Pilomotor seizure
When paroxysmal gooseflesh heralds brain tumor

A 68-year-old man complained of acute and recurrent episodes of diffuse gooseflesh variably accompanied by nonfluent aphasia, emotional distress, and focal myoclonic jerks (video 1 on the Neurology® Web site at www.neurology.org), lasting about 2 minutes and occurring up to every 15 minutes. Contemporaneous EEG recording showed epileptic discharges over left temporal derivations (video 2). MRI showed a left temporal mass extending from the temporal pole to the pulvinar and involving amygdala and hippocampus (figure). Seizures were managed with valproate and levetiracetam. Partial lobectomy was performed demonstrating a grade III anaplastic astrocytoma. Pilomotor seizure1,2 is a rare subtype of autonomic epilepsy related to temporal lesion of which it may be the presenting symptom, usually associated with other manifestations of temporal lobe epilepsy.

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chorea-acanthocytosis (ChAc) that constituted a significant challenge to the generally accepted recessive inheritance of this disorder. This, and other articles concerning the same family, led to a debate in the research community.

Tomiyasu et al. noted that the proband reported by Saiki et al. is indeed compound heterozygous for mutations in the \textit{VPS13A} gene and, therefore, a typical recessive ChAc case. Subsequent to the publication of this report, we found the Correction only via PubMed but missed the original Correction in \textit{Neurology}. In their Correction, Saiki et al. mention with regret that “an error in sequencing occurred and the inheritance pattern should have been reported as autosomal recessive,” with no reference to the recent publication of the full details of the mutations in this family. This Correction effectively changes the impact of the original article and, even more so, its title message. We feel that it would be appropriate to retract the 2003 paper in order to reduce further confusion in the literature.

Alternatively, a more compelling communication from the original authors about the significance of the new data could be offered in a manner readily accessible to the scientific community.

**Author Response: Shinji Saiki, Ishikawa, Japan:**

We regret the error published in our 2003 article. Concerning a pedigree with chorea-acanthocytosis in our previous reports, we would like to correct the genetics materials associated with our pedigree already revised by Tomiyasu et al. In addition to a heterozygous splice site mutation (803G>A, transcript variant A [GenBank NM_033305.2]), which we had reported as 829G>A (using a different accession number) in the \textit{VPS13A} genes of the patients (III-2 and III-3), we identified a novel nonsense mutation (1305G>A, W435X) in the genes of the unaffected mother (II-4) and patients III-2 and III-3. According to them, the inheritance pattern of the pedigree is actually autosomal recessive (pseudodominant).

We apologize for the mistake and deeply regret any inconvenience this publication has caused for others.

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