remains an open question. The study by Tice et al.,¹ which is based on the findings of the Breast Cancer Surveillance Consortium, showed considerable overlap in the risk estimates for women with atypical hyperplasia and the four density categories. It was only women with atypical hyperplasia and extremely dense breast tissue (12% of the entire group with atypical hyperplasia) whose risk did not overlap completely with the fatty-breast category; this group with high-density tissue had a substantially higher risk of breast cancer.

In response to Reimers et al.: their overall cohort consists of women with various risk features, especially a family history of breast cancer.² In their most recent article on density, all benign breast disease is grouped together.³ In this letter, they refer to an unspecified number of women in a subgroup with atypical hyperplasia and suggest that such women with low breast density may not be at increased risk for breast cancer. They do not state how they handled the effect of age on density and breast-cancer risk.

To follow up on the question posed in both letters, we identified a subgroup of women in our atypical-hyperplasia cohort for whom we had readily attainable density information (Table 1).⁴ Although numbers are limited at this time, we see no significant difference in risk according to breast-density measurement in women with atypical hyperplasia, and thus suggest that this important question merits additional investigation.

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Case 39-2014: A Girl with Crohn’s Disease and Pulmonary Nodules

TO THE EDITOR: Nelson et al. (Dec. 18 issue)² report the case of a 9-year-old girl with familial Crohn’s disease and pulmonary nodules. Chronic granulomatous disease was considered and rejected by El Saleeby. We believe this rejection was based on insufficient data. Chronic granulomatous disease can be ruled out only by the measurement of reactive oxygen species (ROS) production by granulocytes. Testing for ROS production, which was not performed, could have had major clinical consequences in this patient, because Crohn’s disease is clinically and histologically indistinguishable from colitis related to chronic granulomatous disease,²³ and in patients with chronic granulomatous disease, it would be essential to rule out infections with pathogens such as burkholderia, nocardia, granulibacter, and aspergillus before initiating immunosuppressive therapy.

Anti–tumor necrosis factor (TNF) treatment in patients with chronic granulomatous disease is associated with increased mortality.⁴ Therefore, testing ROS production to rule out this disease and evaluating the patient for opportunistic infections are needed before initiating anti-TNF treatment in cases such as the reported one. Finally, the exclusion of obvious mutations in the NADPH-oxidase complex that cause chronic granulomatous disease and NOD2 (indicating Crohn’s disease and Blau’s syndrome) would be equally useful before performing whole-exome sequencing.
To the Editor:

In response to the letter from Hornung et al. (Dec. 25 issue)\(^1\), we would like to point out some concerns that we think are relevant. Recalcitrant dermatomyositis is a challenging condition for clinicians, so the efficacy of a new drug is worthy. Nevertheless, the clinical situation reported by the authors should be recognized as a cancer-associated dermatomyositis (hematologic neoplasm occurring within 3 years after the diagnosis of dermatomyositis). This type of dermatomyositis is characterized by a temporal relationship between myositis and cancer. So in this patient, the improvement that was seen after ruxolitinib therapy could be due to the improvement in the hematologic cancer and subsequently the associated dermatomyositis, rather than to improvement in the dermatomyositis itself. It would be interesting to know whether the authors measured the levels of antibody against transcriptional intermediary factor 1 γ (TIF1γ,}

**More on Remission of Recalcitrant Dermatomyositis Treated with Ruxolitinib**

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