# Evaluation of Lipid Profile in Patients with Cherry Angioma: A Case–Control Study in Iraqi Patients

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## Abstract

**Background:** Cherry angioma (CA) is a common lesion among adults and elderly people. Both CA and dyslipidemia have vital common areas, such as their effect on elderly people at a certain age and their interaction with the vascular system. Therefore, we have found it to be an interesting topic for our research to investigate the connection between CA and dyslipidemia. **Objectives:** The motivation of this project is to investigate the association between CA and dyslipidemia among a sample of Iraqi patients. **Materials and Methods:** A total number of 104 Iraqi people were selected (39 male and 65 female); 52 of them were affected with CA, and the other 52 were healthy controls. For the purpose of assessing lipid profile, we used Beckman Coulter/Olympus—AU480 Chemistry Analyser was used in measuring fasting serum lipid profile parameters, including triglyceride (TG), total cholesterol (Tch), high-density lipoprotein-cholesterol (HDL-C). **Results:** The results showed a significant difference between patients with CA and control subjects in TG, CHOL, and LDL. The mean levels of the TG, total CHOL, and LDL were significantly higher in cases with CA compared to a group of controls, in which the differences were significant for TG, total CHOL, and LDL ($P$-value = 0.000). There was no significant difference between the two groups in the mean level of HDL ($P$-value = 0.059). ADditionally, there was no significant difference in levels of lipid value regarding ages and sex distribution between patients with CA and controls ($P$-value > 0.05). **Conclusion:** Based on its outcomes, the study proposed that dyslipidemia could play a vital role in the pathogenesis of CA lesions. Hence, dyslipidemia treatments may represent an effective means for CA therapy.

**Keywords:** Cherry angioma, dyslipidemia

## Introduction

Cherry angioma (CA), also known as Campbell de Morgan spots or Cherry hemangiomas, or senile angioma, is a benign vascular lesion of the skin. CA ranged in color from bright red to purple, and dark blue to black may occur as well. It often appears as small, unruffled, curving, circuitous, or oval papules or macules that are shaped in size from a pinpoint to one-quarter of an inch in diameter.[1,2] CA is a capillary hemangioma composed of an amalgamation of newly formed capillaries within the papillary dermis.[3]

In general, CA appears during or after the third decade, and then the number of lesions increases with the age curve.[4] Statically, it was reported that developing CA exists in 5% of adolescents, 50% of adults, and 50%–75% of adults over 75 years of age. On this basis, the high incidence of Charry angioma in old age indicates that their appearance is an age-related degenerative phenomenon.[4,5]

Hormonal factors may also participate in the development of CA as a result of an increasing level of a certain hormone in the subject's body.[6] It has been noticed that CA s appear increasingly during pregnancy, and that could be sophisticated in the postpartum duration.[7]

Dyslipidemia is a particular alteration of lipids in human blood that results from a deviation of one or more plasma

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lipoprotein traits throughout a biochemical threshold and is usually determined by the age and sex value of the patient.[8] Aging is usually one of the dyslipidaemias' reasons. It has been noticed that triglyceride (TG) levels increase with age across all age groups. That could be more manifested in men aged 50–59 years and in women aged 60–69 years.[9] Furthermore, small dense LDL-c and apolipoprotein B have also increased throughout the same age level, while HDL-c has not been approved to be vary with age.[10] Pregnancy has been identified as one of the affecting factors in dyslipidemia.[11] During the pregnancy period, women manifest an increased level of lipid profile, which includes total cholesterol (Tch) and levels of TG as the gestational grows.[11,12] TG concentrations were higher in women with gestational diabetes mellitus compared to those with nongestational diabetes mellitus.[13] Obesity has an approved association with dyslipidemia. Tumor necrosis factor secreted by keratinocytes in psoriatic patients also contributes to obesity by inhibiting glucose transporters and suppressing insulin receptor activity.[14] It is worth mentioning that dyslipidemia is a common metabolic disorder that reflects the deposition of lipids into vessel walls in a way that obstructs its normal functions and results in hypoxia.[15]

This research aims to investigate the association of lipid profile disorder with CA among Iraqi patients.

