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College of Medicine

Department of Community and Family Medicine

Prevalence of congenital color vision deficiency in a sample of students of medical colleges group at university of AL-Qadissiyah

A thesis

Submitted to the council of the college of medicine / university of AL-Qadissiyah in partial fulfillment of the requirement for the degree of higher diploma in family medicine

By

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1439H 2018AD

بسم الله الرحمن الرحيم

وَمِنْ آيَاتِهِ خَلْقُ السَّمَاوَاتِ وَالْأَرْضِ وَاخْتِلَافُ أَلْسِنَتِكُمْ

وَأَلْوَانِكُمْ إِنَّ فِي ذَلِكَ لَآيَاتٍ لِلْعَالِمِينَ ۝

صدق الله العلي العظيم

سورة الروم / الآية ٢٢

Dedication

To everyone who assist me especially my family and my supervisor

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Thanks for our God

Who gave me the power to produce this thesis which I wish to be useful and objective. I would like also to express my gratitude to my supervisor **Dr. Furkaan Majied Hamied** for her great assistance, kind advice and scientific guidance

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Supervisor's certificate

I certify that this thesis was prepared under my supervision at the scientific council of ophthalmology college of medicine AL-Qadissiyah university as a partial fulfillment of the requirement for the degree of higher diploma in family medicine

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Recommendation the Head of community and family medicine Department

In view of the available recommendations, we forward this thesis for debate by the examining committee.

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2018 / /

Committee certificate

We the member of examining committee certify that after reading this thesis (**Prevalence of congenital color vision deficiency in a sample of students of medical colleges group at AL-Qadissiyah university**) and after examining the student (**Hayfaa Hussin Jabar**) in its contents we found it is adequate for the degree of Higher Diploma in family medicine

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Abstract

Back ground

Color vision deficiency is an important X linked autosomal recessive visual defect affecting the perception of colors.

Objective

To determine the prevalence of color vision deficiency among a sample of students in medical colleges group at university of AL-Qadissiyah (medical colleges , college of pharmacy and nursing college)

Method

Across-sectional study done in university of AL-Qadissiyah at the period from April 2018 to June. 2018 the study was carried out to assess the prevalence of color vision deficiency among a sample of medical colleges group students.

Result

A sample of 814 students were enrolled in this study 252 males and 562 females with age range 18 – 24 years mean age 20.51 ± 1.49 years total positive cases were 15 student 13 males and 2 females.

Conclusion

Prevalence of color vision deficiency in a sample of medical student is (1.8%) with prevalence in male (5.2%) and in female 0.4%

Deutan(green CVD) more than protan(red CVD)

There is no relation between color vision deficiency and the degree of parent relationship.

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List of Abbreviations

CVD	Color vision deficiency
LWS	Long wavelength sensitive
MWS	Medium wavelength sensitive
n	Number
NS	Not significant
SD	Standard deviation
SWS	shortwavelength sensitive
UK	United Kingdom

Chapter One
introduction

Chapter one

1- Introduction

Color vision deficiency is a chief disorder of vision that disturbs high number of people either inherited or acquired. (1 – 3)

People who have color vision Deficiency noticed to be unable to differentiate and to percept the three major colors which are present in every part of the life red-green and blue a red and green are more highly prevalent than blue colors which are relatively rare.(2)

The complete disability to notice the color is termed achromatopasia which is uncommon disorder patient cannot see any color. This disorder is rare, inherited as autosomal recessive due to abnormality in the genes.(3)

CVD had to be classified from partial to complete according to the cause; both eyes might be affected if it is congenital and usually just one if it is acquired that is mean caused by injury or illness.

Color vision is determined by a photo receptor of the mammalians eyes retina termed the [cones cells]. Those [cones cells] contained illumination – perceptive pigment which receive and be aware of the colors, institute at the macula at the a innermost portion in the retina, every one of those [cones cells] are responsible for one of the three main colors [blue ,green ,red] . These[cones cells] can distinguish those colors depending on the wavelength of them".(2)

Generally the commonest variety of color deficiency is the red green which that person who has this color insufficiency may be unable to distinguish these colors totally or partially.

They simply have a difficulty to differentiate those colors and that is related to dimness and brightness of those colors.(3)

The other type from CVD [blue and yellow CVD] which is an exceptional in severity rather than [red and green CVD]. Those citizens who have suffer from blue and yellow CVD mostly had [red and green] CVD also.(2)

Chapter Two

Literature Review

Chapter Two

2-1- definition

The Color vision blindness is the disability to notice some colors or pick out their difference.(3)

2-2 Physiology of color vision

The mammalians retina contain two kinds of cells that receive light. They are termed as rods and cone [Rods] is responsible for the awareness of brightness as well as darkness and the sensitivity to low light level while the Cones cells can detect colors and are concentrated near the center of the vision. [3]kinds of a specific cell which are[cones] that see color: green and red and blue. In the mammalian higher center perception of colors takes place after using input from these cone cells. (4)

There are many theories have been effort to give explanation of the properties of human being colors vision but only two of those theories were generally famous the Trichromatic theory of Young, Helmhoz and Max well postulate three kinds of elements each with resonance which to gather could represent any color. In the recent time, micro spectra photometric studies on creature cone cell have confirmed that there are three classes of cones in the retina;" long wave length sensitive (LWS or red cones) with peak sensitivity 570 – 590 nm. medium wave length sensitive (MWS or green cones) with a peak of 535 – 550 nm. short wave length sensitive (SWS or blue cones) with a peak of 440 – 450 nm". The challenger color theory (of Hering) identifies that some colors appear to be (mutually exclusive). There is no such color as red green and such phenomena is not easy to

be explained on the basis of trichromatic theory only. generally it seems that both theories are useful. Color vision is trichromatic at the level of the photoreceptor, while the color opposes is determined by subsequent neural processing. (5)

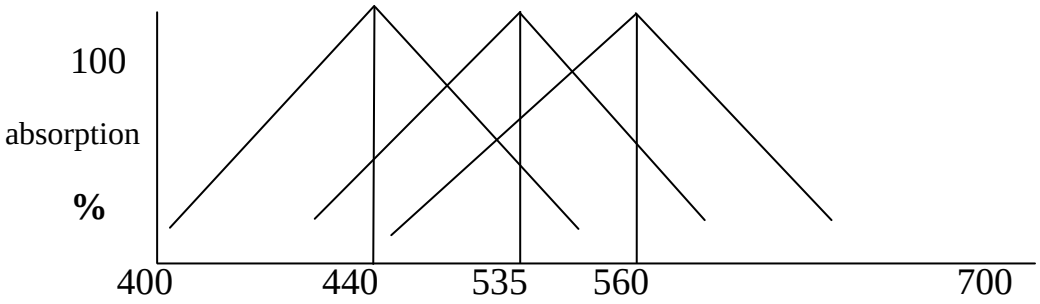


Figure (1- 1) spectral sensitivity of cones pigment. (6)

2-3-The Types of color vision deficiency

The Color vision deficiency happens when one or more of [3]kinds of color receptive[cones]cells red and green as well as blue do not precisely draw together or throw a right color impulses to the optic nerve . The CVD may be hereditary or due to many other causes that affect the color vision. The hereditary is habitually linked to the X chromosome red and green CVD so as it is more occurs in boys than girls. It may be less frequently an autosomal dominant quality blue and yellow CVD and so infrequently an autosomal recessive congenital feature[Achromatopsia] total color vision deficiency (7-8-9).

