tion is the more probable explanation. The addition of a 2-week regimen of gentamicin to a 4-week regimen of penicillin or ceftriaxone is not warranted for the treatment of penicillin-sensitive (minimal inhibitory concentration, <0.12 μg per milliliter) *Strep. gallolyticus* endocarditis.[1]

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No potential conflict of interest relevant to this letter was reported.

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THE DISCUSSANT REPLIES: I suspected that bacterial meningitis developed in the patient from an enteric organism as a complication of the strongyloides hyperinfection syndrome and discussed the tests that I would have used to make the diagnosis in this case. I appreciate the correspondence from Gaffin, because it affords the opportunity to emphasize again the need for clinicians to test for chronic strongyloidiasis before administering immunosuppressive medications to patients who are at risk for this infection. My practice has also been to use serologic testing for strongyloides by means of ELISA to diagnose chronic strongyloidiasis in these patients, for the reasons noted by Gaffin.

I agree with Bush that there is uncertainty regarding whether this patient also had *Strep. bovis* endocarditis as a complication of the strongyloides hyperinfection syndrome. I was not involved in the care of this patient and therefore can only speculate about the rationale behind the treatment decisions made by the infectious-disease consultants in this case. The patient presented to the emergency department with a temperature exceeding 38.0°C, and a transesophageal echocardiogram obtained later in his hospitalization identified an abnormality on the mitral valve that was described as a 2-mm vegetation. Thus, the patient may have had "possible infective endocarditis" according to the modified Duke criteria.[1] The infectious-disease physicians who cared for this patient decided to manage this infection with both penicillin and gentamicin. I agree with Bush that 4 weeks of penicillin or ceftriaxone (or 2 weeks of penicillin and gentamicin) might have been a better choice for the treatment of endocarditis caused by a penicillin-sensitive strain of *Strep. bovis*.

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Since publication of his article, the author reports no further potential conflict of interest.

1. Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis 2000;30:633-8.

DOI: 10.1056/NEJMc1412237

Oral Arsenic and Retinoic Acid for Non–High-Risk Acute Promyelocytic Leukemia

TO THE EDITOR: All-trans retinoic acid (ATRA) and chemotherapy are curative in patients with non–high-risk acute promyelocytic leukemia (APL) (white-cell count <10,000 per cubic millimeter). However, patients can also be cured by treatment with a combination of ATRA and arsenic trioxide without chemotherapy.[1]

The National Comprehensive Cancer Network has adopted ATRA and arsenic trioxide as the first-line treatment for APL in its 2014 guidelines,[2] although arsenic resistance may develop in some patients.[3] Whereas arsenic trioxide must be infused in the hospital, oral arsenic may in some cases be administered outside the hospital. Using a protocol that included chemotherapy in patients with APL, we recently found that oral arsenic (the realgar-indigo naturalis formula [RIF]) provided an outcome similar to that produced with intravenous arsenic trioxide.[4]

From March 2013 through February 2014, we conducted a single-center pilot study to evaluate the efficacy of oral arsenic and ATRA without

chemotherapy in patients with non–high-risk APL. We enrolled 20 consecutive patients and administered oral arsenic RIF (60 mg per kilogram of body weight) and ATRA (25 mg per square meter of body-surface area) as induction therapy until complete hematologic remission. Postremission therapy included RIF on a schedule of 4 weeks on and 4 weeks off and ATRA on a schedule of 2 weeks on and 2 weeks off for 7 months. The primary end point was a complete molecular response, defined as a negative test result for promyelocytic leukemia–retinoic acid receptor alpha transcripts on a quantitative polymerase-chain-reaction assay. Secondary end points included complete remission, numbers of adverse events, length of hospital stay, medical costs, and quality of life as measured with the use of the Functional Assessment of Cancer Therapy — General (FACT-G) questionnaire. Median follow-up was 14 months (range, 8 to 19) by September 2014. All 20 patients had a hematologic complete remission after a median time of 29.5 days.

