In reply:

We are pleased that the correspondents read our article with great interest, but we are concerned that there appears to be confusion.

The three points and conclusion to the letter are answered as follows.

1. We did not ignore the effect of the increase of acinar size on the myoepithelium, and we stated that the decrease of the density of CK14 staining could be attributable to an increase in the size of the acinar secretory cells, but that the greater decrease in the density of staining of the myofilaments indicated a loss of myofilaments. We mentioned that an increase of parenchymal pressure in other situations is resisted by myoepithelial cells, which become more conspicuous. Thus, there is support for the possibility that the myoepithelial cells in sialadenosis lack the ability to resist an increase of acinar pressure, but not for the suggestion in the letter that the myoepithelium suffers pressure atrophy caused by the increase of size of acinar secretory cells.

2. Ki67 is not a nuclear marker but a marker of proliferation, and we stated that 1,000 nuclei of myoepithelial cells were counted in sialadenosis without finding Ki67 staining.

3. Actin indicates the content of myofilaments, and a decrease indicates a loss of muscular function, which is the relevant function in our article.

Finally, functional insufficiency of myoepithelial cells in sialadenosis was not presented as an assumption by us but as a hypothesis as in the definition of the Oxford English Dictionary Online: “3. A supposition or conjecture put forth to account for known facts; esp. in the sciences, a provisional supposition from which to draw conclusions that shall be in accordance with known facts, and which serves as a starting-point for further investigation by which it may be proved or disproved and the true theory arrived at.”

We hope that our response has removed the correspondents’ confusion.

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Are we ready to blame imatinib for palatal pigmentation?

To the Editor:

I read with great interest the article by Mattsson et al. titled “Oral pigmentation in the hard palate associated with imatinib mesylate therapy: A report of three cases.”

Our research group has been observing oral dark pigmentations in chronic myelogenous leukemia (CML) patients treated with imatinib, such as those cases described by the authors in an oncohematologic setting. The authors were very cautious to affirm that oral pigmentation was secondary to the use of imatinib and only hypothesize this association after other possible causal agents were ruled out. However, although imatinib has become a heavily prescribed drug for CML, most patients worldwide have also been submitted to ≥1 previous cycles of hydroxyurea to control blast crisis. It would be interesting to analyze whether the 2 CML patients described in this study were treated with hydroxyurea, because this drug is recognized as a potential mucocutaneous pigmentation drug. Moreover, chloroquine frequently is prescribed to enhance the sensitivity of CML cells to imatinib. Chloroquine also has been long recognized as a potential inducer of oral and particularly palatal hyperpigmentation. We wonder if these differential diagnoses were totally excluded for the oral dark U-shaped pigmentations identified in the original report.

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REFERENCES