The Achromatopsic patient almost always has an additional defect with vision including decreasing visual Acuity and hyper sensitivity to light (photophobia) and small unconscious eye motion (nystagmus) (10)

The inherited type is not pathological, untreatable, and permanent throughout the patient years of life, while the color vision defect caused by

another causes rather than inherited causes for example systemic illnesses or injury causing damage to the optic nerve or the retina be capable of defecting the acknowledgment of coloring visualizion. disease like glaucoma, diabetes mellitus, neurological degenerative diseases , medication with harm full outcome, advancing age , injury . [11]

The condition is divided in to three major categories: red-green CVD . The second categories blue –yellow CVD and a complete absence of color vision a persons with a red-green defect related to a loss or abnormality of the red sensitive pigment are said to have a protan defect protanomaly and protanopia according to the severity of defect while those with loss or abnormality of the green sensitive cone pigment have a deutan defect also according to the severity (Deuteranomalous and deuteranopia). Yellow-blue CVD is a tritan defect also either tritanomalous or tritanopia. [11]

A good number widespread CVD is the red and green color which is called Daltonism [12 – 13]

The deficiency of red green color with it is sub type further widespread than blue(CVD) that is so less frequent. [14 , 15 , 16]

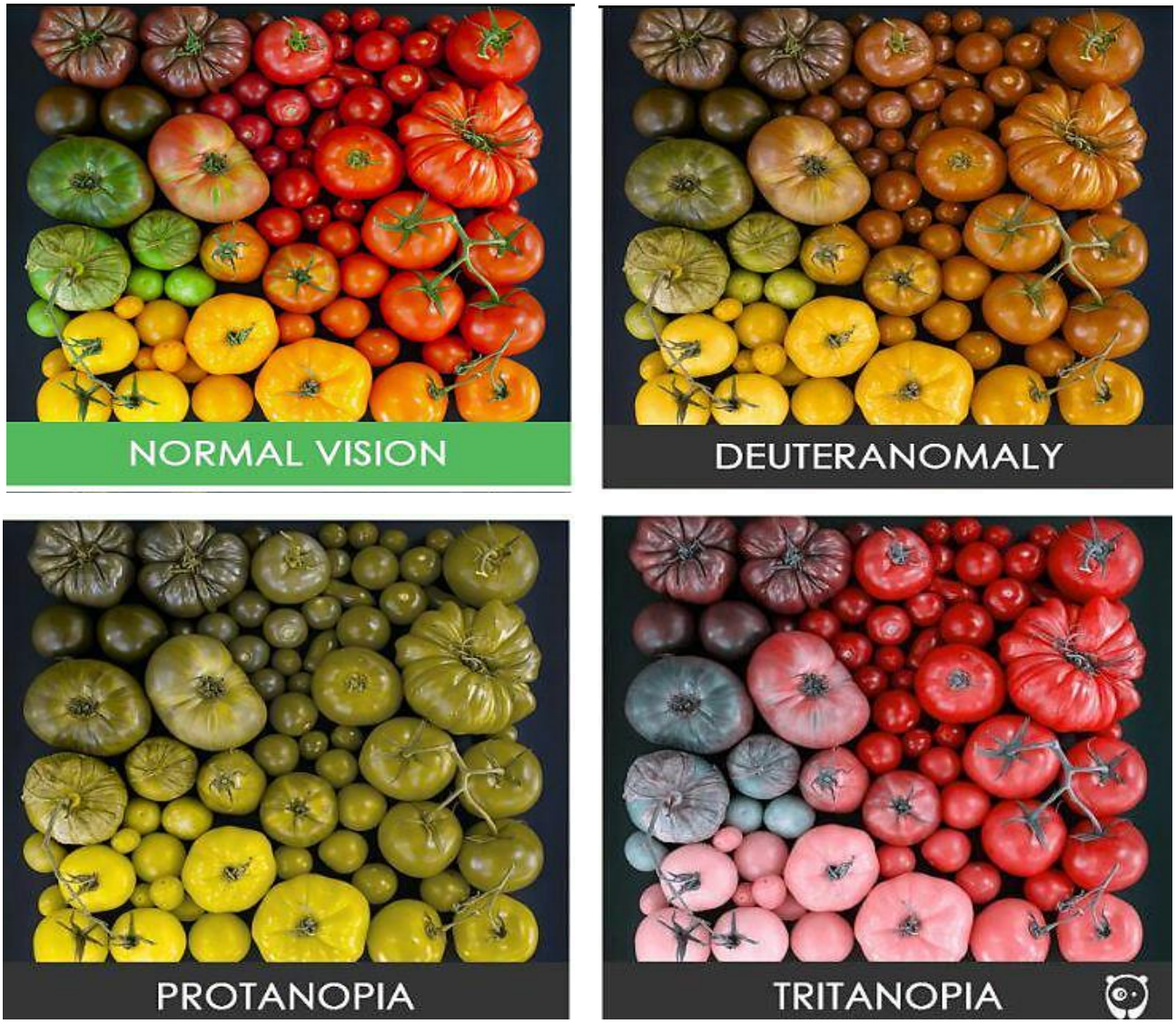


Figure (1 – 2) types of color vision deficiency. [39]

2- 4-The Genetics of color vision deficiency

There is difference at a few amino acid position that controls the spectra of the L (long) and[Medium] canes cells pigment responsible to the many variation that occurs .Genes that are programming Long –medium photo sensitive pigment were set as top to end manner at X gene .Those were severely homologous ;this will subject them to universal cross over resulting in gene deletion and forming of L / M hybrid genes which are code a variety of color vision defect. these event as a whole are present in the retina accountable to causing coloring visualization. They are congenital recessive traits.

Tritanopia a rare autosomal dominant CVD which is caused by mutation in S (short) pigment gene located on chromosome(7).

The complete color blindness (achromatopsia or rod monochromacy) is a infrequent autosomal recessive trait caused by mutation in genes programming the proteins of the photoreceptor cat ion channel or cone transuding that are important for function of all classes of cone [(17)]

Abnormality that happened at [OPN1LW , OPN1MW and OPN1SW] chromosomes lead to formation of CVD. Genetics change that involve [OPN1LW /OPN1MW] chromosomes resulting in [red and green CVD]. And this means that the genes abnormality causing disappearance of the [Long or Medium] cone cells or lead to the invention an incorrect pigment at those cone cells which disturbed the [Red and Green coloring visualization].

The CVD of[Blue and Yellow] type is a consequence of abnormal mutation at [OPN1SW] chromosome. Those are an abnormal mutation resulting in early devastation in [S] cone cells or creation an abnormal [S] cone cells. The functional Impairment of S cone lead to alteration of blue color perception , so that is lead to disability or hardness to distinguish or differentiate the [Blue and Green] colors, causing difficult individualize dime Blue color rather than dime color .[18]

2-5- The inheritance pattern of color vision deficiency

The color vision defect of Red – Green and blue cone monochromic are inherited as X linked recessive pattern so that males need one genetic change in each cell to be affected by the condition. Male are frequently effected more than female because in female genetic change should happened on both copies of the chromosome to cause the disorder. A characteristic of X-linked inheritance is that fathers cannot pass– X- linked trait to their sons.

While Blue – yellow CVD is an autosomal dominant inheritance pattern that means one copy of the abnormal OPN1SW gene in each cell is adequate to cause the disorder the affected person inherits the condition from an affected parent. (18)

The abnormality in the CNGA3 , CNGB3 , and GNAT2 genes are accounting for achromatopsia. Any of these genes provides information to make a protein that is drawn in the normal function of cones in the retina .The alteration in each of these chromosomes causing the [3]type of cone cells not capable to proper reaction for illumination. So , the affected persons with this alteration at any

chromosome will relay on rod cell unaided to visualize object. Those in general without coloring visualization with many vision defects also. Other persons with mutation in [CNGA3] chromosome have partial achromatopsia, that will permit few cones cell gathering with narrow coloring visualization. (19)

A specific entity for partial achromatopsia which is [Blue cone cell monochromacy] ,result from chromosomal mutation put a stop to [Long and Medium] cones cells to work precisely . So those patient will has just [S]cone cell. for this reason that the brain should judge against impulses of just[2]kinds of cone cells to notice coloring object , those with acting just [S]cones cell will be so poorly coloring visualization. (19)

2-6- The Acquired color vision deficiency

CVD can be acquired as an effect of neurologic ,optical, , or general illnesses. There are many different conditions which concern coloring visualization, like illnesses in visual medium in the course of disorder in visual cortex. The acquired CVD is a separated article than inherited CVD, but there is some information that show same overlie.(20)

The acquired CVD has a different subtype according to the what part of the visual system affected including the retina, optic nerve, and visual cortex affected by the disease process or the damage. (21)

The general ageing process by it self may cause different changes to vision as well as color perception. They can progress, for example, from normal trichromacy to anomalous trichromatism on to adichromatic stage and even to monochromatism

(where most color vision is lost) or they may be moderately stable .Sometime after healing or with – drawl of the cause, color vision may usually go back to normal through these phases if the color loss had been considerable. (22)

There are different causes which may disturb color vision like Parkinson's due to the neurological defect on the light sensitive nerve cells in the retina in which vision processing take place.

