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Research Article

Analysis of Arpc1b Gene Expression in Pterygium

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Abstract

Pterygium, or flesh growth in the eye, is a common eye illness that causes the conjunctiva on the cornea to expand out of control. The most common cause of epithelial tissue abnormalities is long-term UV radiation exposure from the sun. Although the precise etiology of the pterygium disease is unknown, additional elements such as dry weather, dust, and a vaporization of the tear film, and other genetic factors, Despite the fact that the exact cause of pterygium illness is uncertain, there seems to be a correlation between outdoor work and the illness's development. Subunit 1B of the Actin Related Protein 2/3 Complex the name of the ARPC1B gene. The human Arp2/3 protein complex has seven subunits, and this gene codes for one of them. Sensory rhodopsin-2 protein is a component that belongs to the SOP2 family. The goal of this research was to see how the ARPC1B gene affected pterygium illness. Tissue was extracted during surgery with the subjects' agreement and consent. We employed the technique expression of ARPC1B gene by extracting cDNA synthesis from group of tissues levels assessed by Real-Time PCR, and the diagnosis of pterygium was validated (polymerase chain reaction). According to the findings of qRT-PCR, there was no discernible change. in ARPC1B gene levels of expression in pterygium and normal conjunctival tissues ($p>0.005$).

Keywords: ARPC1B, Pterygium, qRT-PCR

1.Introduction

Pterygium is frequent eye diseases (Carlos et al., 2018). The growth of fibrovascular tissue along the surface of the cornea distinguishes it. The pathophysiology of the pterygium disease is complex and still being researched (Mologen et al., 2017) The most prevalent cause is long-term exposure to UV radiation from the sun (Farhad et al., 2018). Other variables, including as dry weather, dust, and other hereditary variables, also has a function in it (Kyei et al., 2016; Carlos et al., 2018). Pterygium is usually asymptomatic although mixed humidity beneath the ocular surface may induce dry eye symptoms such as burning, itching, and lacrimation. Advanced pterygium inside the optical zone reduces sharp-sightedness and requires surgery. (Mologen et al., 2017). Although the actual origin of pterygium sickness is unknown, there seems to be a relation between the sickness and outdoor employment (Desheng et al., 2013). Until now, there hasn't been a national, population-based study of pterygium occurrence throughout the world, and it seems that a national, pooled estimate based on the worldwide population would be valuable. Actin Related Protein 2/3 Complex Subunit 1B is encoded by the ARPC1B gene. This gene codes for one of the seven subunits of the human Arp2/3 protein complex. Sensory rhodopsin-2 protein, a member of the SOP2 family, is the name of this component. Because these two proteins are so similar, they may both function as the p41 component of the human Arp2/3 complex, which has been related to cell actin polymerization control. (RefSeq, 2011). It's probable that the p41 subunit aids in the construction and upkeep of the Arp2/3 complex structure. Various variations of the p41 subunit may match the complex's activities to different types of cells. Because it is a substrate and activator of Aurora kinase A this protein has a function in centrosomal homeostasis (RefSeq, 2011). Platelet abnormalities with Eosinophilia, Immune-Mediated Disease, and Combined lymph cell and B cell Immunodeficiency are some of the diseases linked to the ARPC1B gene. (RefSeq, 2011). One of the major causes resulting in pterygium development is thought to be cell cycle disruption, indeed abnormal cell cycle kinetics can cause cellular proliferation and contribute to apoptosis escape. Patients with mutations in the ARPC1B component of Arp2/3 show signs of immunodeficiency and immunological dysregulation, including recurrent viral infections and a low CD8+ T cell count. We show here that a lack of ARPC1B results in a loss of CTL cytotoxicity, with the defect happening on two levels. Across immunological synapse infections, ARPC1B is necessary for the development of lamellipodia, cell movement, and actin remodeling. Second, ARPC1B is necessary for the preservation of TCR, CD8, and GLUT1 membrane proteins in CTL plasma membranes, since recycling via the retromer and WASH complexes is hampered in the absence of ARPC1B. (RefSeq, 2011). The retromer complex identifies cargo proteins that are localized in specific areas of the endosomal membrane, where tubules transport cargo proteins to the right destination, and the WASH complex is a 500 kDa complex that comprises WASH1, FAM21, and SWIP and other proteins (Derivery et al., 2009). ARPC1B have a role in the formation and The ARP2/3 complex, which is involved in actin branching from a filament, is maintained. They speculated that ARPC1B loss in T cells might result in cytoskeleton and functional abnormalities. Six patients with associated early-onset sickness, severe infections, autoimmune symptoms, and thrombocytopenia had biallelic mutation ARPC1B which is identified by RefSeq, Immunological characteristics included T-cell lymphopenia, a low number of T cells, and hyper-immunoglobulin E. (RefSeq, 2011). Actin Related Protein 2/3 Complex Subunit 1B have a role in platelet dysfunction, in which a large number of platelets causes hard tissue sclerosis in the eye, and we know that pterygium disease is