## Materials and Methods

The conduction of this case–control study had been done at the Department of Dermatology and Venereology—Al-Sadr Medical City in Najaf City—Iraq, and Baghdad laboratory in Al-Diwaniya City during the period from January 2020 to January 2021.

### Patients and control groups

A total number of 104 Iraqi people were selected; 52 of them had CA, and the other 52 were healthy controls. All patients with CA were recognized clinically by the dermatologist.

### Exclusion criteria

1. Pregnancy or lactation,
2. Alcoholism,
3. Smoking,
4. Hypertension,
5. Diabetes,
6. Thyroid dysfunction,
7. Underlying malignancy,
8. Eruptive form of CA,
9. Familial dyslipidemia,
10. Chronic inflammatory skin diseases such as psoriasis or lichen planus.

### Methods

All patients were subjected to:

I. Full history taking: A complete history was taken from each individual regarding age, sex, the commencement of disease and its duration, aggravating factors, and family history of the same situation.
II. Laboratory tests.

#### *Sample collection*

Three milliliters of peripheral venous blood were aspirated from each patient and controlled after 12 h of fasting. The blood samples were put in a gel test tube and centrifuged at 4000–6000 r.p.m for 15 min to secure serum for TG, Tch, and high-density lipoprotein-cholesterol (HDL-C). Furthermore, the rest of the blood samples were stored at -02°C for examining insulin levels later.

#### *Assessment of lipid profile levels*

Beckman Coulter/Olympus—AU480 Chemistry Analyzer was used in measuring fasting serum lipid profile parameters, including TG, Tch, and HDL-C. The tests were performed according to the normal values that applied to the standards of Al-Sadr Medical City Laboratory, which could be determined as follows:

- Tch: 50–199 mg/dl.
- TG: 0–149 mg/dl.
- High-density lipoprotein: 40–60 mg/dl.
- Low–density lipoprotein: 0–100 mg/dl.

### Statistical analysis

Statistical analysis has been done by utilizing SPSS (Version 20.0, IBM Co., Chicago, IL), in which we use mean, numbers, percentages, and standard deviation as descriptive statistics. For analysis, we use the chi-square test for categorical variables and the independent sample t-test for numerical variables. On this basis, $P$ value ≤ 0.05 is considered significant.

### Ethical approval

Ethical approval has been provided by the Scientific Council of Dermatology and Venereology—Arab Board of Medical Specializations. All participants in this study have been enlightened with signed consent. The date for the approval was January 2020 to January 2021.

## Results

In this study, a total of 104 males and females were included. The participant individuals consisted of two groups; the first group numbers ($n$ = 52) were complaining of CA, and the second group numbers ($n$ = 52) were healthy controls. The ages of both groups ranged between 30 and 69 years. About 39 participants were males and 65 were

females. Mean levels of lipid value regarding the periods of age of both patients and controls were not significantly different among groups, as shown in Table 1. Mean levels of lipids regarding sex distribution of both patients and control were not significantly different between males and females, as shown in Table 2. Thirty patients had less than five lesions, 13 patients had 5–10 lesions, and nine patients had more than 10 lesions, as shown in Table 3 and Figure 1.

The study found a notable difference between patients who had less than five lesions and patients who had more than 10 lesions. The percentage of male patients who had > 10 lesions was greater than the percentage of female patients, while the percentage of male patients who had > 5 was less than the percentage of female patients, as shown in Figure 2.

The mean levels of the TG, total CHOL, and LDL were significantly higher in cases with CA compared to a group of controls, in which the differences were significant for TG, total CHOL, and LDL (*P*-value = 0.000). No significant difference between patients and controls in HDL level (*P*-value = 0.059), which has been shown