The diseases of the eyes may cause CVD like cataracts, glaucoma, diabetic complication to eyes, retinitis pigmentosa and age related macular degeneration.

Other inherited syndrome like Kallman's syndrome inherited disorder involve failure of the pituitary gland also cause CVD. Drugs and chemical agent like Tiagabin antiepileptic drug can cause CVD in about 41% of those taking the drug, although outcome do not appear be permanent. (23)

There are some points that should be distinguished in acquiring color vision deficiency like Color loss may be confined to one eye and or restricted in one part of the visual field.

Color loss may be accompanied by defect in other visual areas, remarkably a decrease in visual acuity, visual field defect impaired dark adaptation, brightness perception contrast sensitivity or flicker sensitivity.

The blue – green – yellow vision abnormality are as more common than red – green vision in acquired forms also females are seems to be affected in the same percentage with male .the elderly population is more susceptible due to high incidence of damage(cataract)

The degree of severity of the defect is erratic according to the severity of diseases or degree of exposure to the drug or chemical.

temporary chromatopsia (appearance of color on white surface / objects) may be present and the color may name in a corrected way by the people with acquired color deficiency on the background of their previous memory for color before the injury.

There is also a difficulty in the diagnosis especially in differentiation the defect during clinical testing takes place. Sometime acquired defect may be mimic inherited defect so that very carefully examination is required.

Every abnormal or sudden color vision disturbance or report of change in color perception should rise the suspicions of an acquired anomaly.. [22]

2-7- The effect of advancing age

Generally deterioration in color vision naturally happened after the age of 40 years with a reduction in visual acuity. This is because of a reduction in the sensation of the cons receptors, also the buildup of yellowish pigments in the lens of the eye and at the center of the retina (fovea) this will decrease the quantity of blue light available for perception, consequently, very slight blue defect are notes with advancing age in many individual. [22]

2-8- How patient can present

The colors are seen different by people with normal color vision can look identical to people with defective color vision, for example apple may appear green with normal color vision but look to be unchanged color as orange for population with a particular color vision defect.

At the same time a noticeable reduction in the quantity of separated colors which can be illustrious in the spectrum. (23)

Terminology of color blindness is confusing, that is due to most color blind individuals are see colors, however their color perception is limited and imprecise. Red –green CVD is the frequent form of color abnormality causing incorrect perception of the colors red and green so it easy to confuse them. (24)

Figure (1-3) comparison between normal and C.V.D picture. (39)

2-9- Effect of color vision deficiency on the person life

Population with abnormal color vision face many difficulties in everyday life which is normally done easily by normal people who are not aware of problems can arise with even a very simple activities as choosing and prepare food,

agriculture, activity, driving car and clothes choosing ,cosmetic make up and arts. (25)

Many of everyday jobs like food processing to the proper color or choose ready products can be difficult for a persons and their children may found the food without happy colors less appetizer.

The traffic light during driving also make challenges, that is because they have to be understand by the position of the light since most light are vertical with green on bottom and red on top if alight is positioned horizontally color defected people has to do immediate mental rotation to read it, so as map reading or clothes buying that match color can also be difficult, however these are relativity minor that every affected people are learn to adapt. (26)

In some countries like the UK color blindness is not presented to be a disability, while in others color defected persons are regarded as disability. For example in Japan, color blind citizens are excluded from number of jobs and in many countries color blind people are not acceptable for driving because they are not able to read colored lights appropriately. (25)

2-10- How CVD diagnosed

During examination there are some point should be under taking

The inspector should be examined firstly for color vision deficiency.

The examiner should be qualified well for testing and explanation of the result.

The correct kind and intensity of illumination as specified is very important as it has been shown that color blind people can pass clinical test under differing illumination condition.

The place of testing should have either a natural sky illumination or artificial day light fluorescent illumination Tungsten lighting is unsuitable.

The test book should be closed after using and stored in their box away from direct sun light.

Each eye should be examined separately when acquired defect is suspected. (22)

Types of CVD tests

1- The Ishihara plate test.

This test is an important color perception test for red and green color defect the first in a class of successful color vision test called pseudo – iso chromatic plates (PIP). It was named after its designer Dr. Shinobu Ishihara, a professor at the university of Tokyo, who first published his tests in 1917. (27)

The Ishihara test consists of a number of colored plates which called Ishihara plates each of which contain a circle of dots appearing different in color and size.(28)

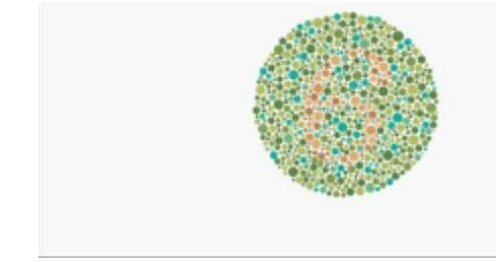
In these plate there are dots which form a number or shape noticeably visible to normal color vision people and indistinguishable or difficult to see to those with red and green CVD .The other plates are purposely designed to reveal number only

to those with red and green CVD and be invisible to those with normal red and green color vision.

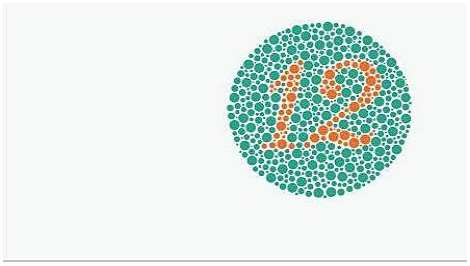
A complete test consist of 38 plate but in the presence of sever deficiency is usually apparent after only a few plates.

There is also an Ishihara test consisting (0 , 14 or 24 test plates) [29] .The plates make up several different test designs. [30]

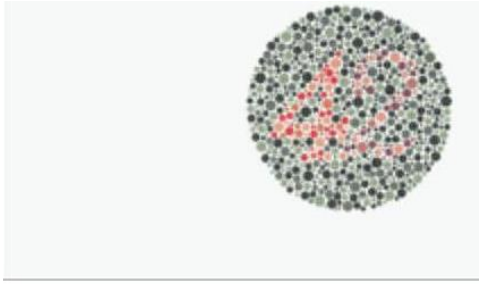
- The demonstration plate number one the figure 12 designed to be detectable by all person, whether normal or CVD. For demonstration only and not considered in a scoring for screening purposes.
- The vanishing plates: the human being with normal color vision could known the figure only.
- The hidden digit plate: the persons with CVD could recognize the figure only.
- The diagnostic plate: projected to resolve the type of color vision defect (protanopia or deuteranopia) and the severity of it.



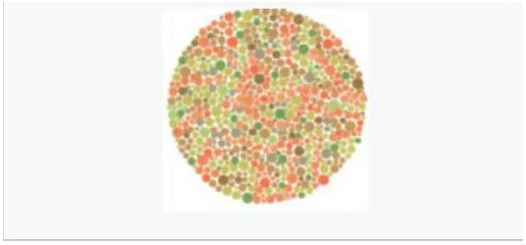
Ishihara Plate No. 13 (6)



Ishihara Plate No. 1 (12)



Ishihara Plate No. 23 (42)



Ishihara Plate No. 19 (Nothing (hidden digit plate); Red-Green deficiency sees 2)

Figure (1-4) sample of Ishihara plates. [30]

Limitation of Ishihara plate test:

- Not as such a diagnostic test, the test does not screen for blue tritan defect and unsuitable for testing acquired defect.
- There is no record sheet and the examiner has to understand the principles of the test.
- The test is easy to use, however, interpretation of the results is not always easy if only a few plates are failed.[32]

Way of examination

The examination is done by holding the plate by an examiner hand at arm length just about 70 cm parallel to the eye and the examiner turns the plate, with viewing time about 3-4 seconds for each plate [31 - 32]

Interpretation

Many of the color defective group make 12 or more errors on the 16 transformation and vanishing design with 99% of color deficient recognized at least six errors. This pass / fail criterion is adequate as screening formula to determine color deficiency. Although subject with normal color vision may make one or two mistakes but errors and miss readings differ qualitatively. [22]