the formation of triangular tissue on the cornea. The goal of this study is to look at the influence of the ARPC1B gene on pterygium disease. According to the literature there is no study has been done on the ARPC1B gene and the formation of pterygium tissue, the work that will be done is groundbreaking in Turkey and throughout the globe. Pterygium is a noncancerous conjunctival tumor that is benign. localized in front of the sclera. In certain situations, it may cause vision impairment and become inflamed, resulting in redness and agitation in the affected region. Additionally, certain eye malignancies, such as ocular surface squamous neoplasia, may develop from pterygium (Ting et al., 2013). The name "pterygium" derives from the Latin form of a-Pterygion"-"pterygos" and meaning "wing" (Hovanesian, 2012). It is separated into three distinct parts: the apex, the neck, and the body. The body of the pterygium is an elevated triangular component with its base toward the canthus, the neck contains the head invades the cornea, and the limbus is superficial. and forms the apex of the triangle (Chinyelu, 2019). Pterygium is a degenerative disorder caused by benign conjunctival development that is characterized by fibro vascularization, conjunctival invasion, and collagen elastic degradation. However, since active cell growth occurs with low apoptosis, several investigations have shown that pterygium has several characteristics with malignancies. Pterygium causes vision problems by blocking the visual axis and causing considerable astigmatism (Helen et al., 2019). Pterygium is a highly common fibrovascular disease in people who live close to the equator, often referred to as the "pterygium zone" (Ting et al., 2013). At these tropical locales, prevalence might be as high as 22%, with fewer than 2% in latitudes above 40°. (Algahtani., 2013; Gingereke et al., 2018). Pterygium mostly affects persons who are exposed to more sunlight; it has been proven that males are twice as likely as women to get the condition, which might be due to the fact that males in certain nations spend more time outdoors than women. (Ting et al., 2013). Human papillomavirus infection, which is frequent in some populations, is another key environmental component. (Di Girolamo, 2012). Familial occurrence of pterygium, on the other hand, suggests that genetic factors have a function in pterygium pathogenesis (Ting et al., 2013). Even with this knowledge, the cause of pterygium illness is unknown. In recent years, investigations using molecular genetic research methods on patient tissue samples have revealed vital new insights on disease pathogeneses. It confirmed the effect of UV radiation it also added viral infections, epigenetic disorders, immunological and antiapoptotic Angiogenic and lymphangiogenic processes stimuli, growth factors, extracellular matrix remodeling, Extracellular matrix modulators are dysregulated. and epithelial changes within the mesenchymal transition. A number of these variables have also been linked to UV light exposure, either directly or indirectly. (Cárdenas-Cantú (Perra et al., 2006; Cárdenas-Cantú et al., 2016).

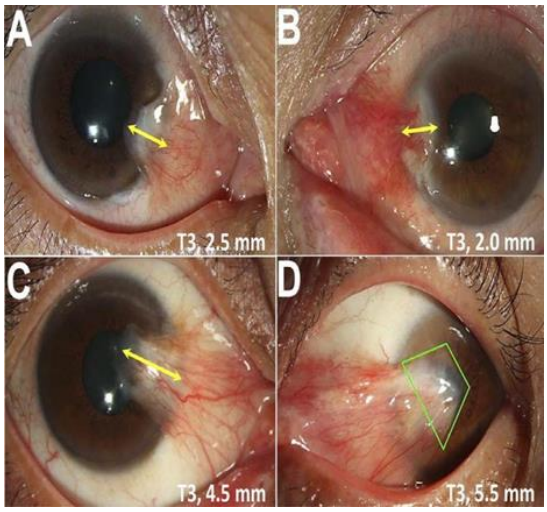

Figure 1. Chu et al;2016 primary pterygium of grade T3

According to a big North American research, pterygium disease is 2.5 to three times more common in black people than in white people which is approximately twice as prevalent among people who work outdoors but just one fifth (0.2) as prevalent among those who wear sunglasses all the time outdoors. (Chimdia, 2019). As previously said (UV) light exposure might not be the sole factor connected with the evolution of pterygium. Dust and sand may participate to the happening of pterygium. In recent years, raised Transcript factor levels, cAMP response element binding protein, cytochrome P450 1A1 protein, phospholipase D, and aquaporin-1 and -3 have all been identified as risk factors. (Zhou et al., 2016). UV radiation (UVA and UVB) has been linked to a variety of biological consequences, including DNA damage, oxidative stress, activation of cell surface receptors, and activation of intracellular signaling pathways (Zhou et al., 2016). UV-C is antibacterial and may induce skin cancer (Gingereke et al., 2018). P53 is overexpressed and may be shown immunohistochemically (Yangwuyue et al., 2013). The main genetic joint marker of human neoplastic development is p53 mutations. Instead of degenerative dysfunction, pterygium is classified as a neoplastic- like growth condition (Chui et al., 2011; Ting et al., 2013).

