

## WHERE IS THE OUTRAGE?

### To the Editor:

The world's largest breast cancer advocacy organization, the non-profit Susan G. Komen for the Cure, recently held a conference in Alexandria, Egypt. Leading researchers from the U.S. and several mideast countries were meeting to discuss the latest research findings regarding breast cancer.

Dr. Mohammed Shaalan of Egypt's Breast Cancer Foundation told Reuters on Monday: "The week's events are a demonstration of the cooperation between countries, governments, civil society, advocates, survivors, and the global community as a whole." He further went on to say, "It shows that breast cancer has *no boundaries* and reveals *the beauty of the world's unity* in its fight against breast cancer." (my italics).

Indeed. These statements were uttered the day after all Israeli attendees were informed that their invitations were rescinded. No explanation for this action was given. The order was given by the Egyptian Health Minister Hatem el-Gabali.

Now, I – an American Jew – am presently in the position of having been invited to speak at the first Congress of Organ Transplantation to be held in November in Cairo, Egypt. My wife and I were very much looking forward to attending this meeting – particularly since my mother was born in Egypt – and I had yet to see the splendor of this historic country. In addition, I was slated to participate in a multinational cooperative to help the Egyptians set up a heart transplant/advanced heart failure center at Maadi Armed Forces Hospital to serve their population.

Given the severe action by the Egyptian government vis-à-vis Israelis attending the breast cancer conference, I have rescinded my offer to speak in Egypt. The racism that served as the springboard for Egypt's decision cannot and should not be tolerated. In the present era, after so much effort has been devoted and so

many lives have been sacrificed, how can the Middle Eastern Arab nations expect peace when Israeli doctors and scientists are excluded from a meeting whose sole purpose is to rid the world of breast cancer? If we cannot cooperate in saving lives wasted by cancer, how can we ever hope to cooperate in preventing further lives being lost through the horror of the Israeli-Arab conflict?

As a doctor, I am deeply disappointed. As a Jew, I am very worried. As a human being, I am utterly disgusted. Moreover, ADL national director Abraham H. Foxman urged the Susan G. Komen for the Cure to stipulate that this exclusion of Israeli breast cancer researchers is unacceptable. Initially silent, the Foundation announced several days later that "our efforts led to confirmation that all advocates would be welcome to participate in the events." Leading to further confusion, Nancy Brinker, founder and head of the Susan G. Komen for the Cure organization, claimed last week that despite threats in Egypt, Israelis were *not* barred from the Cairo conference. Well, which is it? The initial silence and later waffling are reprehensible, given the fact that Susan G. Komen – a victim of breast cancer and the organization's namesake – was a Jew herself. How ironic, pitiful, and tragic.

In short, the responses to the Egyptian Health Minister's declaration were pathetic. The medical world should be outraged that Egypt would pull such a stunt, and the Susan G. Komen foundation should be criticized for such a lame response. Everyone lauds the foundation's efforts on behalf of breast cancer victims. However, this achievement is irrelevant to the issue at hand: racism and the absence of a firm response to its ugly expression. Egypt should be ashamed, and Susan G. Komen for the Cure should be ashamed. The appropriate response on the part of Egypt would have been to have its Health Minister apologize to the Israeli scientists or, if he refused to do so, fire him. The appropriate response on the part of Susan G.

Komen for the Cure would have been to cancel the conference and relocate it to a more tolerant location. Only by such actions would these entities truly demonstrate the spirit of cooperation that they were trying in vain to project.

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## NEUROMUSCULAR AND CHROMOSOMAL CO-MORBIDITY IN LV NON-COMPACTION/HYPERTRABECULATION

### To the Editor:

We want to add the following comments to the interesting review by Schwartzberg et al. [1] on left ventricular non-compaction/hyperturbation (LVHT) as an under-diagnosed cause of congestive heart failure. To date there is no evidence that LVHT is a congenital cardiomyopathy in every case. There are a number of reports on adults in whom LVHT appeared or disappeared during their lifetimes [2]. Furthermore, LVHT seems to be neither a disease nor a distinct cardiomyopathy but rather a myocardial abnormality that can be found in normal hearts, dilated and normally sized left ventricles, with and without systolic dysfunction, and with/without severe rhythm abnormalities.

