

Chronotherapies and leiomyomas

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Abstract

Data provide evidence of a common origin for a subset of physically distinct leiomyoma nodules on patients.

This finding may explain in part the frequent occurrence of multiple synchronous leiomyomas, and is in line with findings in some early reports karyotyping.

Keywords: HMGA2; leiomyomas; Chromothripsis

Introduction

Data provide evidence of a common origin for a subset of physically distinct leiomyoma nodules on patients.

This finding may explain in part the frequent occurrence of multiple synchronous leiomyomas, and is in line with findings in some early reports karyotyping.

Two tumours in one patient, two tumours in a second patient, and five tumours in a third patient were shown to have a common clonal karyotyping.

All nine tumours represented the leiomyoma subclass that overexpresses HMGA2.

We observed CCRs resembling chromothripsis in many of the examined lesions.

Such arrangements are a major cause of chromosomal aberrations in leiomyomas and thus an important generator of tumorigenic changes in these lesions.

Until we know more about the mechanisms underlying CCRs, it is not possible to say with certainty whether the CCRs with low number of breakpoints represent the products of chromothripsis or arise through a different mechanism.

We have therefore used the term “chronotherapies” to describe events resulting in more than 20 intrachromosomal breakpoints.

Chromothripsis events are not rare in leiomyoma precursor cells as the vast majority of events are not expected to produce targeted changes.

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