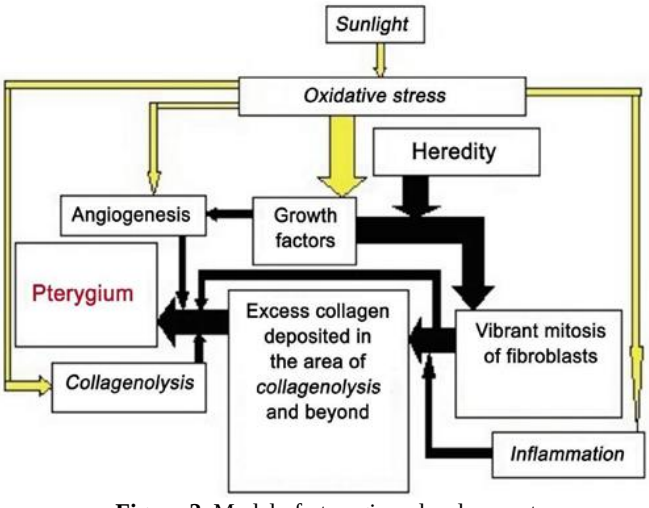

Figure 2. Model of pterygium development

Table 1. Cárdenas-Cantú et al., 2016 The function of overexpressed inflammatory mediators in the pathophysiology of pterygium

Mediator of inflammation	Potential pathogenic role
IL-1	Other mediators and extracellular matrix modulators are upregulated.
IL-6	Angiogenesis, cell differentiation, tissue invasion, and inflammation are all aided by this substance.
IL-8	Angiogenesis, cell differentiation, tissue invasion, and inflammation are all aided by this compound.
TNFa	Angiogenesis, cell differentiation, tissue invasion, and additional inflammation are aided by this substance.
COX2	Further inflammatory cascades
Phospholipase D	Further inflammation and cellular differentiation
Cystatin C	Regulation of extracellular matrix modulators

2-General Information

2.1-ARPCIB

The ARPC1B gene is a Protein Coding gene that codes for Actin Related Protein 2/3 Complex Subunit 1B. human Arp2/3 protein has seven subunits, and this gene codes for one of them. The SOP2 family of subunits includes sensory rhodopsin-2protein. (RefSeq, 2011). The similarity of these 2 proteins implies that they may both play a role as the p41 component of the human Arp2/3 complex, which have been linked to cell actin polymerization regulation. It's possible that the p41 subunit has a role in constructing and maintaining the Arp2/3 complex's structure. Various variations of the p41 subunit may match the complex's activities to several kinds of cells. This protein also has s role in centrosomal homeostasis by acting as an Aurora activator and substrate. Diseases linked to ARPC1B consisting of Platelet Abnormalities with Eosinophilia, Immune-Mediated Inflammatory Disease, and Combined T Cell and B cell Immunodeficiency (RefSeq, 2011). Patients with mutations in the ARPC1B component of Arp2/3 have lack of immunity and immunological dysregulation signs and symptoms, such as viral infections that reoccur and a down CD8+ lymphocyte count. ARPC1B is necessary for lamellipodia generation, cell movement, and actin remodeling throughout the immune synapse infections, as shown by loss of Actin Related Protein 2/3 Complex Subunit 1B. resulting to loss of CTL cytotoxicity, with the deficiency originating at two separate levels (RefSeq, 2011).

2.1.1-ARPC1B gene and structure

Actin Related Protein 2/3 Complex Subunit is the name of the gene ARPC1B. this gene is localized on the 7th chromosome (7q22.1) and consists of 14 exons, and it has 27127 bp (base pair).

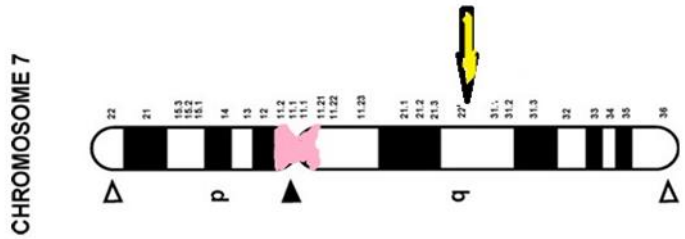

Figure 3. ARPC1B gene localization on the 7th chromosome (7q22.1)

