A Lipemic Sample?
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CASE DESCRIPTION
A 69-year-old male patient with nonischemic cardiomyopathy was admitted for biventricular systolic heart failure. Initial laboratory tests were cancelled due to a high lipemic index of 7 (Beckman UniCel® DxC). A redrawn sample yielded the same flag. The lipemic index did not change following ultracentrifugation in an airfuge. The sample was clear upon visual inspection but precipitated upon mixing with diluent (Sample Diluent 1; Beckman UniCel DxC) (Fig. 1).

Fig. 1. The patient’s plasma (A) and a manual dilution (100 µL plasma:900 µL diluent) of the patient’s plasma with diluent (Sample Diluent 1; Beckman UniCel DxC) (B).

QUESTIONS
1. How is the lipemic index calculated?
2. What are common causes of false lipemia flags?
3. Are results from analysis of this sample likely to be correct?

The answers are below.

ANSWERS
Automated chemistry analyzers determine lipemia on the basis of the absorbance at specific wavelengths following dilution of the sample (1). False-positive results with this method are possible; in particular, the precip-
Ammonium ion inhibition of paraproteins has been shown to correlate with high lipemic indices in clear samples (2). Formation of these precipitates can interfere with the measurement of other analytes, especially those that rely on spectrophotometric detection. This patient had Waldenstrom macroglobulinemia (IgM kappa, 1280 mg/dL).

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References


News & Views

Predictive Testing and Alzheimer Disease
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Alzheimer disease (AD),2 the most common form of dementia, is a progressive degenerative neurological disorder characterized by the accumulation of β-amyloid plaques and τ protein neurofibrillary tangles in brain gray matter. There are currently no therapies to halt or reverse AD progression and no laboratory tests to make a definitive diagnosis. To date, the bulk of AD research has focused on amyloid-β deposition. In response to the largely disappointing results of β-amyloid–targeting therapies in clinical trials, it appears that interest may now be shifting toward apolipoprotein E (ApoE) as described in the recent Nature article, “The Forgetting Gene,” by Laura Spinney (1). With a projected 13.8 million Americans over the age of 65 living with AD by 2050, renewed focus on ApoE as both a therapeutic target and predictive test could have major impacts in delaying symptoms and slowing disease progression (2).

ApoE is not a newcomer to the AD research scene, yet the time and money dedicated to researching its role have been largely overshadowed by β-amyloid. Research from 2004 showed an association between the presence of the APOE (apolipoprotein E)3 allele e4 (ApoE4) and an earlier onset of AD and the extent of AD-related brain pathology (3). Although the exact role of ApoE4 is not known, it is hypothesized that production of ApoE4 by stressed neurons may generate mitochondrial-toxic breakdown products and/or promote β-amyloid deposition. Possible therapies include the development of “corrector” molecules to revert ApoE4 to the less harmful ApoE3 form, and molecular targeting of DNA coregulatory elements of both ApoE and the mitochondrial support protein, Tom40.

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2 Nonstandard abbreviations: AD, Alzheimer disease; ApoE, apolipoprotein E; TOMM40, translocase of outer mitochondrial membrane 40 homolog (yeast).

3 Human genes: APOE, apolipoprotein E; TOMM40, translocase of outer mitochondrial membrane 40 homolog (yeast); BRCA, early-onset breast cancer.