one-half of our CSF samples revealed hypoglycorrhachia.

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References

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Multidrug-Resistant HIV-1

Str—The article by Gandhi et al. [1] about the transmission of multidrug-resistant HIV-1 was very interesting, but they left out the “piece de resistance”—namely, information about what happened to the patient’s viral load and CD4 cell count down the line. They reported only that, after starting to receive 5-drug therapy, his viral load was <50 copies/mL and his CD4 cell count was 203 cells/mm³ after 24 weeks. How long did the virus stay undetected, and if the viral load rebounded, what were the genotypic mutations? We are faced with these situations daily, and it would be nice to know the outcome.

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Reference

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Clinical Infectious Diseases 2004; 38:1507
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Reply

Sir—Dr. Tayloro [1] asks for follow-up data on a patient who acquired multidrug-resistant HIV-1 infection [2]. The patient’s viral isolate had genotypic and phenotypic resistance to multiple nucleoside reverse-transcriptase inhibitors and all available protease inhibitors. Despite exhibiting initially impaired viral fitness, the transmitted viral isolate evolved to develop increased replicative capacity. Within 1 year after infection, the patient’s CD4 cell count decreased to <100 cells/mm³, indicating that the virus retained significant pathogenicity. The patient was treated with tenofovir, lamivudine, didanosine, stavudine, and efavirenz. While receiving this first regimen, his HIV-1 RNA level decreased to <50 copies/mL, and his CD4 cell count increased to 203 cells/mm³.

However, ~7 months after therapy with this combination was started, the patient developed pancreatitis, which was thought to be due to the didanosine. After discontinuation of use of all antiretroviral medications, his pancreatitis resolved. His HIV-1 RNA level increased to 310,000 copies/mL, and his CD4 cell count decreased to 113 cells/mm³ within 3 months of discontinuation of his first drug regimen. Antiretroviral therapy was reintiated with enfuvirtide, tenofovir, stavudine, lamivudine, and efavirenz. He has tolerated this second regimen without difficulty and has not had recurrence of the pancreatitis. After 24 weeks of this regimen, his HIV-1 RNA level decreased to <50 copies/mL, and his CD4 cell count increased to 230 cells/mm³.

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Persistence of HIV Drug Resistance Mutations: More Clues from Clinical Observations

Sir—Gandhi et al. [1] describe the long persistence of HIV drug resistance mutations in a patient infected primarily with drug-resistant HIV and describe subsequent slow, step-wise genotypic drift of the HIV isolate toward wild type during an interruption in therapy, a development in contradistinction to the stated usual re-

Table 2. Summary of CSF glucose measurements for 55 consecutive patients who were positive for herpes simplex virus by PCR.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Range</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSF glucose level, mmol/L</td>
<td>&lt;1–12</td>
<td>3.60 ± 2.00</td>
</tr>
<tr>
<td>Ratio of CSF to serum glucose levels</td>
<td>0.07–0.78</td>
<td>0.50 ± 0.15</td>
</tr>
</tbody>
</table>