The Arp2/3 complex is made up of seven subunits: Arp2, Arp3, Arc-p16, Arc- p20, Arc-p21, Arc-p34, and Arc-p41. (Tyler J et al., 2016). Arc-p41, also known as ARPC1B, is one of these subunits that is thought to have a regulatory function in the complex's formation and maintenance. As indicated by the broad immunological abnormalities identified when this control is disrupted, the exact control of actincytoskeleton dynamics is crucial to just about each stage of the immune system's reaction (Swaney and Li, 2016). As a result, it's not unexpected that the molecules that control this crucial activity are connected to immunodeficiency's pathogenesis. A different mutation in this gene was lately discovered in a patient with platelet, neutrophil abnormalities (Kuijpers et al., 2017). ARP2, ARP3, ARPC2, ARPC3, and ARPC4, as well as one molecule each of the ARPC1A and ARPC1B, and ARPC5A and ARPC5B isoform pairs, make up the mammalian Arp2/3 complex. On the human 7th chromosome, ARPC1A and ARPC1B are found together (Laurila et al., 2009). The isoforms they express share 68 percent of their both the amino acid sequence and feature 6 WD40 domain repeats expected to shape a b-propeller fold (UniProt, 2015). (Marko, 2017). ARPC1A has been connected to Oral cancer (Laurila et al., 2009) and has been identified in pancreatic cancer as a regulator of cell migration and invasion (ductal carcinoma) (Laurila et al., 2009). (Auzair et al., 2016). ARPC1B gene has been discovered as a centrosomal protein taken part in in mitosis, independent of its activity in Arp2/3 (Molli et al., 2010; Julia, 2017). Arp2/3 deficiency causes embryonic death, whereas its suppression inside cells prevents lamellipodia production and migration (Jie, 2017). (Wu et al., 2012). There has yet to be documented an inherited human ARPC1B deficiency.

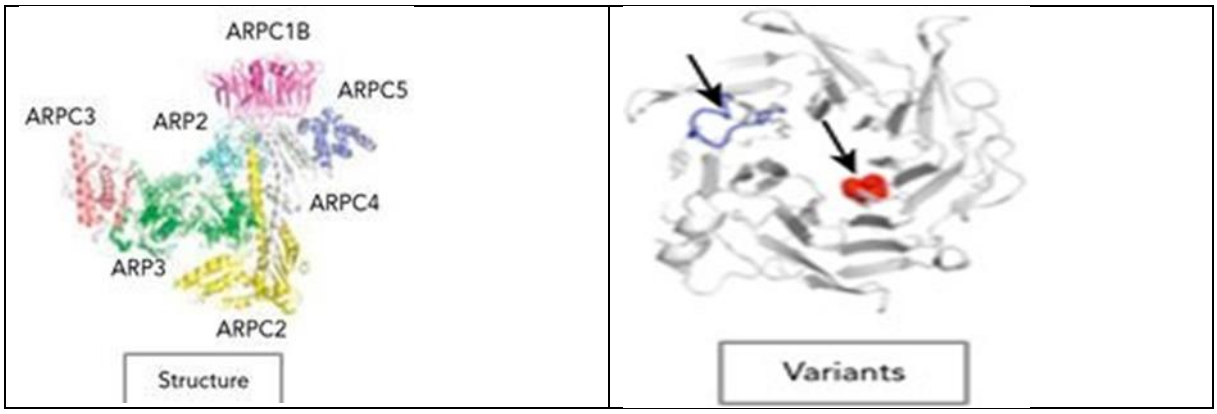

Figure 4. Gene structure and Variants

2.1.2-The relationship between ARP 2/3 complex and WASP gene

WASP is known as The Wiskott–Aldrich syndrome protein controls actin polymerization by activating the Arp2/3 complex, which allows new actin filaments to be formed and cross-linked from end to side branch. (Pizarro-Cerda, 2017). WASP, which causes Wiskott–Aldrich syndrome was the first actin-related protein to be identified and researched as a cause primary of immunodeficiency (Somech et al., 2017). Microthrombocytopenia, eczema, coupled T and B cell immunodeficiency, and a higher prevalence of autoimmune symptoms and cancers characterize WAS, an X-linked immunodeficiency illness with a a unique clinical appearance. Mutations in the Arp2/3 complex or its activators have yet to be recognized as a cause of immunodeficiency syndromes with WAS-like disorders, owing to the fact that many of these genes are required for healthy development and their loss of function would result in premature death. (Moulding et al., 2013). A different mutation in this gene was lately discovered in a patient with platelet and neutrophil abnormalities (Kuijpers et al., 2017). WASP is one of several nucleation-promoting factors that induce F-actin branching through the actin-related protein 2/3 complex (Arp2/3), which is involved in the process. It is required for cell migration, in ., addition to cytokinesis (Rotty et al., 2013). Activated WASP binds Arp2/3 in a 2:1 stoichiometry by engaging with the ARP2, ARP3, and ARPC1 subunits, inducing conformational changes that improve actin monomer binding and daughter filament production (Boczkowska et al., 2014).