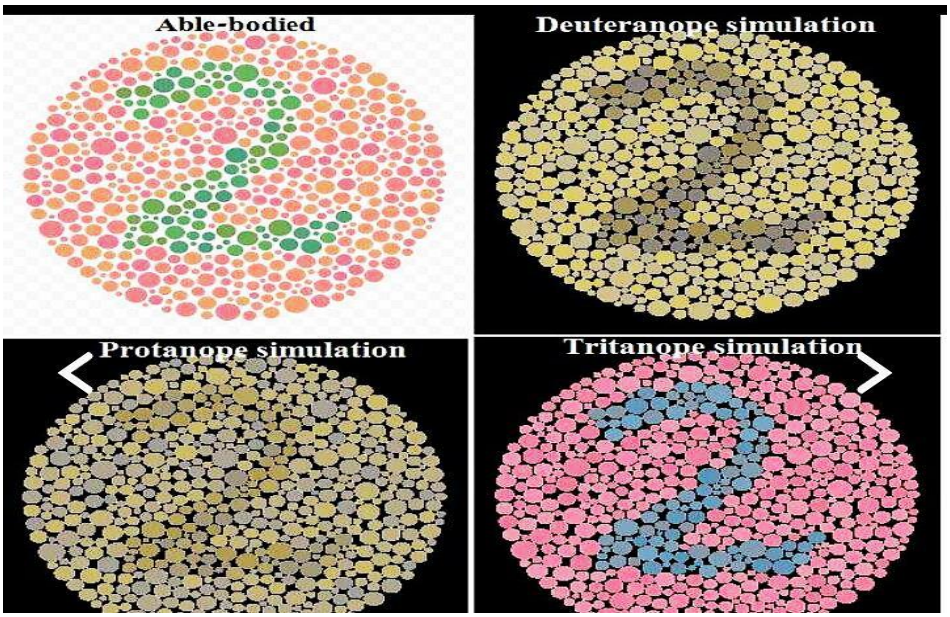


Figure (1-5) How the patient with CVD can see the number. [39]

2- The city university test.

The City University Test [TCU] made of 10 plates every one containing a central color and four peripheral color. The person choose one of the peripheral colors which more closely matches the central color. The degree of defect is determined by the number of errors, if a mild defect showing few errors and a sever defect making a maximum number of errors .but is seldom used in practice.(31)

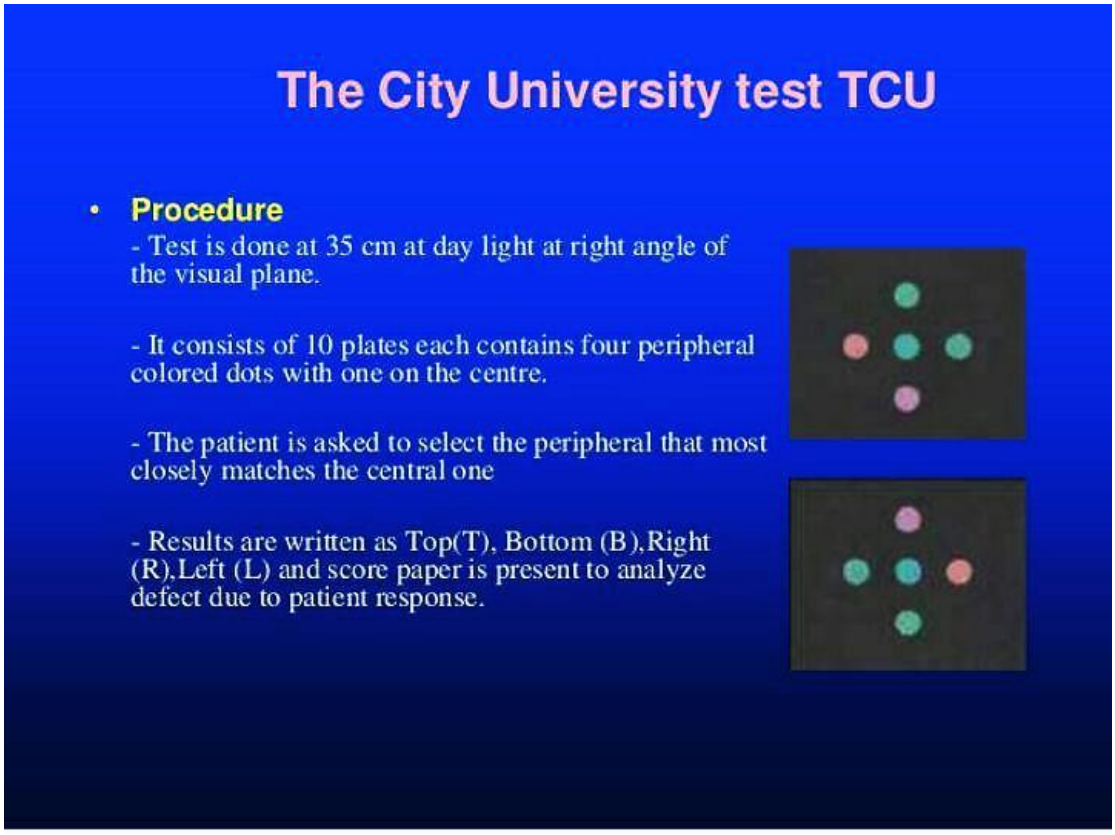


Figure (1-6) example of city university test. (39)

3- Richmond HRR test 2002 :

This test a good toil that includs at fundamental testing series to the judgment of the coloring visiualization. It is considered as affirmation the product of a [Ishihara test] able the examiner if the person had been informed about the presses response to a Ishihara test . It is a sensitive and specific as a Ishihara test. Tritan defects can be discovered by it. The use of signs is able the kids to do the test , a circular, a triangle and a cross, that could be name or identify by kids. on the other hand, key cards is prepared so kids able to name the signs that seen [22-31]

20 plate are present [4]to test Red /Green (protan / deutan)color vision defect [2] to Blue / Yellow (Tritan / Tetartan)color vision defect , ten to classify /assess the rigorousness of Red /Green color vision defect .[Four]to classify /assess rigorousness of[Blue /Yellow]. [33]

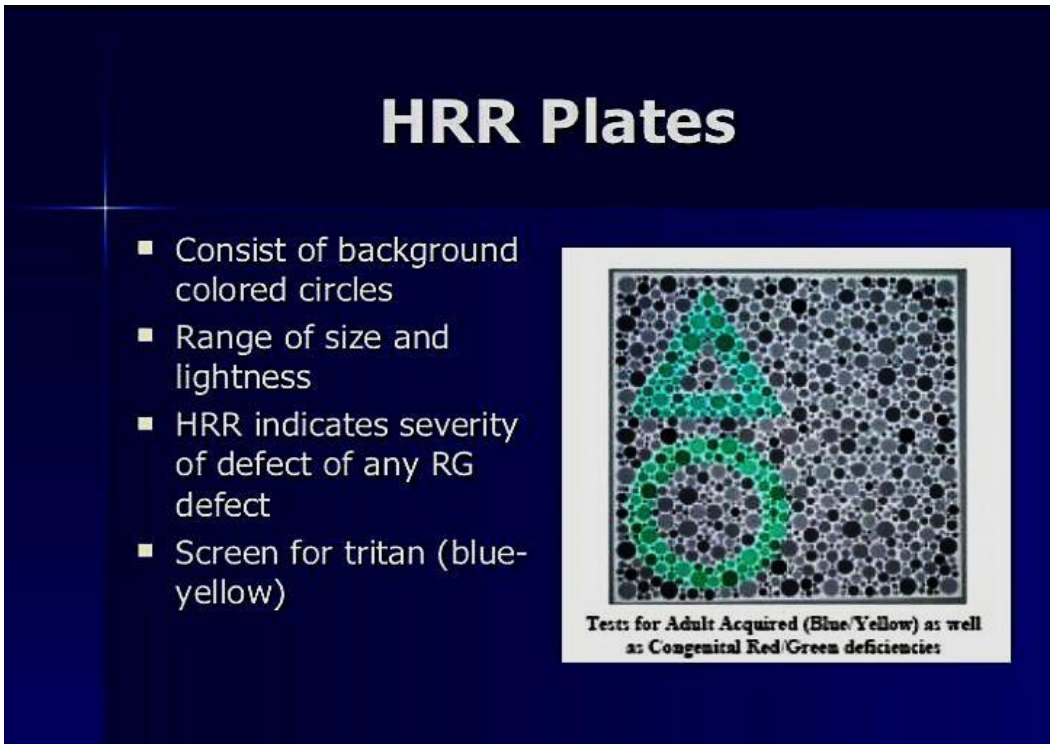


Figure (1-7) diagnostic plate from the Richmond HRR test. [39]