The rate of complete molecular remission was 65% at 3 months and 100% at 6 months (see Fig. S1 in the Supplementary Appendix, available with the full text of this letter at NEJM.org). At the time of the last follow-up, no patient had had a molecular relapse.

During induction, differentiation syndrome occurred in 2 patients, grade 3 or grade 4 adverse events affecting the liver occurred in 3 patients, and increases in white-cell count to more than 10,000 cells per cubic millimeter occurred in 7 patients. Among the 20 patients in the study, 10 completed the induction therapy on an outpatient basis, without hospitalization. Once complete remission had occurred, all 20 patients received postremission treatment at home and visited the doctor regularly. The median of total medical costs was $4,675 (range, $3,174 to $12,698). Patients resumed their usual lifestyle during postremission therapy, and their quality of life was rated as nearly normal on the FACT-G questionnaire (Table 1, and Table S1 in the Supplementary Appendix).

The results of our pilot study, in which we tested a largely home-based treatment protocol with two oral molecularly targeted drugs and no chemotherapy in 20 patients with non–high-risk APL, showed that the treatment was effective, convenient, and economical. A prospective multicenter, randomized trial comparing oral RIF and ATRA with arsenic trioxide and ATRA is now

Table 1. Characteristics of the Study Patients.*

Characteristic	Patients (N=20)
Before treatment	
Age — yr	
Median	35
Range	20–58
Male sex — no. (%)	10 (50)
White-cell count — per mm³	
Median	1500
Range	500–4400
Platelets — per mm³	
Median	39,500
Range	5000–241,000
Myeloblasts as % of bone marrow	
Median	74
Range	51–92
% of PML-RARA/ABL transcripts	
Median	31.3
Range	5.3–109.6
Type of transcript	
Long	10
Short	8
Variant	2
FLT3 internal tandem duplication mutations — no. (%)	3 (15)
Cytogenetic features — no. (%)	
Solo t(15;17) translocation	11 (55)
Additional abnormal translocation	9 (45)
Fibrinogen — mg/dl	
Median	219.5
Range	63–401
D-dimer — ng/ml	
Median	1172
Range	252–9533
After treatment	
Liver damage — no. (%)	
Grade 1–2	8 (40)
Grade 3–4	3 (15)

Table 1. (Continued.)

Characteristic	Patients (N=20)
Units of platelets infused	
Median	0
Range	0–5
Patients hospitalized — no. (%)	10 (50)
Hospital stay — days	
Median	2
Range	0–32
Time to achieving hematologic CR — days	
Median	29.5
Range	28–40
Time to achieving CMR — mo	
Median	3
Range	2–6
Medical costs — U.S. $	
Median	4,675
Range	3,174–12,698
Score for quality of life†	
Physical well-being	
Median	2.5
Range	0–10
Emotional well-being	
Median	5
Range	4–10
Social–family well-being	
Median	24
Range	22–27
Functional well-being	
Median	27
Range	20–28
Continued CMR — no. (%)	20 (100)

* CR denotes complete remission, and CMR complete molecular remission.

† Scores for quality of life were measured with the use of the Functional Assessment of Cancer Therapy — General. For physical and emotional well-being, a score of 0 indicated the best status and a score of 28 indicated the worst status. For social–family well-being and functional well-being, a score of 0 indicated the worst status and a score of 28 indicated the best status.

under way in China (Chinese Clinical Trial Registry number, ChiCTR-TRC-13004054).

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Disclosure forms provided by the authors are available with the full text of this letter at NEJM.org.

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CORRECTION

Ectopic Fat in Insulin Resistance, Dyslipidemia, and Cardiometabolic Disease (September 18, 2014;371:1131-41). In the legend for Figure 2 (page 1134), the third sentence should have read, "Activated PKCε binds to and inhibits the insulin receptor tyrosine kinase, leading to decreased insulin-stimulated glycogen synthesis in the liver owing to decreased phosphorylation of GSK3," rather than ". . . leading to decreased insulin-stimulated glycogen synthesis in the liver through increased glycogen synthase kinase 3 (GSK3) phosphorylation." The article is correct at NEJM.org.