3-Materials and Methods

3.1-Equipment

3.1.1-Working groups

The research was carried out at Tokat Gaziosmanpaşa University's Faculty of Medicine's Eye Diseases Department. There were 27 cases diagnosed with pterygium (18 men, 9 women). They were taken during the procedure in the construction of working groups, with pterygium tissues as the patient group and healthy conjunctival tissues belonging to the same eye of the same patient as the control group. Tokat Gaziosmanpaşa University Faculty of Medicine's Clinical Research Ethics Committee With the project at the Tokat Gaziosmanpaşa University Scientific Research Projects Directorate meeting dated By19-KAEK-024 Project number 2019/110 is being backed up.

3.1.2-Chemical materials used, kits and kit contents

cDNA synthesis kit (GeneAll, Hyperscript, First strand synthesis kit Catalog # 601-005, Korea), Reverse Transcriptase Enzyme 10X RTase Reaction Solution, 0.1 M DTT, 10 mM dNTP mix RNase Blocker Oligo (dT) 20, Random hexamer (primary) Nuclease-free water, Ethanol (Sigma-Aldrich Catalog No: E7023, USA), Qubit ssDNA Assay Kit (Invitrogen Catalog No: Q10212, USA) Qubit RNA HS-Assay Kit (Invitrogen Catalog No: Q32852, USA) RNA isolation kit (Thermo, Catalog No: 12183018A, USA)

Lysis Solution Washing Solution 1

Washing Solution 2 RNase-free Water

RNaseZap (Thermo Catalog No: AM9780, USA) Running Solution (ThermoFisher, NP0002, USA).

3.2. Method

3.2.1. Protocol for RNA isolation

1- Homogenize pterygium tissue samples in 0.75 ml RiboEx^{TM} LS per 50 ~ 100 mg of tissue using homogenizer.

2- Lyse 0.25 ml the sample in 0.75 ml RiboEx^{TM} LS by pipetting or vortexing.

3- Incubate the homogenate for 5 minutes at room temperature.

4-Centrifuge at 12,000 x g for 10 minutes at 4°C and transfer the supernatant to a fresh tube.

5-Add 0.2 ml of chloroform per 0.75 ml of RiboExTM LS. Shake vigorously for 15 seconds, store for 2 minutes at room temperature.

6-Centrifuge at 12,000 x g for 15 minutes at

4°C, then transfer the aqueous phase to a fresh tube.
7-Incubate samples for 10 minutes at room temperature.
8-Centrifuge at 12,000 x g for 10 minutes at 4°C, and discard the supernatant.
9-Add 1ml of 75% ethanol to wash the RNA pellet. The RNA precipitate can be stored in 75% ethanol at 4°C .
10- Centrifuge at 7,500 x g for 5 minutes at 4°C. Carefully discard the supernatant, ethanol, and air-dry the RNA pellet for 5 minutes.
11- Dissolve RNA in DEPC-treated water or in 0.5% SDS solution by incubating for 10 ~ 15 minutes at 56°C.

3.3-cDNA Synthesis Protocol

To obtain optimum performance with cDNA Synthesis Kit, make sure that RNA samples is not contain PCR inhibitor, reverse transcription inhibitor, RNase activity and genomic DNA. Mix the kit components in a micro tube in the recommended proportions below. Briefly centrifuge the tube to reduce the volume of the contents and remove any air bubbles Place the tube in the freezer until the reverse transcriptase process is complete.

Table 2. Reverse transcriptase reaction volume (20 µl)

For 20 µl Reverse Transcriptase Reaction	**Volume**
10X Reaction Buffer	2 µl
dNTP mix (2.5 mM each)	1 µl
Random hexamer (50 µM)	2 µl
Reverse Transcriptase (200 U/µl)	1 µl
RNase Inhibitor	0.5 µl
RNase free Water	3.5 µl
RNA Template	10 µl

Table 3. PCR program applied for cDNA synthesis

RT Steps	**Temp (°C)**	**Time**	**Cycle**
Step 1	25	10 min.	1
Step 2	37	120 min.	1
Step 3	85	5 min.	1
Step 4	4	∞	1

The obtained cDNA samples were frozen at -80 ° C until the Real-Time PCR step.

3.3.1-qRT-PCR step

Real time for expression analysis after cDNA extraction from isolated RNAs**.** At this stage, control and study for the target ARPC1B gene, Beta-actin was used . as the reaction mixtureA.B.T. ™ 2X qPZR SYBR-Green MasterMix (with ROX) (Cat no: Q03- 02-01) used. The reaction mixture was prepared as follows:

Table 4. PCR reaction components (20 µl) The reaction cycle was set up on the Applied Biosystems Real Time PCR instrument as follows

A.B.T.™ 2X qPCR SYBRGreen Master Mix (with ROX)	10 µl
1X ROX Dye (50X)	0.4 µl
Forward Primer	1 µl
Reverse Primer	1 µl
Template cDNA	3 µl
Rnase-free Water	4.6 µl

QRT-PCR program implemented
50 ° C 20 sec
95 ° C 10 min
95 ° C
15 sec ←
60 ° C 1 min Melt Curve: 95 ° C 30 sec, 60

42 cycles

4.Results

4.1-Results of working group

This research was conducted at the Tokat Gaziosmanpaşa University Faculty of Medicine Medical Biology and Genetics labs and included 27 individuals with pterygium illness.The study group consisted of 18 males and 9 females, age range 43-78 (mean 58
± 8.43).