4- Medmont c- 100 test

This test is used to measure relative spectral sensitivity using flicker photometry to differentiate Red and Green CVD. [33]

The unique action of this test is to make a distinction Red /Green CVD along with patient having Red /Green atypical coloring visualization . The test is sensitive /specific but easy on the pocket take just minutes to manage. [22]

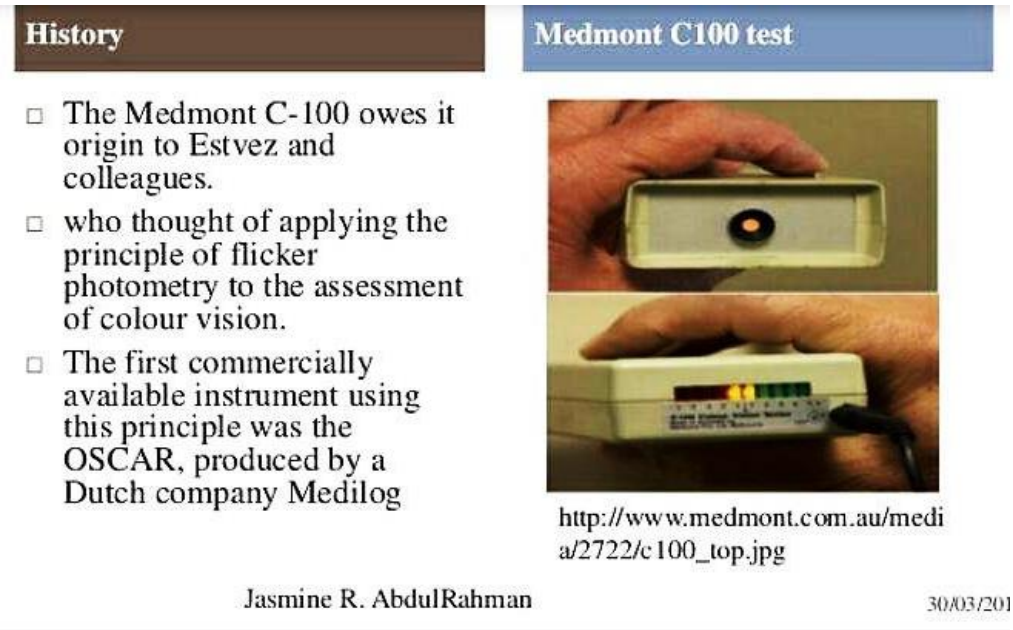


Figure (1-8) Medmont c.100 test. [39]

5- Fransworth D 15 test

This test termed as(dichotomous) for the reason that the design of it dividing objects into single from ;

a. powerfully coloring defect
b. slightly coloring defect /normal coloring vision.

That will be skilled by collection the drenched coloring box.

A normal coloring percept showing good mark .Medium/Sever CVD will show low marking . [34]

Those person that undergo examination arrange the 15 loose color caps in order of color starting from the fixed color cap. A persons having good coloring visualization does a miner incorrect answer , also is done by mildly color vision defected individual .While Those with moderately and severely color vision defected individuals made many striking mistaking as setting colors which be positioned at a reverse area of a coloring encircle, these which site at the mystification site, then to any other. [33]

6- Fransworth – Munsell 100 – hue

This is one of the important type of CVD test which were discovered by Dean Farnsworth in the 1940. It is used to determine the ability to differentiate colors using the value and Chroma that have been prescribed by the Munsell color system. [35]

Despite it is sensitivity for inherited and acquired CVD but it is rarely used . the test made of 85 hue caps which contained in 4 different racks. In each of which the 2 end caps are fixed and the others are mobile so the examiner can change their site then the examined person is tolled to repeat the arrangement of the mobile caps as the previous order in the box after closing the box it will turned vertically and opened to visualize the inside markers accumulative manner then used to collect the finding on a circular chart and any of the dichromatic sub type is represented by failure in a special line of the chart. [22 , 31).

Figure (1-9) fransworth – munsell 100 hue test. [39]

2-11- Treatment of color vision deficiency

There is no treatment available for abnormal color vision, although recent studies offer a prospect for gene therapy.

"Gene therapy has cured color blindness in monkeys according to study results announced in September 2009 by researchers at the university of Washington and university of Florida.[23]"

Most of color blind persons reimburse for their disability by color cues and details that are not knowingly manifested by people with a normal color vision. There are ways to work around the inability to see certain colors by.

- In the Eastern provinces of Canada horizontally mounted traffic lights are generally differentiated by shape to facilitate identification for those with color blindness.[36]
- Make labeling for the clothing, furniture or any colored objects using the help of friends or family to be easily recognize.
- By depending on remembering the order of things rather than their color which can increase the chances of identifying colors for example a traffic light as red on the top, yellow in the middle and green on the base. [37]
- There are also a glasses with colored lenses or colored contact lenses are designed for helping those with abnormal color vision to a degree they can help pass color vision test but does so by altering the colors in the tests not by improving color vision.. [37]

- A colored filters can be used in specific everyday jobs, for example it is often difficult for medical practitioners and optometrists who have defective color vision to discriminate melanin pigment spots from small hemorrhages when examining the retina of the eye by ophthalmoscope.. [37]

2-12- What are the possible complication of color blindness?

The complications which are linked to color blindness include.

- livelihood limitations.
- Difficulty in performing certain regular daily task.
- Difficulty in the driving, specially distinguishing between traffic light colors[38]
- In some position like in the armed force customs and excise officers, fire service officers, hospital laboratory technicians pharmacists, electricians, certain flying related roles, such as pilots and air traffic controllers, jobs involving paint, paper or textile manufacture train drivers and railway maintenance staff. [36]

2-13- How can color blindness be prevented?

- As the color blindness are mostly inherited, so as these may not be prevented
- Nevertheless; the early detection of the disorder in kids can help understand its nature and severity, which would allow the use of suitable appraise to control any complication and to reduce learning difficulty soon after .
- So that the screening of vision should be routinely done.[38]

2-14- What is the prognosis of color blindness?

Commonly, the CVD is a lifelong condition, nevertheless, the affected persons are able to go ahead in a normal life, but only with some mild difficulties[38].

2-15- Children with color blindness

If a child has color vision deficiency, he may struggle at school unless the teacher is made aware of the problem .That is because many learning materials are color roundabout, the effected child may find it more difficult than most if their learning environment is not adapted to meet their specific needs [36].

AIM of the study

The aim of our study is to look for the prevalence of CVD in a sample of students in the medical colleges group (college of Medicine , college of pharmacy and college of nursing) at university of AL-Qadissiyah in AL-Diwaniah city.

Chapter Three
patient & Method

Chapter three

3-patient and Method

3-1- The design of study

Across sectional study designed to find the prevalence of CVD among a sample of students in the medical collages group at AL-Qadissiyah university in a period from April 2018 – June. 2018 a sample of 814 student 562 female and 252 males

3-2- Inclusion criteria

1- Healthy student age 18 – 24 years.
2- Best corrected visual acuity of 6/6 or better.

3-3- Exclusion criteria

1- Student with history of ocular Trauma or surgery.
2- History of medical diseases like Diabetes or Hypertension.
3- History of using drug that affect color vision like digoxin , anti-epileptic drug and barbiturate.

3-4- Collection of data

Data were collected using a pre-constructed data collection form, which was formulated for the purpose of this study. The general characteristic of the collection formula were

1- Name.
2- Age.
3- Gender.
4- Occupation.
5- Past medical history.
6- Past ocular history.
7- Family history.
8- Dose the parent relative or not ? second degree relative considered as positive any other considered negative.
9- Result of examination.

3-5- Way of examination

All students after taking their permission for examination were examined for best corrected visual acuity using Snellen chart. CVD was tested by using pseudo-iso chromatic Ishihara plates which is a good and quick process of examine the defected color vision from that vision which is normal . We consider using Ishihara plates of 38 plate were used by putting the plate in front of the Student at 70cm in the day light not direct sun light . Each plate has been offered to the student for three to four seconds and they were asked to read all numbers presented in the plate .

