Opinion

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Khat consumption induced chronic liver disease: is it all autoimmune?

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Khat (Catha Edulis) consumption is exceedingaly common in East Africa and Southern Arabia and is reported to cause both acute and chronic liver disease (CLD).[1,2] The primary ingredient in Khat is Cathinone[3] which is an alkaloid with amphetamine like properties. Young buds and leaves of khat are chewed to attain a state of euphoria. The mechanism of khat induced hepatotoxicity is not clearly elucidated; however a direct hepatotoxic effect and underlying autoimmune etiology are postulated.[3]

In a recent study by Orlien et al.[2] the authors have performed a case control study and have convincingly demonstrated a significant association between chewing khat and the risk of developing CLD. However, autoimmune markers and a liver biopsy were not performed and chronic hepatitis B (CHB) was noted in around 36.7% of patients who consumed khat in this study. It would thus be debatable to attribute khat as a sole cause of CLD in the absence of a conclusive liver biopsy. Future studies should exclude these patients in order to truly ascertain the impact of khat consumption on liver.

In a study by Riyaz et al.[3] six patients with acute hepatitis were evaluated with a significant history of khat consumption. The authors excluded viral hepatitis in all their patients and noted positive autoimmune markers and elevated serum immunoglobulin G level in around 50 % and 83% of their patients respectively. The authors also performed a liver biopsy in majority of their patients and all these patients had evidence of interface hepatitis on biopsy. In spite of deducting 4 points in the revised criteria for diagnosing autoimmune hepatitis (in view of potential hepatotoxicity of khat), five out of six patients had a pre treatment score of 10 to 15 which placed them in the probable group for autoimmune hepatitis. All the five patients depicted partial response to immunosuppressive therapy, while two attained a complete response. Alhaddad et al.[4] in their report of 2 cases with acute hepatitis due to khat consumption noted interface hepatitis in one and positive autoimmune marker in another patient. In another report by Fallatah et al.[5] the authors noted positive autoimmune profile in all three patients with liver disease who attained remission after instituting immunosuppressive therapy.

This opens up views to two very pertinent questions. Firstly, should autoimmune markers and liver biopsy be routinely done to rule out underlying autoimmune etiology before labelling a patient to have khat induced CLD? This would not only have diagnostic, but also therapeutic consequences as treating patients with underlying autoimmune liver disease could be very rewarding and would potentially impact the natural history of patients liver disease. Secondly, the mechanism of khat induced hepatotoxicity needs to be carefully elucidated and addressed in future studies, as other than a direct hepatotoxic effect, khat might also be responsible to trigger an underlying autoimmunity in genetically susceptible individuals.

I therefore suggest a vigilant workup to rule out any associated autoimmune liver disease in patients presenting with either acute hepatitis or CLD who have had a significant history of khat consumption. Associated chronic viral hepatitis should probably be excluded in future studies in order to unmask the real impact of khat consumption on liver.

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Conflict of interest

The author declares that there is no conflict of interest regarding the publication of this article.

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