4.2-Quantitative Real-Time PCR (QRT-PCR) Analysis Results

QRT-PCR image and evaluation of the ARPC1B gene

SYBR Green based quantitative Real-Time PCR (Polymerase Chain Reaction) method was used to determine the expression level of ARPC1B mRNA expression. For ARPC1B and the gene preferred as a reference (Actin: ACTB), samples of cDNA (1/10: 1/100: 1/1000 and 1/10000) at different concentrations were prepared and serial dilution was made.
In the figures below, the amplification curve and melting peak of ACTB and ARPC1B genes observed as a result of qRT-PCR are given, respectively (Figure 5,6,7)

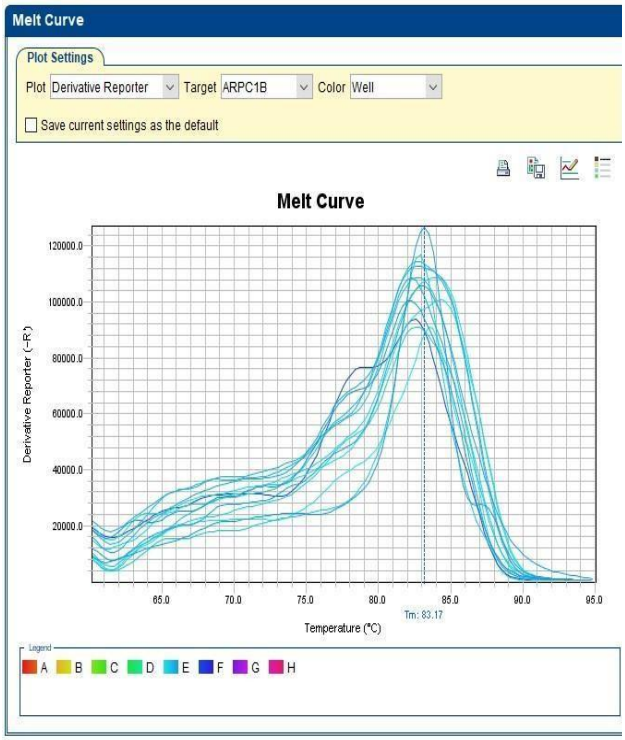

Figure 5. ARPC1B amplification plot

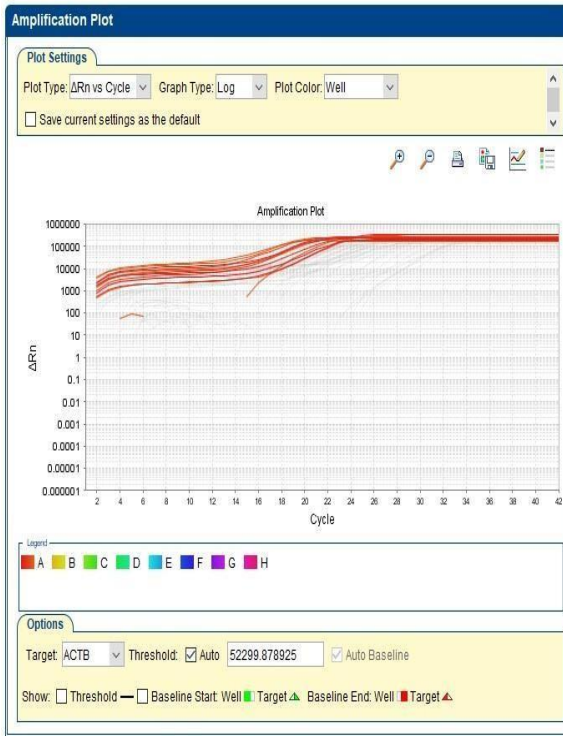

Figure 6. ARPC1B melt curve

4.3-Statistical analysis of qRT-PCR findings of the ARPC1B mRNA gene

QRT-PCR analysis was performed with step one plus software v2.3 for the expression of ARPC1B gene in pterygium and control conjunctiva tissues at the mRNA level. Normalized with ACTB, which is used as housekeeping gene, $2^{-\Delta\Delta Ct}$ formula was used to calculate the floor change. Statistical analysis was performed with the data we obtained. Analysis of the results was made according to the range 0.9 - 1.1. It was accepted that the expression level of the relevant gene decreased in the pterygium tissue compared to the normal conjunctival tissue in values less than 0.9, it did not change when compared with the normal conjunctival tissue in the range of 0.9-1.1, and there was an increase in the expression of the gene in values greater than 1.1 (Schmittgen and Livak., 2008). Statistical analysis of expression data was performed using SPSS 16.0 software. The significance levels of the data was made with Student test analysis If $p \leq 0.05$ was significant, if $p > 0.05$, it was evaluated that there were no difference.