Plates from 1 to 17 revealed the normality or abnormality of color vision if 17 plates reads correctly this mean normal color vision, when the student sees thirteen or less this mean defect in color vision red - green defect.

The plate 22 to 25 were used to differentiate red color defect kind and green color defect kind.[40]

The plate 30 to 38 were used when the patient cannot read the number in plates determined the lines between a two X should be done and completed at ten seconds .

Finally the result of an examination were collected in preformed formula analysis statistically by soft were programmed version 23 Descriptive statistics were presented as (mean ± standard deviation) for age. While categorical variables (gender, degree of parent relativity, test result) were presented as frequencies (number) and proportions (%)

Chi square test and T test were used to assess statistical significances a p- value of< 0.001 regarded as statistically significant.

Chapter four
Result

Chapter four

4-Results

The study, as stated in the chapter of patients and methods, included 814 students with a mean age of 20.82 ±1.58 years and an age range of 18 to 24 years. Male subjects comprised 252 out of 814 (31.0 %), whereas, female subjects contributed to 562 out of 814 (69.0%), as shown in table (4-1). Mean age of male subjects was not significantly different from that of female subjects, 21.52±1.56 years versus 20.51± 1.49 years, respectively (*P*=0.137).

Table(4-1): General features of the study group

Characteristics	Value
Number of cases	814
Age	
Mean ±SD years	20.82± 1.58
Range (Min.-Max.) years	6 (18 – 24)
Gender	
Male, n (%)	252 (31.0%)
Female, n (%)	562 (69.0%)
Color blindness	
Total , n (%)	15 (1.8%)
Protan defect , n (%)	4 (0.5%)
Deutan defect, n (%)	11 (1.3%)
Parent, relative, n (%)	244 (30.0%)

N: number of cases; SD: Standard deviation,* Independent samples t-test; NS: not significant

4.2 Rate of color blindness

The rate of color blindness in the study sample was 15 out of 814 (1.8%), as shown in figure 4-1. Patients with protan (red color) blindness accounted for 4 out of 814 (0.5%), whereas, patients with deutan (green color) blindness were more frequent and accounted for 11 out of 814 (1.3%), as shown in figure 4-2 and table 4-2.

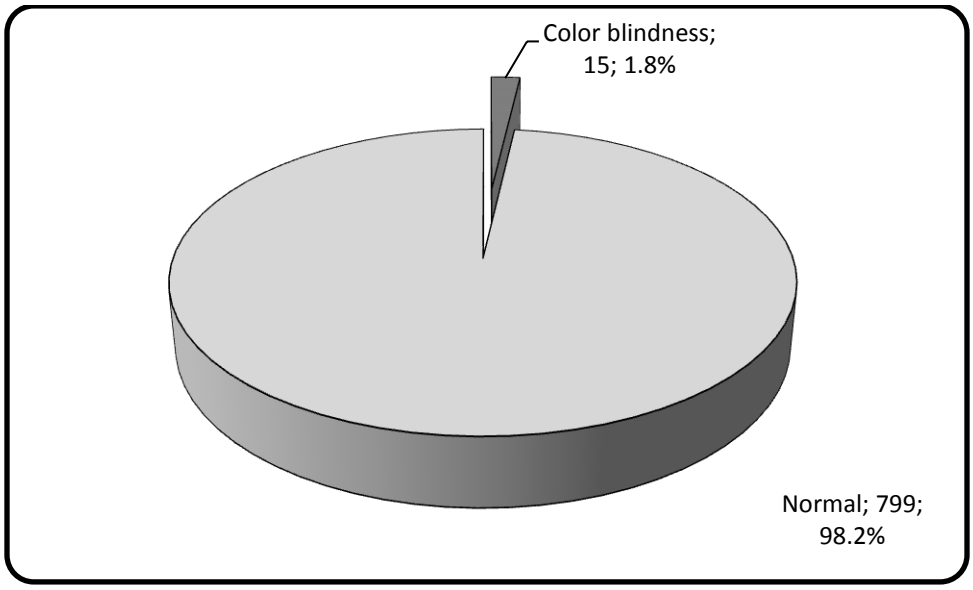


Figure 4-1: Pie chart showing the rate in percentage of color blindness among study sample

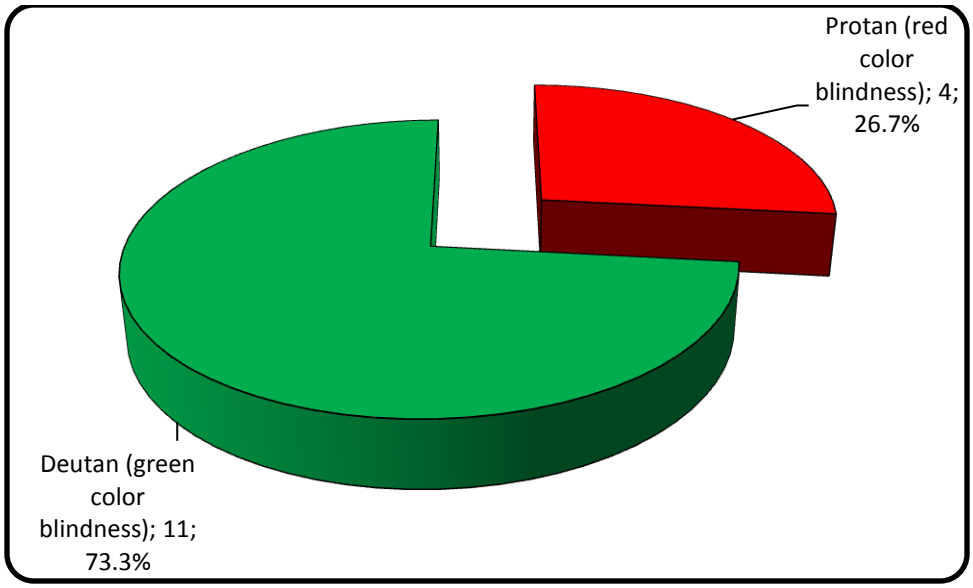


Figure 4-2: Pie chart showing the proportion of patients with protan (red color) and deutan (green color) blindness

Table 4-2: Proportions of patients with color blindness

Characteristic	***n***	**% out of total**	**% out of patients**
Color blindness	15	1.8	100
Protan (red color) defect	4	0.5	26.7
Deutan (green color) defect	11	1.3	73.3

- No case of total CVD is found
- No blue – yellow CVD can be detected.

4.3 Association between age and color blindness

Mean age of all patients with color blindness was 21.33 ±1.68 years, whereas, mean age of normal subjects was 20.81 ± 1.58 years and there was no statistical difference in mean age between patients with color blindness and normal subjects (P=0.205), as shown in figure 4-3. Mean age of patients with protan (red color) blindness was 20.25 ± 1.26 years and that of patients with deutan (green color) blindness was 21.73 ± 1.68 years and there was no statistical difference in mean age between the two groups (P = 0.136), as shown in figure 4-4.