**Table 1: Mean levels of TG, total CHOL, LDL, and HDL regarding the period of age of both patients and controls**

| Lipid profile (mg/dl) | Age (years) | *N* | Mean level of lipid profile | SD | *P*-value* |
|---|---|---|---|---|---|
| TG | 30–39 | 26 | 190.77 | 120.056 | 0.349 |
| | 40–49 | 26 | 231.73 | 153.564 | |
| | 50–59 | 26 | 259.54 | 179.273 | |
| | 60–69 | 26 | 265.38 | 156.536 | |
| | Total | 104 | 234.61 | 154.059 | |
| CHOL | 30–39 | 26 | 219.81 | 142.032 | 0.337 |
| | 40–49 | 26 | 223.46 | 165.228 | |
| | 50–59 | 26 | 285.46 | 144.836 | |
| | 6–69 | 26 | 251.92 | 127.727 | |
| | Total | 104 | 245.16 | 145.865 | |
| LDL | 30–39 | 26 | 123.46 | 31.520 | 0.199 |
| | 40–49 | 26 | 126.19 | 32.283 | |
| | 50–59 | 26 | 145.85 | 64.270 | |
| | 60–69 | 26 | 141.08 | 42.277 | |
| | Total | 104 | 134.14 | 44.967 | |
| HDL | 30–39 | 26 | 62.85 | 24.891 | 0.214 |
| | 40–49 | 26 | 56.00 | 11.842 | |
| | 50–59 | 26 | 55.00 | 10.811 | |
| | 60–69 | 26 | 54.73 | 11.470 | |
| | Total | 104 | 57.14 | 15.997 | |

**P*-value (<0.05) significant

**Table 2: Mean levels of TG, total CHOL, LDL, and HDL regarding the sex of both patients and controls**

| Lipid profile (mg/dl) | Gender | *N* | Mean | SD | *P*-value* |
|---|---|---|---|---|---|
| TG | Male | 39 | 231.67 | 160.490 | 0.874 |
| | Female | 65 | 236.37 | 151.312 | |
| CHOL | Male | 39 | 253.38 | 159.789 | 0.396 |
| | Female | 65 | 240.23 | 137.900 | |
| LDL | Male | 39 | 134.18 | 41.348 | 0.974 |
| | Female | 65 | 134.12 | 47.318 | |
| HDL | Male | 39 | 57.28 | 22.122 | 0.106 |
| | Female | 65 | 57.06 | 11.011 | |

**P*-value (< 0.05) significant

**Table 3: The number of lesions (cherry angioma) and mean age (years) in patients' group**

| Subgroup of patients | Number of patients | Mean age (years) | SD |
|---|---|---|---|
| A. <5 lesions | 30 | 46.9 | ±1.949 |
| B. 5–10 lesions | 13 | 48.8 | ±0.002 |
| C. >10 lesions | 9 | 58.2 | ±3.628 |

in Table 4. The mean level of TG among patients and controls increased gradually with age. The curve started to increase in the age group 40–49 and continued to increase till the highest level in the age group 59–69, as shown in Figure 3. The mean level of CHOL among patients and controls slightly increased in the age group 40–49, then gradually increased to be the highest level in age 58–60. Then, the curve gradually declined in ages 61–69, as shown in Figure 4. The mean level of LDL among patients and controls slightly increased in age groups 40–49, then gradually increased to be the highest level in ages 58–59. The curve then gradually declined in the age group 60–69, as shown in Figure 5. The mean level of HDL among patients and controls is the highest level in the 40–42 age group. The curve then declined with age to be at the lowest level in the age group 60–69, as shown in Figure 6.