T-Test

Table 5. Statistical analysis of $2^{-\Delta\Delta Ct}$ values of ARPC1B and conjunctival tissues

Gene	Tissue	
	Pterygium tissue (N=27)	Normal Conjunctiva (N=27)
ARPC1B (AVG±SEM)	2,514±0,837	
P	0,082	

(2,514±0,837-Foldchange, p=0,082).

4.3.1-Analysis results

Expression level of ARPC1B gene in pterygium compared to normal conjunctive tissue According to the findings of qRT-PCR, there was no discernible change. in ARPC1B gene levels of expression in pterygium and normal conjunctival tissues ($p>0.005$).

5-Discussion

Pterygium is a degenerative disease that manifests itself as a result of development of triangular tissue on the surface of the eye, conjunctival in the peripheral cornea Proliferation, inflammatory infiltrates, fibrosis, angiogenesis, and the extracellular matrix are all factors that affect cell proliferation. (Demurtas et al., 2011). Pterygium is normally asymptomatic, although it may induce dry eye symptoms including burning, itching, and lacrimation due to mixed humidity on the ocular surface. Advanced pterygium in the optical zone reduces visual acuity and necessitates surgical treatment (Mologen et al., 2017). Recent research suggests EMT (epithelial-mesenchymal transition) might have a function. in the etiology of pterygium, which might explain where pterygium fibroblasts come from (Jeanie, 2008). The pathogenesis of pterygium by p53 gene mutation has been studied extensively. It has been established that they are linked. As a result, excessive cell proliferation in pterygium is more akin to a tumor than to malfunction and a growth condition like neoplasia. (2015, Huang et al.). Pterygium causes vision problems by blocking the visual axis and causing considerable astigmatism (Helen et al., 2019). ARPC1B is required for the formation and maintenance of the ARP2/3 complex, it has a role in actin branching from a filament that already exists. They hypothesized that ARPC1B deficiency could cause cytoskeleton and functional defects in T cells, and they discovered biallelic mutations in ARPC1B in 6 unrelated patients with clinical features of combined immunodeficiency (CID), whose T lymphocytes were not studied but neutrophils and platelets were studied. (Dobbs K et al., 2015). They speculated that

ARPC1B loss in T cells might result in cytoskeleton and functional abnormalities.
Pancreatic cancer cell lines and primary pancreatic tumors account for 25% of all pancreatic cancer cell lines and tumors. ARPC1B was identified in a region of chromosome 7q22.1 that was amplified according to Laurila et al. (2009) study. Amplification resulted in enhanced ARPC1B expression, according to RT-PCR. ARPC1B silencing slowed However, there was no cell migration. impact on cell proliferation or invasion. ARPC1B may have a role in platelet dysfunction, in which a large number of platelets causes hard tissue sclerosis in the eye, and we know that pterygium disease is the formation of triangular tissue on the cornea. The goal of this research was to see how the ARPC1B gene affected pterygium
illness. Platelet Abnormalities with Eosinophilia, Immune-Mediated Inflammatory Disease, and Combined T Cell and B Cell Immunodeficiency are just a few of the conditions that may cause platelet abnormalities. diseases connected to the ARPC1B gene, and elevated ARPC1B has been associated to oral squamous cell carcinoma OSCC (Auzair et al., 2016). According to the findings of qRT-PCR, there was no discernible change. in ARPC1B gene levels of expression in pterygium and normal conjunctival tissues ($p>0.005$). Several human malignancies have been linked to caveolin-1 and Actin-Related Protein 2/3 Complex, Subunit 1B Based on their past study (Cheong et al., 2009), they identified two candidate genes, Caveolin 1 and Actin-Related Protein 2/3 Complex, Subunit 1B that were found to be differentially expressed in oral squamous cell carcinoma samples. It has been suggested in the literature that these two genes, Caveolin 1 and Actin-Related Protein 2/3 Complex, Subunit 1B are highly involved in (Routray, 2014). A recent research found that over-expression of ARPC1B increases tumorigenicity in breast cancer via causing centrosome amplification (Molli et al., 2009). ARPC1B has been Radiation-resistant intraocular choroidal melanoma cells were found to be overexpressed. in addition to breast cancer (Chia et al., 2015). The difference in expression of ARPC1B and Cav-1 between OSCC and normal oral epithelium in the present research suggested that these genes are involved in carcinogenesis. They also discovered that decreased ARPC1B expression was linked to lymph node metastases and advanced tumor stages in OSCC patients, indicating that the ARPC1B gene may play a role in the aggressive development of OSCC. Only a few researches have looked at the expression of ARPC1B and its clinical implications different malignancies, such as breast cancer (Molli et al., 2009) and intraocular choroidal melanomas (Molli et al., 2009) (Lukman et al., 2015). The recent investigation found a link between low ARPC1B protein expression and advanced stage of the illness and lymph node metastases. Methylation of the Arp2/3 complex's member p41- Arc may also cause downregulation of the complex, resulting in loss of expression and the formation of dysplastic morphology. Furthermore, Zucchini et al. hypothesized that the downregulation of ARPC1B gene in osteosarcoma is one of the causative consequences that leads to metastasis inhibition by reducing dynamic actin disassembly, which is required for a tumor cell movement. These findings point to ARPC1B's migratory activity in the modulation of focal adhesions and actin filaments, which aids tumor cell metastasis (Chorev et al., 2014). Although deletion of ARPC1B has been linked to a variety of malignancies, the mechanism by which this gene causes OSCC is unknown, and further downstream research is needed to determine its function in oral carcinogenesis. Increased expression of ARPC1B in OSCC and its link to advanced stage and lymph node metastasis adds to the proof that it plays a role in the genesis and progression of OSCC, the pterygium disease, which is unclear but is a tumor-like