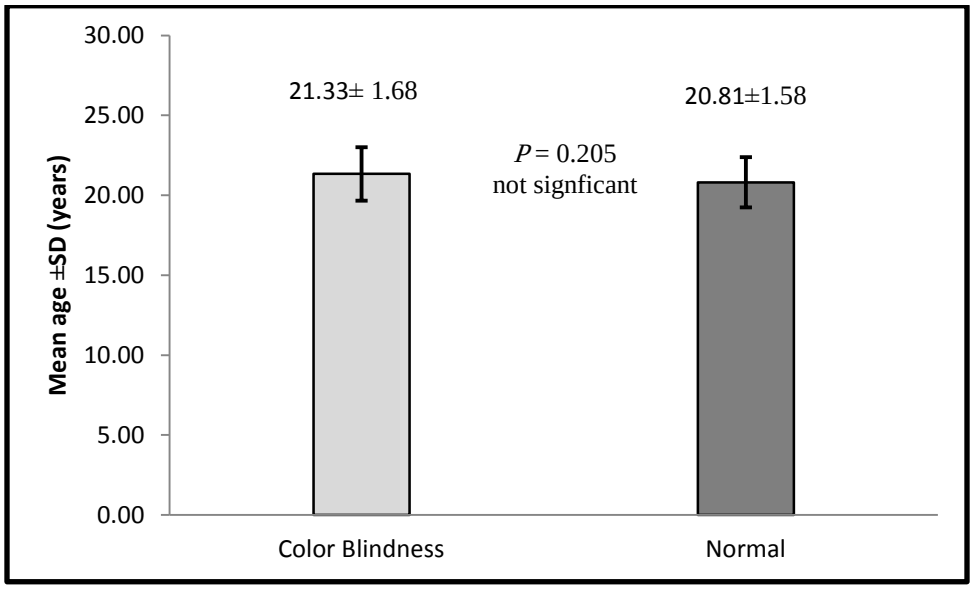


Figure 4-3: Bar chart showing mean age in patients with color blindness in comparison to normal subjects

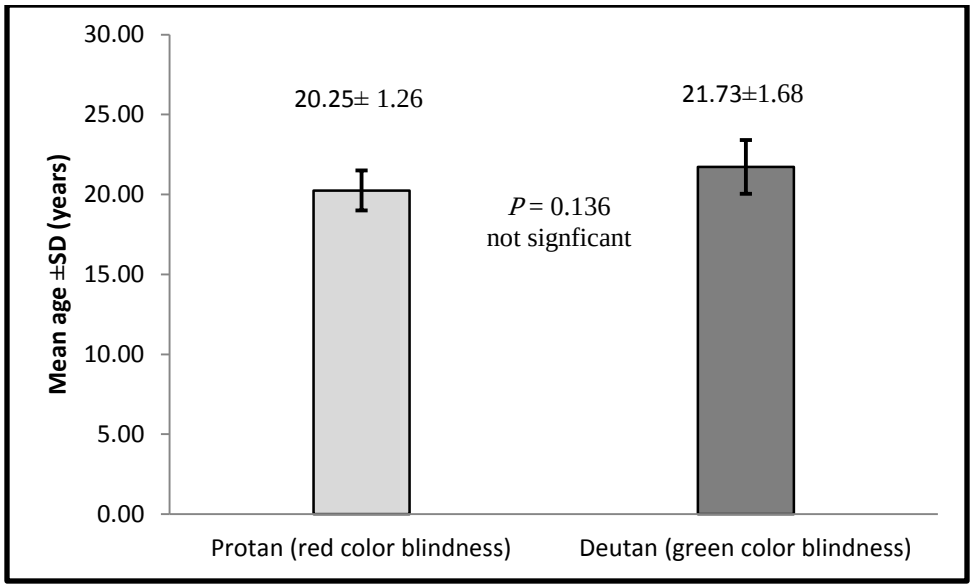


Figure 4-4: Bar chart showing mean age in patients with protan (red color blindness) versus patients with deutan (green color blindness)

4.4 Association between gender and color blindness

Out of all patients with color blindness, 13 were male patients accounting for 5.2% out of all male participants and 2 were female patients accounting for 0.4% out of all female participants. The difference statistically was highly significance (P<0.001) and the risk of having color blindness was 15.23 in male subjects in comparison with female subjects with a 95% confidence interval of 3.41 - 68.01. On the other hand, patients with protan (red color) blindness included 3 male and 1 female subjects accounting for 1.2 % and 0.2% out of all male and female participants, respectively, the difference was statistically not significant (*P*=0.171); however, the risk of having protan color blindness in male subjects was 6.67 in comparison with female subjects with a confidence interval of 0.70 - 65.30. Moreover, patients with deutan (green color) blindness included 10 male and 1 female subjects accounting for 4.0 % and 0.2% out of all male and female participants, respectively, the difference was statistically highly significant

(*P*<0.001); the risk of having deutan color blindness in male subjects was 23.18 in comparison with female subjects with a confidence interval of 2.95 - 182.10.

Table 4-4: Association between gender and color blindness

Color blindness	Male *n* = 252	Female *n* = 562	*P**	Odds Ratio	95% CI
All, *n* (%)	13 (5.2)	2 (0.4)	<0.001	15.23	3.41 - 68.01
Protan, *n* (%)	3 (1.2)	1 (0.2)	0.171	6.76	0.70 - 65.30
Deutan, *n* (%)	10 (4.0)	1 (0.2)	<0.001	23.18	2.95 - 182.10

n: number of cases; *Chi-Square after Yates correction for continuity; CI: confidence interval

4.5 Association between CVD and parent relationship

We found that 3.3% of cases of CVD have closed relationship parent. 1.2% have no close relationship parent but this difference is not statistically significant (p. value = 0.088)

Table: Association between color blindness and parent whether relative or not

Parent, relative	Total	Positive test	Negative test	χ^2	*P**
Yes	244	8 (3.3%)	236 (96.7%)	2.919	0.088 Not significant
No	570	7 (1.2%)	563 (98.8%)		
Total	814	15 (1.8%)	799 (98.2%)		

*Yates corrected Chi-Square test for continuity

Chapter Five
Discussion

Chapter five

5- 1- Discussion

CVD is one of the common visual defects which is related to visual performance that is under looked most of the time.

CVD is either congenital or acquired. Acquired type is caused by many causes like trauma or disease or chemical agent or drugs as mentioned previously in the review of literatures.

While the congenital type which is mostly inherited as X linked autosomal recessive disorder ; genetic cause is the common cause of CVD about 8% of Caucasian males are born with same degree of CVD. The females are typically just carriers of CVD gene though about 0.5% of females have CVD.[2]

In our study which is designed to find the prevalence of CVD among sample of student in the medical collages group (medical college , college of pharmacy and nursing college) at university of AL-Qadissiyah.

We found that male also affected more than female ; out of all student participate in the study (814) student 15 student are color blind; 13 of them were males student accounting for 5.2% out of all male participants (252) and 2 were females student accounting for 0.4% out of all females participants (562).

Studying the other researches result for CVD prevalence throughout the world shows that it is 0.8 – 9.3% among males and 0.4 – 3.2% among females. [41]

Many other studies done in Iraq showed result near to our result for example:- prevalence of CVD among the student in Erbil city of 8.47% in male and 1.37% in the females [41].

Among adult males in Baghdad were 6.75%; 1005 males were enrolled in the study,72 males were color vision deficient [42]. Study done in Shekhan city in AL-Duhok province, Kurdistan Region in Iraq show prevalence of 6.36% in male and 0.84% of female of high school student [43].

Another study done at AL-Diwaniah city AL-Qadissiyah province for prevalence of congenital red- green CVD among medical student and medical personal in AL-Diwaniah teaching hospital show 4.8% prevalence among male and 1% among female [44]

Other studies in the neighboring countries show the prevalence of CVD either higher or lower than our study for example a study done in young Turkish men show prevalence of 7.3%, 941 healthy male personnel of Turkish army were tested for congenital red – green color blindness by using Ishihara.[15]

In Saudi Arabia 2.9% among Arab boys from Riyad by using Ishihara and the D15 test . [45] And 4.7% in male in a study with 1000 male participant. [46] And among female were 0.35% ;7467 females tested by Ishihara and Farnsworth D15 test 26 were CVD ,16 deutan defect 10 protan defect [47]

In Iran many studies were done to look for the prevalence of CVD in different cities Mashhad, Qazvin and Tehran . The prevalence were in Mashhad 15.85% for male and 12.96% female; 2628 individual participated in the study Farnsworth D. 15 test have been used.[48]

in Qazvin 3.49% of the total population had CVD 2.56% male and 0.93% were female; 1853 individuals with age 10 – 25 years old were enrolled in the study.[49]In Tehran 8.18%; 2058 students 12- 14 years old 1136 males, 922 females all examined with Ishihara 93 males (8.18%) 4 females deutan defect 73 cases 24

protan defect,[50] while a study in Jordan showed the prevalence as 8.72% in males, 0.33% in female out of 1418 university students included in the study.[51]

Study for CVD in European countries show in a Denmark male were 8.7% while in Greek males were 7.95%. [52]

The congenital type of CVD is usually permanent and mostly red – green defect (protan defect and deutan defect) which present the Highest incidence in the general population while blue – yellow CVD (tritan defect) is relatively rare [14]