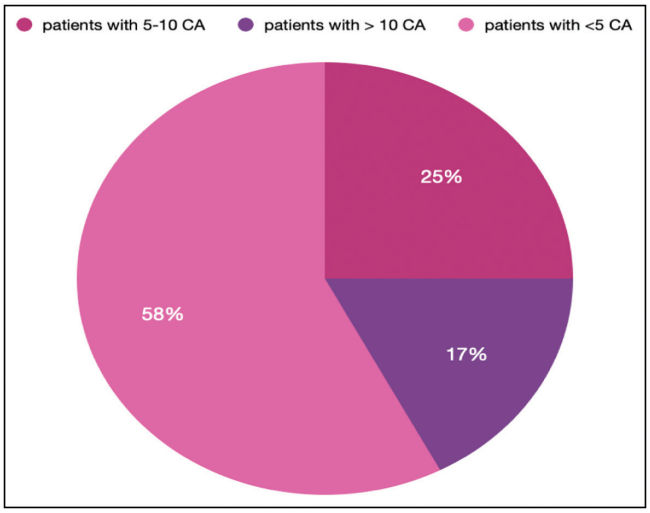


**Figure 1:** Number of lesions (CA) among patients

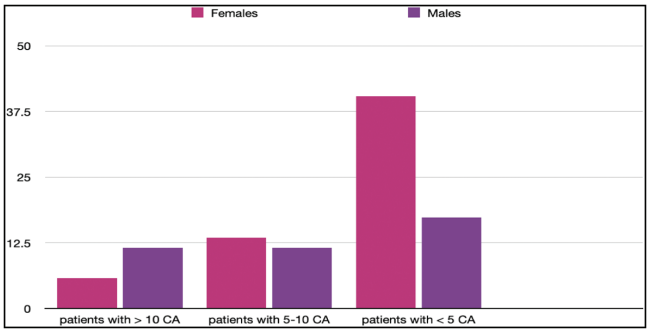


**Figure 2:** Male and female distribution in relation to percentage of lesions (CA). (1) 30–39 years, (2) 40–49 years, (3) 50–59 years, (4) 60–69 years

## Discussion

CA is a benign vascular lesion of the skin. It often appears on the torso and upper extremities of the body as one or multiple spots, but it can be found on other parts.[1] CA takes the shape of small, unruffled, curving, circuitous, or oval papules or macules that are shaped in size from a pinpoint to one-quarter of an inch in diameter.[2] CA is a common lesion among adults and elderly people, and it notably rises with the age graph, particularly after the third decade of age. In fact, the mechanism of endothelial proliferation in CA indicates that decreased mir-424 expression and increased levels of MEK1 or cyclin E1 could be the main cause of abnormal cell proliferation in these tumors.[16]

Specialists agreed that the etiologies of CA are categorically unidentified.[17] However, CA's association with dyslipidemia has not been specifically approved in previous studies.

Pathologically, both CA and dyslipidemia may be sourced from the same pathogenesis process. This could be clearer when mast cell mediators trigger elements that collaborate in neovascularization and increase the number of mast cells simultaneously. As a result, this process could participate effectively in both generating CAs and developing dyslipidemia concurrently.[18]

For CA increases with age curve, increased levels of serum lipids in the elderly could be as an inducer element to release chemokines and cytokines, which may cause endothelial cell proliferation and CAs.[19,20] Studies have found a clear association between hypercholesterolemia and endothelial cell dysfunction, which might result from toxic products obtained from the disintegration of lipoproteins.[21] On this basis, the ordinary pathogenic

**Table 4: Mean level of TG, total CHOL, LDL, and HDL in patients and controls**

| Lipid profile (mg/dl) | Group | *N* | Mean | SD | *P*-value* |
|---|---|---|---|---|---|
| TG | Control | 52 | 162.29 | 87.521 | 0.000 |
| | Patient | 52 | 306.92 | 172.073 | |
| CHOL | Control | 52 | 188.87 | 100.606 | 0.000 |
| | Patient | 52 | 301.46 | 162.437 | |
| LDL | Control | 52 | 114.48 | 19.073 | 0.000 |
| | Patient | 52 | 153.81 | 54.142 | |
| HDL | Control | 52 | 59.33 | 9.960 | 0.059 |
| | Patient | 52 | 54.96 | 20.196 | |

*$P$-value (<0.05) significant

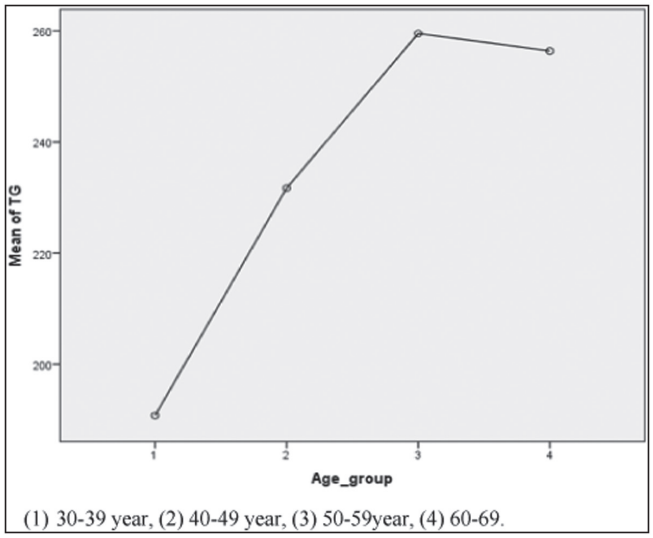


**Figure 3:** Mean of TG (mg/dl) among cases and controls. (1) 30–39 years, (2) 40–49 years, (3) 50–59 years, (4) 60–69 years.

