

is a subgroup of patients with heregulin-induced activation of normally expressed HER2, it is likely to be so small that we are not currently able to identify it clinically. There is no evidence that specific anti-HER2 therapies will be useful in the absence of conventionally defined HER2 overexpression or amplification.

Mir et al. highlight and provide support for the concern previously identified in the existing package insert for fetal toxicity associated with gestational exposure to trastuzumab. As with most drugs, use during pregnancy should be avoided if possible.

Several people wrote that they were concerned that Figure 1 of my article suggests that the same site on the extracellular domain serves both for dimerization (Panel A) and trastuzumab binding (Panels C through F). In fact, the binding and dimerization sites are distinct (juxtamembranous domain IV for the former and the more distal domain II for the latter), and I regret that this fig-

ure, intended to be generally representational, did not make that distinction clear.^{4,5}

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Taenia solium Neurocysticercosis

TO THE EDITOR: A 73-year-old man, visiting the United States from Nepal, presented to our institution with multiple tonic-clonic seizures. In order to control the seizures, the patient was intubated, admitted to our intensive care unit, and treated with intravenous benzodiazepines and anticonvulsant drugs. Computed tomography (CT) of the head showed a 1-cm enhancing lesion with surrounding vasogenic edema in the left frontal lobe and a focus of increased attenuation within the lesion suggestive of petechial hemorrhage or calcification. Magnetic resonance imaging (MRI) of the brain confirmed these findings and was diagnostic of a neoplastic lesion. A neurosurgical consultation was sought, and on day 5 of the hospitalization, an excisional biopsy was performed. Examination of the biopsy specimen established the diagnosis of neurocysticercosis (Fig. 1). Treatment with albendazole and dexamethasone was started on day 7 of the hospital stay. Fortunately, no recurrent seizures were observed, and the patient was discharged home on day 11.

Neurocysticercosis is the major cause of adult-onset epilepsy in the developing world¹ and is caused by infection of the central nervous system by the larvae of *Taenia solium*. Although humans are the only definitive hosts, cysticercosis develops

when humans become intermediate hosts by ingesting the embryonated eggs of the tapeworm, which release oncospheres that penetrate the intestinal wall, enter the bloodstream, and develop into cysticerci in tissues, with the brain being a high-impact target organ. These eggs may come from the environment (heteroinoculation), may be regurgitated from proglottids into the stomach

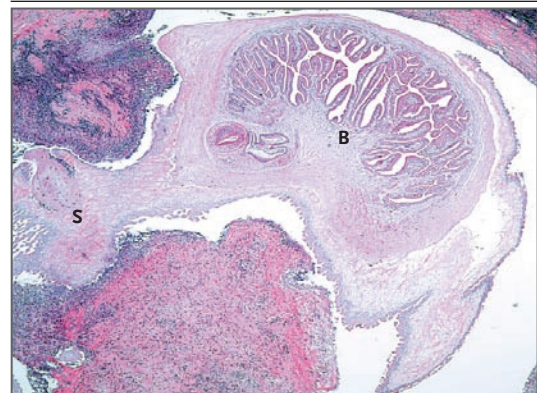


Figure 1. Biopsy Specimen Showing Neurocysticercosis.

Periodic acid–Schiff staining shows inflamed brain parenchyma with gliosis surrounding an inflamed cyst. The cyst contains a *Taenia solium* cysticercus with the scolex (S) and a fluid-filled bladder (B).

(internal autoinoculation), or may be transferred from the fingers of an infected person (external autoinoculation).

The diagnosis of neurocysticercosis is confirmed by means of neuroimaging (CT and MRI) and serologic analysis. The enzyme-linked immunoelectrotransfer blot has a sensitivity and specificity of more than 98% and is the serologic assay of choice for the detection of cysticercosis.² Detection of the parasite in a biopsy specimen of skin nodules may also aid in the diagnosis. Cysticidal treatment is complicated by the fact that it initiates an inflammatory response that may precipitate seizures.³ However, a recent randomized, placebo-controlled trial showed a reduction in the rate of generalized seizures among the patients treated with albendazole.⁴ Corticosteroids are the primary form of therapy for cysticercotic encephalitis, angitis, and arachnoiditis.⁵ Although corticosteroids are recommended in conjunction with anthelmintic treatment to prevent an inflammatory response, the dose, duration, form, and — most important — timing of this treatment still remain controversial.

The diagnosis of neurocysticercosis should be suspected in all visitors and immigrants from Asia, South America, and Central America who have adult-onset epilepsy. In the case of native Americans with this form of epilepsy, asking whether they have a household worker from a country where the infection is endemic may provide a clue.

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STAT3 Mutation in the Original Patient with Job's Syndrome

TO THE EDITOR: In 1966, a report on two unrelated young girls who had had recurrent staphylococcal abscesses since infancy was introduced with the biblical quote, "So went Satan forth from the presence of the Lord, and smote Job with sore boils from the sole of his foot unto his crown" (Job 2:7).¹ The authors called this disorder the Job syndrome because of the phenotypic similarity to the biblical figure Job. Subsequently, similar patients were recognized and noted to have markedly elevated IgE levels, leading to the name "hyper-IgE syndrome" (HIES). (Details about the second girl, who died at the age of 19 years, are provided in the Supplementary Appendix, available with the full text of this letter at www.nejm.org.)

The cause of the syndrome remained elusive until 2006, when a homozygous mutation in the tyrosine kinase 2 (*TYK2*) gene was discovered in a patient with a variant form of HIES, which suggested that defects in signaling between Janus kinase (JAK) and signal transducer and activator of transcription (STAT) may cause the Job syndrome.² We and other investigators^{3,4} have recently identified heterozygous hypomorphic mutations in

the *STAT3* gene in a majority of patients with the classic autosomal dominant form of Job's syndrome.

The protocol for our research was approved by the institutional review board at the University of Washington School of Medicine and Children's Hospital, Seattle. All subjects in the study provided written informed consent.

One of the two original patients with the Job syndrome¹ remains alive in our cohort of patients with classic HIES. This patient (Subject II-3),¹ who is now 50 years of age, has had lifelong eczema, multiple atraumatic fractures, hyperkeratotic fingernails owing to candida infection, recurrent *Staphylococcus aureus* abscesses, and pneumonia with lung abscesses and formation of pneumatoceles. She gave birth to three boys by two fathers (Fig. 1). Her first son (Subject III-1) died 3 days after birth from pneumonia and sepsis with *Pseudomonas aeruginosa* and acinetobacter. Her second son (Subject III-3) was hospitalized shortly after birth with *S. aureus* pneumonia. Later, eczema developed, along with rising IgE levels (104,000 IU per milliliter in his second year of life [normal value, <64]),