The color vision blind patient will not just confuse red and green only because the peak of sensitivity of red and green cone cells (cone cells present in the center of the retina responsible for color vision) is very close to each other so those person will be unable to discriminate any color which contain red or green for example:- they will see purple as blue because they cannot perceive the red part of the light spectrum which is added to blue to form the color purple thus all reds, greens, oranges, browns, purples, blues and grays will be impossible to identify precisely [55]

In our study we found that deutan CVD (green CVD) is more than protan CVD (red CVD); (11)case from the total student affected by CVD which are (15) student (10) male and (1) female subject accounting for 4.0% and 0.2% out of all male and female participants, respectively while protan CVD included 3 cases male and 1 case female student accounting for 1.2% and 0.2% out of all male and female participant respectively. Many other studies done in different part in Iraq showed the deutan CVD more than protan CVD as in Baghdad deutan were 53 (72.23%) While Protan defect were 19 (26.39%)[42]in Erbil city deutan defect more than protan defect [41]

A study done in AL-Duhok province showed 14 deutan CVD and 7 cases protan CVD[43] other study in AL-Diwaniah city showed 8 cases deutan defect and 8 cases protan defect . [44]

When we compare our findings with other researches in neighboring countries most of them show that also deutan defect more than other types for example in Jordan 12 cases of deutan and 7 cases of protan defect . [51] In Turkish show 5.1% protan and 2.23% deutan and this differe than our result. [45]

The results of similar studies done in other countries: in Nepal; 16 cases deutan and 3 case protan defect , 964 participants ,474 boys and 490 girls age 10-19 years 18 boys (3.8%) none of girl color vision deficient.[56]At Al- Philippine 78.9% were deutan defect a total sample of 1,258 male high school students aged 12-16 years examined by Ishihara and Farnsworth D15 .[11]

In a study done in India; a total of 1028 healthy subject (6-15 years old) were enrolled in the study CVD were determined by using the Ishihara test the prevalence of CVD ranged from 5,26% to11.36% among males and 0.00% to 3.03% among female, 7.9% deutan and 3.22% protan[54]

The cause of this classification of CVD as protan and deutan that at first it is the most common CVD[14]. The second cause is that we use only Ishihara plate for testing the CVD which can only use for red – green color blindness not blue – yellow color blindness, also it is simple and popular. Ishihara test has the mean sensitivity of 96% it cannot used for blue – yellow CVD (Tritan). [53]

Another part of our study was if there is a relation between CVD and closed parent relationship. We considered only 2nd degree as positive and any other relationship as negative we found that 3.3% was positive 1.2% was negative (have no close parent relationship) but this difference is not statistically significant p.

value = 0.088 and this is the same in the study done in Duhok city which was also not significant. [43]

Although this disease can cause marked limitation and disability in the patient life and usually affect a considerable percentage of people depending on the population and it is characteristic. [56]

What's very important to be mentioned is that only very minority from our test positive patient were aware of their disease this rise a question which is to which extent it may affect their life?

The color blindness prevalence in the public needs extensive studies and the awareness of its prevalence is so effective on the social and individual activities.

Finally the limitation in this study were the small size sample and the number of females student in the medical collages group were more than male.

The other limitation is the use of Ishihara test plates only so that we could not look for the other type of CVD rather than red – green or specific red – green differentiation.

5-2 -Conclusion

The prevalence of CVD is 1.8% in total sample of student with a prevalence of 5.2% in male and 0.4% for female student which is similar to other worldwide studies.

The Deutans CVD were more than the protans CVD deutan 4% in male and 0.2% in females

While protan 1.2% in male and 0.2% in female also similar to the other studies in the world.

Also we found that there is no significant relationship between CVD and the degree of close relationship of the parent.

5-3- Recommendation

1. As the CVD is an important visual disorder which can affect the performance and learning ability in many jobs and life activities also as the CVD is invisible disability so routine screening and planning for examination for abnormal color visualization method in pre-school test and education of teachers and families about the ways of how to deal with the affected child this may be very important for humanizing feature and performance of child with this vision abnormality.
2. Further studies should entail larger sample and a randomized choice to generalize the result
3. Also to look for other types of color vision deficiency using test other than Ishihara like Richmond HRR 2002 and Farnsworth D 15.

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Appendix

Questionnaire to the participant in the study

1- Name

2- Age

3- Gender

4- Occupation

5- Address

6- Marital status

7- Medical history

8- Surgical history

9- Ocular history

10- is the father or mother affected by the color blindness ?

11- is any one of brothers or sister affected by the color blindness?

12- are father and mother are relative?

13- The result of visual acuity

14- The result of color vision test

نموذج الاسئلة الموجة للمشاركين في الدراسة

١- الاسم

٢- العمر

٣- الجنس

٤- المهنة

٥- عنوان السكن

٦- الحالة الزوجية

٧- التاريخ المرضي

٨- التاريخ الجراحي

٩- التاريخ الخاص بالعيون

١٠- هل الام او الاب مصاب بمرض عمى الالوان

١١- أي احد من الاخوة او الاخوات مصاب بالمرض

١٢- هل الام او الاب اقارب (ابناء عم – ابناء خال)

١٣- نتيجة فحص قوة البصر

١٤- نتيجة فحص عمى الالوان

الخلاصة

خلفية البحث

مرض عمى الالوان الوراثي هو مرض وراثي مهم يتوارث كصفة وراثية متنحية متصلة بالجين نوع X ويتمثل بعدم القدرة على تصور وتمييز الالوان.

هدف البحث

لمعرفة معدل انتشار المرض في عينة من طلاب كليات المجموعة الطبية (الطب والصيدلة وكلية التمريض) في جامعة القادسية.

الطرق

اجريت هذه الدراسة في مدينة الديوانية خلال الفترة من شهر نيسان ٢٠١٨ ولغاية شهر حزيران ٢٠١٨ المشاركون في الدراسة هم طلاب من كليات المجموعة الطبية ٨١٤ طالب منهم ٢٥٢ ذكور و ٥٦٢ اناث وبمعدل عمر ١٨ – ٢٤ سنة كل المشاركين فحصوا بواسطة فحص الايشهارا.

النتائج

معدل عمى الالوان في الرجال كان ٥.٢% والنساء ٠.٤% ونسبة المصابين بعمى الالوان للون الاخضر اكثر من اللون الاحمر كذلك لا توجد علاقة بين عمى الالوان ودرجة القرابة بين الوالدين.

الاستنتاجات

مرض عمى الالوان مرض شائع ومعدل انتشاره ٥.٢% الذكور و ٤.٢% الاناث والمعدل العام ١.٨%.

إقرار المشرف

أني الأستاذة الدكتورة فرقان مجيد حميد المشرفة على رسالة طالبة الدبلوم العالي (المعادل للماجستير) هيفاء حسين جبار ، قد أطلعت على رسالة الطالبة المذكورة والتي أنجزت تحت أشرافي ،أقر وأؤيد صلاحيتها للمناقشة لاستيفائها كافة المتطلبات العلمية لدرجة الدبلوم العالي

التوقيع

المشرف: الأستاذة الدكتورة فرقان مجيد حميد

مصادقة

أني رئيس فرع طب الأسرة والمجتمع في كلية الطب /جامعة القادسية ،أصادق على إقرار المشرف على رسالة طالبة الدبلوم العالي (المعادل للماجستير) هيفاء حسين جبار وأعتبر الرسالة صالحة للمناقشة من قبل اللجنة الممتحنة لهذا الغرض

التوقيع

م.د.علي عبد الحسين موسى

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وزارة التعليم العالي والبحث العلمي
جامعة القادسية
كلية الطب
قسم طب المجتمع والاسرة

معدل انتشار مرض عمى الالوان الوراثي في عينة من طلاب كليات المجموعة الطبية في جامعة القادسية

دراسة مقدمة للمجلس العلمي في كلية الطب / جامعة القادسية كجزء من متطلبات نيل شهادة الدبلوم العالي في طب الاسرة

اعداد

هيفاء حسين جبار

بكالوريوس طب وجراحة عامة

اشراف

الاستاذ الدكتورة

فرقان مجيد حميد

المجلس العلمي لطب العيون

جامعة القادسية

كلية الطب

١٤٣٩هـ ٢٠١٨م

رقم الايداع في المكتبة الوطنية ( )