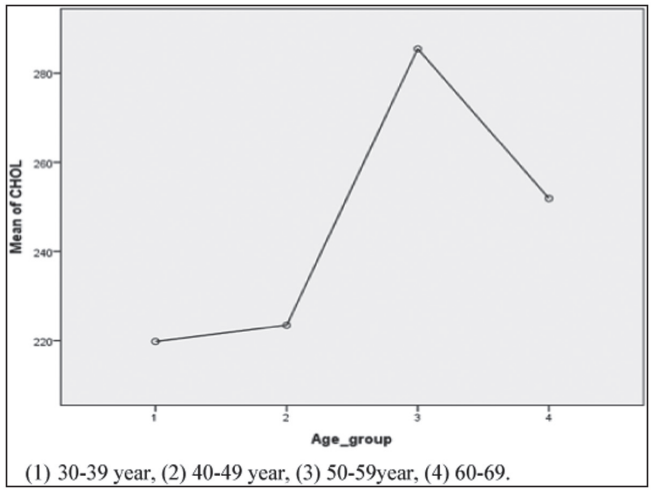


**Figure 4:** Mean of CHOL (mg/dl) among cases and controls. (1) 30–39 years, (2) 40–49 years, (3) 50–59 years, (4) 60–69 years.

process could be implicated in CA and hyperlipidemia; for instance, the engagement of insulin-like growth factors (IGF) to their receptors catalyzes the proliferation of endothelial cells and dyslipidemia.[22] Statistically, it has been mentioned that CA lesions are prevalent in patients who suffer from type 2 diabetes mellitus and have extra IGF,[23,24] while this fact has not been approved in obese patients.[25]

This study tried to evaluate the association between CA and lipid profile in patients who attended the dermatology outpatient clinic at Al-Sader Medical City in Najaf City—Iraq. Based on the inclusion criteria of selected patients, our study lasted for one academic year (2020–2021), and a total of 104 Iraqis, including 52 patients with CA and 52 other controls, were selected.

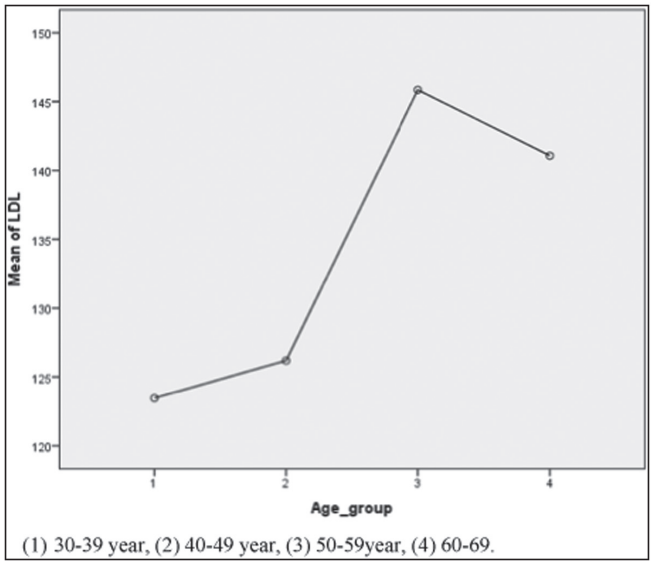


**Figure 5:** Mean of LDL (mg/dl) among cases and controls. (1) 30–39 years, (2) 40–49 years, (3) 50–59 years, (4) 60–69 years.

