scopic ultrasound-guided fine-needle aspiration of cystic or solid lesions along the upper gastrointestinal tract or mediastinum show a low prevalence of procedure-related bacteremia.1-4 Aspiration of suspected mediastinal cysts should be undertaken with caution. The American Society for Gastrointestinal Endoscopy recommends the use of antibiotic prophylaxis periprocedurally and postprocedurally (for 3 to 5 days) in all patients undergoing endoscopic ultrasound-guided fineneedle aspiration of mediastinal cysts (grade of recommendation, 1C; intermediate-strength recommendation).5 In a large North American series (which included 22 patients with mediastinal cysts who underwent endoscopic ultrasound-guided fine-needle aspiration) that involved antibiotic prophylaxis, there were no cases of infectious complications related to fine-needle aspiration.²

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Since publication of their article, the authors report no further potential conflict of interest.

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Chronic Lymphocytic Leukemia with SF3B1 Mutation

TO THE EDITOR: Somatic mutations in SF3B1 have been studied as potential predictors of time to initial treatment in chronic lymphocytic leukemia.^{1,2} Wang et al.¹ assessed a cohort of both untreated and treated patients.

We have data that show the influence of SF3B1 mutations on the natural history of chronic lymphocytic leukemia. Our study involved a cohort of 279 untreated patients with chronic lympho-

Table 1. Outcomes in Untreated Patients with Chronic Lymphocytic Leukemia, According to SF3B1 Mutational Status.*			
Variable	Cumulative Incidence		P Value
	Unmutated SF3B1	Mutated SF3B1	
	% (95% CI)		
5-yr progression in patients with Binet stage A disease†	34 (27–42)	73 (51–96)	0.002
10-yr overall survival among all patients	77 (70–84)	30 (6–54)	0.002

* Data are from Quesada et al.³ CI denotes confidence interval.

† The Binet staging system ranges from A to C, with stage A indicating a good prognosis, stage B an intermediate prognosis, and stage C a poor prognosis. cytic leukemia; this allowed us to obtain robust clinical correlates.³ Patients with mutations in *SF3B1* had more aggressive disease than patients without these mutations (Table 1). Cox analyses showed that the mutational status of *SF3B1* had a prognostic value that was independent of clinical stage or expression of ZAP70 or CD38.³ Finally, the fact that all patients with chronic lymphocytic leukemia in our series had not received chemotherapy suggests that rather than being acquired after initiation of treatment, *SF3B1* mutations probably occur during disease development.¹

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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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