tissue, and the molecular basis for pterygium growth, as well as supporting its neoplastic property. Pterygium is a condition defined by increased basal epithelial cell proliferation, neovascularization, and invasion of the neighboring corneal epithelium; nonetheless, viral infections play an important role.

Pterygium is a frequent eye illness, particularly in the tropics; as a consequence, ophthalmologists operating in hot climates must keep up to speed on current ideas in the understanding of the nature of pterygium as well as current treatment choices in order to achieve better outcomes. The start and maintenance of pterygium are dependent on hereditary susceptibility. Hereditary factors are more likely to influence the magnitude and seriousness of pterygiums. Predisposition to pterygium incidence almost certainly follows the polygenic model's multifactorial method of inheritance. Two kinds of genes, one for inhibitory smad proteins and the other for smurf proteins, may be inactive, leading to the development of pterygiums. Pterygium development seems to be dependent on fibro genic growth factors, and pterygium angiogenesis seems to follow fibroblast proliferation, collagen synthesis, and collagen lysis. Pterygium is triggered by sunlight, or maybe reactive oxygen species. Inflammation and collagen damage, which are almost certainly caused by oxidative stress, seem to increase pterygium growth. Nonetheless, there have been considerable breakthroughs in our knowledge of the nature of pterygium recently, and breakthroughs in therapy have not only continued to lower recurrence rates, but they may also allow alternate methods and treatments to be used instead of surgery. Current treatments try to reduce the pterygium's recurrence rate after it has been surgically removed. Furthermore, advances in understanding the molecular basis of pterygium pathogenesis, in conjunction with treatments and surgical adjuvants, point to the development of effective pterygium treatments, as well as the prevention and eradication of recurrence following surgery. The current research reveals that pterygium has an influence on ocular surface characteristics, including direct changes in meibomian gland pattern and tear film. The results show that the ARPC1B gene has no influence on pterygium illness, and further research is required in the future to establish the effectiveness and see whether there is a link between the ARPC1B gene and pterygium. More research on the gene ARPC1B should be done in the future, employing various studies with more patients to gain various findings and better understand the influence of the ARPC1B gene on pterygium. The start and maintenance of pterygium are dependent on hereditary susceptibility. Hereditary factors are more likely to influence the magnitude and seriousness of pterygiums. Predisposition to pterygium incidence almost certainly follows the polygenic model's multifactorial method of inheritance. Two kinds of genes, one for inhibitory smad proteins and the other for smurf proteins, may be inactive, leading to the development of pterygiums. Pterygium development seems to be dependent on fibro genic growth factors, and pterygium angiogenesis seems to follow fibroblast proliferation, collagen synthesis, and collagen lysis. Pterygium is triggered by sunlight, or maybe reactive oxygen species. Inflammation and collagen damage, which are almost certainly caused by oxidative stress, seem to increase pterygium growth. Nonetheless, there have been considerable breakthroughs in our knowledge of the nature of pterygium recently, and breakthroughs in therapy have not only continued to lower recurrence rates, but they may also allow alternate methods and treatments to be used instead of surgery. Current treatments try to reduce the pterygium's recurrence rate after it has been surgically removed. Furthermore, advances in understanding the molecular basis of pterygium pathogenesis, in conjunction with treatments and surgical adjuvants,

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Declaration of Author Contributions

The authors declare that they have contributed equally to the article. All authors declare that they have seen/read and approved the final version of the article ready for publication.

Declaration of Conflicts of Interest

All authors declare that there is no conflict of interest related to this article.

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