LVHT is most frequently associated with neuromuscular disorders and chromosomal abnormalities and more rarely with hematologic or renal disorders. Today LVHT is associated with at least ten different neuromuscular disorders and at least ten different chromosomal abnormalities [3]. It is still unclear whether LVHT is a distinct cardiac abnormality or represents cardiac involvement in one of the associated disorders.

The pathogenesis of LVHT is unknown. That it is due to an arrest of the myocardial compaction process is only one of several hypotheses. LVHT may

also result from compensatory growth of myocardium in a failing heart or reactivation of embryonic cell growth mechanisms due to an unknown trigger.

LVHT is not only underdiagnosed but also increasingly over-diagnosed, which induces considerable anxiety among patients and their families. Over-diagnosis may occur by echocardiography as well as by magnetic resonance imaging and cardiac computed tomography. LVHT may be mimicked by aberrant bands, false tendons or prominent papillary muscles, especially if the diagnosis is based purely on short axis views, where a differentiation between trabeculations and the above-mentioned structures is not always possible. For a definitive diagnosis of LVHT, not only Jenni's criteria but also our criteria should be applied to avoid an erroneous diagnosis. LVHT is the correct diagnosis only if at least four trabeculations are visible apically to the papillary muscles with intertrabecular spaces perfused from the ventricular cavity [4].

We agree with Schwartzberg et al. that screening first-degree relatives of LVHT patients is necessary. For a correct diagnosis, however, generally accepted diagnostic criteria are required for echocardiography, cardiac CT and cardiac MRI. For risk stratification concerning progression, development of complications and death, close cardiologic follow-up of asymptomatic and symptomatic LVHT patients and reports about the efficacy of therapies are necessary.

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### SWEET SYNDROME AFTER GASTRO-INTESTINAL TRACT INFECTION

#### To the Editor:

**A** 33 year old woman with rheumatoid arthritis and vitiligo presented with an abrupt onset of fever > 38°C and very painful red-to-purple papules, nodules and plaques on the lower extremities that had developed over 5 days [Figure]. On anamnesis, the patient reported having had a gastrointestinal tract infection 2 weeks earlier. Laboratory evaluation showed leukocytosis with an increased absolute neutrophil count (9.9 x 10<sup>9</sup>/L) and high levels of erythrocyte sedimentation rate and reactive-C protein. The rest of the serum analyses, renal profile, X-ray chest, hepatitis study and ultrasound of the abdomen were within normal limits. Skin biopsy revealed a dense perivascular and interstitial inflammatory neutrophilic infiltrate.

The rapid onset of fever and painful erythematous-edematous lesions, peripheral neutrophilia and neutrophilic infiltration of the dermis is known as acute febrile neutrophilic dermatosis or Sweet syndrome. This entity ranges from classic Sweet disease, which usually occurs after an infectious process, to a more aggressive neutrophilic process that may be associated with other inflammatory diseases or malignancy. Classic Sweet syndrome is the most common presentation and accounts for more than 50% of cases. Sweet syndrome associated with a (malignant) neoplasm accounts for approximately 20–25% of the cases.

Diagnosis is based on clinical, laboratory and histological findings. An elevated erythrocyte sedimentation rate and peripheral leukocytosis with neutrophilia are the most consistent laboratory findings in Sweet's syndrome. The classic histopathologic pattern consists

Very painful tender red-to-purple papules and nodules that coalesce to form plaques located on lower extremities.



of a dense, diffuse neutrophilic infiltrate in the dermis. The clinical differential diagnosis of Sweet's syndrome includes infectious and inflammatory disorders (bacterial sepsis, cellulitis, erysipelas, herpes simplex virus infection, leprosy, lymphangitis, panniculitis, pyoderma gangrenosum, sporotrichosis, syphilis, systemic mycosis, thrombophlebitis, tuberculosis, varicella-zoster virus infection, and viral exanthem), neoplastic conditions (chloroma, leukemia cutis, lymphoma, and metastatic tumor), reactive erythemas (erythema multiforme, erythema nodosum, and urticaria), vasculitis (erythema elevatum diutinum, leukocytoclastic vasculitis, and perarteritis nodosa) and other cutaneous conditions [1].

The therapeutic mainstay for Sweet's syndrome is systemic corticosteroids. After initiation of treatment with systemic corticosteroids, there is a prompt response consisting of dramatic amelioration of both the dermatosis-related symptoms and skin lesions [2].

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