A Case of Seborrhoeic Keratosis and Family Survey

F Xie, R Wang, Y Chen, Y Jia, X Jia, M Chen, L Yang, X Lan

ABSTRACT

This study is a family survey based on a typical patient with seborrhoeic keratosis (SK). Family members comprised 47 people of four generations, including 22 affected members (12 males and 10 females). The 17 people of the fourth generation were aged less than 26 years old. The proband in this study had typical clinical manifestations and pathology of SK, and all affected members of the family had characteristic skin rash that was easy to be identified. The pattern is consistent with autosomal dominant inheritance and SK is indicated to have a certain age onset.

Keywords: Family, heredity, seborrhoeic keratosis

Un Caso de Queratosis Seborreica y Encuesta Familiar

F Xie, R Wang, Y Chen, Y Jia, X Jia, M Chen, L Yang, X Lan

RESUMEN

Este estudio es una encuesta familiar basada en un paciente típico con queratosis seborreica (QS). Los miembros de la familia comprendían 47 personas de cuatro generaciones, incluyendo a 22 miembros afectados (12 varones y 10 hembras). Las 17 personas de la cuarta generación tenían menos de 26 años de edad. El probando (caso índice) en este estudio tenía manifestaciones clínicas típicas y patología de QS, y todos los miembros afectados de la familia presentaban la erupción de la piel característica, fácil de identificar. El patrón concuerda con la herencia autosómica dominante, y se indica que QS tiene su inicio a una cierta edad.

Palabras claves: Familia, herencia, queratosis seborreica

West Indian Med J 2016; 65 (1): 219

INTRODUCTION

Seborrhoeic keratosis (SK) is also known as senile wart or basal cell papilloma, and is a benign skin tumour because of the slow maturation of keratinocytes. It is thought that this disease is a kind of epithelial nevus, skin benign tumour or senile skin changes. The aetiology of this disease is unknown, and some universal lesions are shown to have an autosomal dominant inheritance predisposition (1), but the exact pathogenesis remains unclear. Due to lack of unified manifestations, this disease can easily be ignored. To date, there is rarely a complete report on familial SK. In this study, the survey and analysis were conducted on familial SK with complete clinical manifestations. The objective is to further explore the genetics of SK.

From: Department of Dermatology, 303rd Hospital of People's Liberation Army at China, Nanning 530021, Guangxi Province, China.

Correspondence: Dr F Xie, Department of Dermatology, 303rd Hospital of PLA, Nanning 530021, Guangxi Province, China. Fax: +86-771-5305543; e-mail: fangmingxiecn@163.com

CLINICAL DATA

The proband, a 48-year old male (Fig. 1), attended the clinic with brown papules and plaques of the face and chest for more than 20 years. The brownish papules and plaques in the chest were slowly increasing in size and number, thickening, becoming darker in colour, were not accompanied by itching and there were no symptoms. This study was conducted in accordance with the Declaration of Helsinki and with approval from the Ethics Committee of 303rd Hospital of People's Liberation Army (PLA). Written informed consent was obtained from the participants.

The patient had no particular previous medical history, and denied consanguineous marriage. In the physical examination, nothing special was discovered and no systemic superficial lymph node was felt. The dermatologists described multiple brown or light brown patches of different sizes on the face and chest; part of the rashes coalesced, with clear edge and rough surface, slightly bulging in the skin. Skin biopsy showed hyperkeratosis; papillomatous hyperplasia of the epidermis was mainly of basal-like cells, and the base of the tumour was at the same level of both sides of the normal epidermis (Fig. 2).

DOI: 10.7727/wimj.2014.221

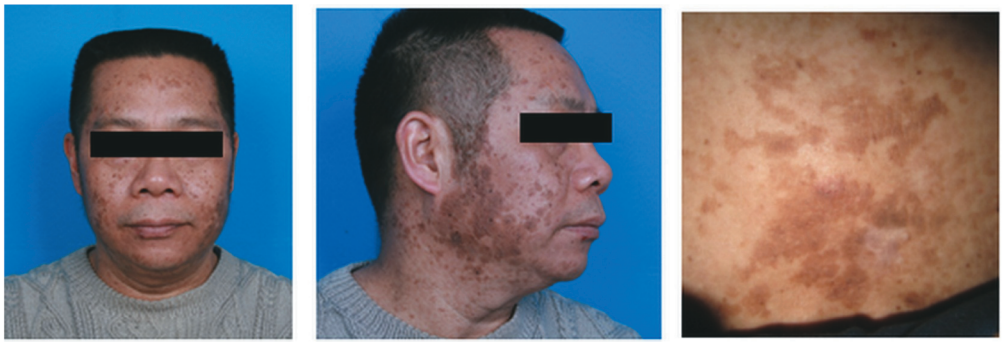

Fig. 1: The face and chest lesions of the proband.

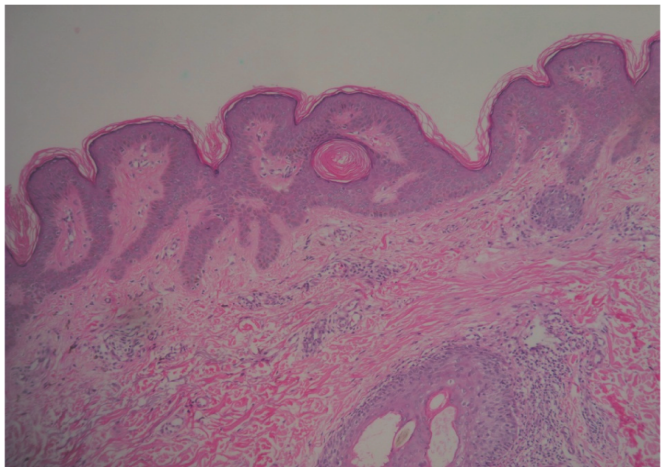

Fig. 2: The histopathology of seborrhoeic keratosis.

