



Understanding velocardiofacial syndrome: how recent discoveries can help you improve your patient outcomes

Sivakumar Chinnadurai and Steven Goudy

Purpose of review

Improved recognition of velocardiofacial syndrome (VCFS) has led to increasing awareness of VCFS by otolaryngologists. Understanding the developmental biologic processes affected in VCFS patients will help improve treatment and outcomes. Advanced application of molecular labeling techniques has better outlined the role of T-Box transcription factor 1 (TBX1) as the primary genetic anomaly leading to VCFS. TBX1 plays multiple roles during branchial, cardiac, and craniofacial development and increased understanding of how these systems are affected by TBX1 mutations will improve patient outcomes. Furthermore, additional modifiers of TBX1 expression have been identified that may explain the variability of VCFS phenotypes. The phenotypic spectrum of VCFS may include cardiac anomalies, velopharyngeal insufficiency, aberrant calcium metabolism, and immune dysfunction. Recent interest has focused on the cognitive and neuropsychiatric manifestations of VCFS. Improved understanding of the biology of VCFS associated mutations has the potential to improve therapeutic outcomes.

Recent findings

This article will discuss recent developmental biologic understanding of the role of TBX1 and genetic modifiers generating the phenotypic variability seen in VCFS patients. Special attention is given to advances in the realms of immunodeficiency, hypocalcemia, cardiac and arterial patterning anomalies, velopharyngeal insufficiency, as well as cognitive and psychiatric problems.

Summary

Enhanced understanding of the multiple systems affected by TBX1 mutations will result in improved patient outcomes and improved family education. Future research will lead to improved detection of potential targets for gene therapy and change the way physicians counsel families and treat patients.

Keywords

22q11.2, DiGeorge, T-Box transcription factor 1, velocardiofacial, velopharyngeal insufficiency

INTRODUCTION

Since the initial description of syndromic hypoparathyroidism, athymia with immune compromise, and cardiovascular anomalies by DiGeorge in the 1960s and the introduction of the term velocardiofacial syndrome (VCFS) by Robert Sphrintzen in 1978, much has been learned about this syndrome with diverse phenotypic presentation [1,2]. As DNA analysis became more sophisticated, genetic testing revealed that these syndromes, in addition to several others, share a common genetic basis: a deletion on the short arm of chromosome 22 [3]. Multiple eponymous syndromes are now commonly united under the names 22q11.2 deletion syndrome or VCFS. This article will focus on understanding the phenotypic variation of patients with VCFS and how the

multiple systems affected from a developmental perspective are relevant to otolaryngologists.

EMBRYOLOGY

Branchial arch formation begins very early in human development during the 3rd and 4th week of gestation. The paired six arches are composed of

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The Official Patients Sourcebook On Velocardiofacial Syndrome

James Chambers



The Official Patients Sourcebook On Velocardiofacial Syndrome:

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