren
48	63	45 y/F	Perigenital	Papules, nodules and sinuses	HIV
49	64	24 y/M	Abdominal wall	Fistula	Psoas abscess (Tuberculosis?)
50	65	87 y/M	Buttock	Scaly plaques and polypoid nodules	No significant PMH
51	66	66 y/M	Lower abdomen	Abscesses, nodules and fistula	Poor overall health
52	current article case	51 y/M	Left groin	Plaque	Kidney Tx

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