RESULTS

The family had a total of 47 members over four generations, with no consanguineous mating history. Twenty-two members, including the proband, were affected with the condition, but with different severity. The fourth generation so far had no such affected members, which may be related to their age (Fig. 3).

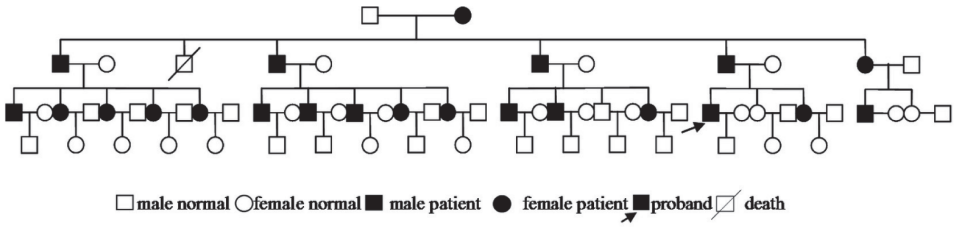

Fig. 3: The family tree of the seborrhoeic keratosis patient.

Observing the family tree profiles, it was found that the whole family had the following features: the first three generations had such affected members, there was no gender difference, there was one affected parent and there was continuous passage, which is consistent with the autosomal dominant inheritance.

DISCUSSION

Many domestic and foreign reports show that SK has a familial and genetic tendency, and its mode of inheritance is autosomal dominant (2, 3). Few studies have found that familial SK might be related to mutations of FGFR3 and PIK3CA in body cells (1, 4−6). In the members of this SK family, the first three generations have SK. The male and female prevalence rates are equivalent, with no gender difference. There is one affected parent of each person. The family heredity features indicate that SK is an autosomal dominant genetic disease. In addition, there is no onset in the fourth generation. The maximum age of the fourth generation is 26 years old and the onset age of the proband is about 26 years old. It is reported that in subjects aged 15−25 years old, the incidence of SK is only 12%. The incidence increases with age (1, 7). Therefore, the onset of SK is closely associated with age. Due to autosomal dominant heredity, SK will appear in the future of the fourth generation. The tracing and observation of the SK family, especially the fourth generation, can contribute to further confirming the genetic mechanism of this disease and locating the pathogenic chromosomes.

Seborrhoeic keratosis, also known as senile wart or basal cell papilloma is the most common benign tumour of epidermal hyperplasia of the elderly, it is often located on the face, back of hand, chest, back, *etc*. In the beginning, it is one or a plurality of light yellow or light brown flat papules, round, oval or irregular in shape, with a clear boundary and rough surface,

later gradually increasing in size, thickness and in numbers. Part of the lesions may connect, often with greasy scales on the surface; there are generally no symptoms. Seborrhoeic keratosis should be differentiated from pigmented nevus, melanoma, actinic keratosis and pigmented basal cell carcinoma. This disease generally does not need treatment. For cosmetic reasons, liquid nitrogen freezing (cryotherapy), laser treatment or surgical excision can be adopted.

In conclusion, the proband in this study has typical clinical manifestations and pathology of SK, and all affected members of the SK family have characteristic skin lesions that are easy to be identified. The number of members of this SK family is large. Therefore, this study has further research value.

CONFLICTS OF INTERST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

1. Hafner C, Hartmann A, van Oers JM, Stoehr R, Zwarthoff EC, Hofstaedter F et al. FGFR3 mutations in seborrheic keratoses are already present in flat lesions and associated with age and localization. Mod Pathol 2007; **20:** 895−903.
2. Hafner C, Toll A, Fernández-Casado A, Earl J, Marqués M, Acquadro F et al. Multiple oncogenic mutations and clonal relationship in spatially distinct benign human epidermal tumors. Proc Natl Acad Sci U S A 2010; **107:** 20780−5.
3. Rongioletti F, Corbella L, Rebora A. Multiple familial seborrheic keratoses. Dermatologica 1988; **176:** 43−5.
4. Hafner C, van Oers JM, Hartmann A, Landthaler M, Stoehr R, Blaszyk H et al. High frequency of FGFR3 mutations in adenoid seborrheic keratoses. J Invest Dermatol 2006; **126:** 2404−7.
5. Hafner C, Hartmann A, Vogt T. FGFR3 Mutations in epidermal nevi and seborrheic keratoses: lessons from urothelium and skin. J Invest Dermatol 2007; **127:** 1572−3.
6. Hafner C, Vogt T, Landthaler M, Müsebeck J. Somatic FGFR3 and PIK3CA mutations are present in familial seborrhoeic keratoses. Br J Dermatol 2008; **159:** 214−7.
7. Yeatman JM, Kilkenny M, Marks R. The prevalence of seborrheic keratoses in an Australian population: does exposure to sunlight play a part in their frequency? J Br Dermatol 1997; **137:** 411−4.