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Recurrent Cardiac Myxoma: A Puzzle to be Solved

LETTER TO THE EDITOR

To the Editor,

Cardiac myxomas have always been the most interesting type of benign cardiac neoplasms in clinical practice.[1-4] In this context, the interesting nature of these neoplasms is largely attributable to their inherent characteristics such as existing constitutional symptoms, potential risk for local and systemic recurrences, genetic background in certain settings, and strong association with systemic inflammation.[1-7] The recent report by Yalçınkaya Öner and Karakulak[1] has described a didactic case of cardiac myxoma (as part of Carney complex) with its fourth recurrence. We would like to comment on cardiac myxoma recurrences based on this rare case.

First, frequent recurrences in this case may be largely due to its genetic origin associated with the Carney complex (presenting with dermatological and endocrinological abnormalities).[1,3,5] However, persistent systemic inflammation may not only account for constitutional (fever, etc.) and embolic findings in patients with cardiac myxoma but may also play a dominant role in tumor recurrences (local or systemic).[3,5,6] Moreover, the impact of systemic inflammation on clinical recurrences might be even higher in the setting of myxomas with a genetic origin.[3,5] In this context, systemic inflammation may originate from the neoplastic tissue itself or other co-existing inflammatory sources (chronic infections, etc.).[3,5] Mechanistically, certain cytokines including interleukin-6 (IL-6) were previously suggested to have a strong link with cardiac myxoma recurrences.[3,5,6] In particular, persistent elevation of inflammation markers following myxoma resection may indicate 2 relevant implications: (1) a substantial tumoral burden and (2) consequent stimulation of growth factors, both of which may impact tumor recurrences in time.[3,5,6] Therefore, we wonder about the levels of systemic inflammation parameters (previous (if available), and current levels in the pre- and postoperative periods) along with existing constitutional symptoms and systemic inflammatory conditions in the patient (if any).[1]

Second, recurrences in the setting of cardiac myxomas might also arise in the form of systemic recurrences.[3,5,6] This form of recurrences namely "distant tumoral seeding" mostly involve the central nervous system (CNS) (in the forms of cerebrovascular involvement and/or parenchymal lesions).[3,5,6] The patient reportedly suffered previous cerebrovascular events[1] (possibly considered to be of embolic origin). However, these events might also be associated with an insidious and progressive CNS involvement as part of a systemic myxoma recurrence in the patient. Therefore, we wonder about previous or current CNS findings on computed tomography and/or magnetic resonance imaging in the patient (if any).[1]

Third, eradication of persistent systemic inflammation, if any, following myxoma resection might prevent or mitigate further myxoma recurrences.[3,5,6] On the other hand, in the general population, there may be no overt source of persistent systemic inflammation even in apparently healthy subjects.[8] Similarly, the exact origin of systemic inflammation may not be easily identifiable and treatable in certain patients with cardiac myxoma following their tumor resection.[3,5,6]

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Therefore, it may be speculated that off-label use of certain anti-inflammatory agents including cytokine blockers may be of significant benefit for the prevention of future myxoma recurrences in the setting of an idiopathic and persistent systemic inflammation.[3,5,6] Did the authors consider such innovative strategies for their patients?

Finally, the patient reportedly had global left ventricular (LV) dysfunction.[1] This may be due to the chronic impact of acromegaly (due to the elevation of insulin-like growth factor, etc.) on the myocardium.[9] However, severe valvular dysfunction associated with valvular impingement by the mass impact of hypermobile myxomas is also a well-known phenomenon.[3] Therefore, progressive valvular dysfunction (mostly regurgitation) by the previous and/or current myxomas might have also led to "valvular cardiomyopathy" in the patient.[1] Accordingly, we wonder about any valvular dysfunction and history of valvular surgery (during previous myxoma surgeries) in the patient.[1]

Notably, cardiac myxomas were previously suggested to have a particular link with the evolution of takotsubo cardiomyopathy (TTC).[6] Takotsubo cardiomyopathy evolution in this setting might be attributable to cerebrovascular embolism (potentially facilitated by systemic inflammation), CNS involvement by the myxoma (as a manifestation of systemic recurrence), and direct impact of persistent systemic inflammation, all of which appear to be closely interrelated and serve as triggers of severe adrenergic discharge and adrenergic hypersensitivity.[6] Based on his suspicious findings including chest pain, the patient[1] might have also had an atypical pattern of TTC (namely global TTC[10]) that might have accounted for or contributed to the LV systolic dysfunction. Based on the aforementioned notions, TTS evolution might have a strong association with myxoma recurrences.[6] In this context, we also wonder about the levels of cardiac biomarkers (troponins, etc.). Did the systolic dysfunction recover on follow-up?

In conclusion, this didactic case[1] explicitly demonstrates how challenging it might be to combat cardiac myxomas in certain settings. In other terms, these enigmatic neoplasms, despite their benign tissue characteristics, may behave quite aggressively as analogous to certain malignant tumors.[3,5,6] In the setting of recurrent cardiac myxomas, systemic inflammation should also be taken into consideration as a strong determinant of aggressive behavior and hence should be thoroughly investigated and managed in an effort to improve the overall prognosis of these neoplasms.[3,5,6] Finally, there may also exist a significant proclivity for emerging ventricular dysfunction in the setting of recurrent cardiac myxomas possibly due to associated factors including systemic inflammation, coexisting endocrinological abnormalities, and impact of recurrent valvular impingement by the tumoral mass.[1,2,6]

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