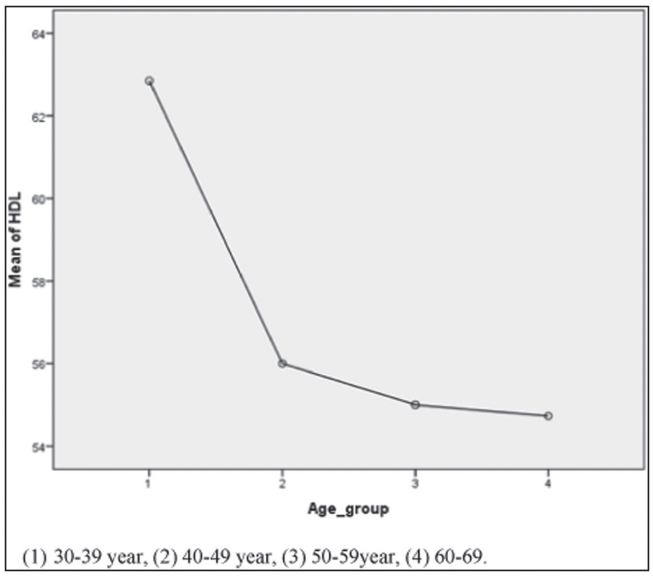


**Figure 6:** Mean of HDL (mg/dl) among cases and controls

On this basis, the study had shown a significant association between CA and dyslipidemia. The relation with hyperlipidemia might not be confirmed as a confined cause for developing CAs, but it definitely had been reported in many cases as an undeniable association. In fact, the risk of developing CA maximizes with the presence of multiple factors such as diabetes, pregnancy, HIV, or any other immunosuppressive situations.

The study manifested a noticeable association between CA and dyslipidemia. This may be consistent with other studies that found most affected individuals with CA had dyslipidemia. That means dyslipidemia has a remarkable role in the development of CA in these subjects. On this basis, dyslipidemia therapy could represent a valuable means for the objective of CA treatment.

There was a frequent occurrence of hypertriglyceridemia and hypercholesterolemia, while there were low levels of HDL together with higher levels of LDL in this study compared with the evaluated prevalence of hyperlipidemia in Iraqi patients. It has been mentioned that dyslipidemia is a common disease in Iraq, and it is often higher than its occurrence in countries such as Saudi Arabia, Lebanon, and Turkey.[26]

Referring to the research statistics, the results indicate that in the older age group the lipid changes are more, so the number of lesions more.

The results of this research were comparable to that of another study done by Abbas Darjani *et al.* in 2018 in Guilan City in Iran, on 122 cases with CA and 122 healthy controls. The mentioned study suggested that dyslipidemia might not play a major role in the pathogenesis of CA.[27] However, it confirmed a significant association between CA and dyslipidemia in most of the study fields, which generally converges with our findings.

Shah's study[28] on 100 nondiabetic persons and 100 diabetic persons found a clear association between diabetes and CA. The cause of this relationship, as they mentioned, is related to the vascular and cellular levels. In the same way, Arora *et al.*[29] studied 125 diabetics and 125 nondiabetic control; they highlighted that CA was associated with diabetes, especially the uncontrolled type of diabetes. The pathogenesis of these lesions, as they noticed, was linked to abnormal carbohydrate metabolism, other altered metabolic pathways, neuron degeneration, microangiopathy, impaired host mechanisms, and atherosclerosis.

James and Paterson,[5] in their study named "Vascular Tumors," suggested that CAs existence is an age-related degenerative process. These lesions histopathologically manifest to be the dilated and interconnected portion of venous capillaries and postcapillary venules in the dermal papillae.

Both of these studies had mentioned lipid profile disorder as a secondary condition that may cause CA. Their studies discussed the impact of its subjects on blood vessels regardless of their participation with dyslipidemia and the mechanism of biologically developing CAs.

In comparison, our study suggested that CA could be a sign of dyslipidemia, which is considered a primary condition for many other health conditions, especially those related to cardiovascular morbidity and mortality. On this basis, the association between CA and dyslipidemia might be considered a significant risk factor that requires more medical attention and further research.

This study faced various restrictions as it covered a relatively small number of subjects, which left it with a perspective of not representing the whole Iraqi population. The research was also conducted in a specific area (Al-Najaf Province), a particular place (Al-Sadr Medical City) and a limited time that was confined to a period of one year. ADditionally, the comparative studies were, unfortunately, rare at both national and international levels, which required us to double our efforts in fulfilling the research requirements.

## Conclusions

Our study showed a noticeable association between CA and dyslipidemia, which means that dyslipidemia has a role in the development of CA. On this basis, dyslipidemia therapy could represent a valuable way for the purpose of CA treatment.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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