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Panacea Journal of Medical Sciences

Journal homepage: https://pjms.in/

Original Research Article

Expression of telocytes in cardiovascular diseases

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Abstract

Background: Telocytes are recently discovered cells that reprogram the terminally differentiated cardiomyocytes to re-enter the cell cycle. Their activity is also altered in atherosclerotic blood vessels. Telocytes express CD117/c-kit. This study focuses on the significance of changes in the expression of telocytes in the heart and the coronary arteries in various diseases.

Materials and Methods: This is a retrospective study. Histopathology of post-mortem examination (PME) of adult and foetal hearts between March 2021 and March 2023 were reviewed. Sections from normal and diseased heart and coronary arteries were selected for immunohistochemistry with CD117/c-kit to highlight telocytes. Telocytes were counted.

Results: 62 heart (46 adult and 16 foetal) specimens were examined. All foetal and 11 normal adult hearts showed 5 and 3 telocytes per 10 fields respectively. Recent myocardial infarction (MI) seen in 10 cases showed 10 telocytes per 10 fields in the peri-infarction zone while 8 cases of old scars showed only 1 telocyte per 10 fields. Cardiac hypertrophy was seen in 22 cases. Telocytes were seen in 6 cases only, showing 1 telocytes per 10 fields. 92 coronary arteries were examined. 27 arteries were normal with 4 telocytes per 10 fields. 33 arteries showed marked atherosclerosis with 14 telocytes per 10 fields.

Conclusion: An increase in telocytes signifies their stem cell-like activity. In recent MI cases increased telocytes suggest the likelihood of cardiac tissue regeneration after an MI. In cardiac hypertrophy, telocytes increase during a continued stimulus of increased workload, however, the timing of the biopsy plays a significant role. In older lesions with no ongoing stimulus for telocytes, there is no increase observed. In atherosclerosis, telocytes are altered causing vessel wall changes. Modulation of telocytes is a novel step that may change the management of cardiovascular diseases.

Keywords: Telocytes, CD117/c-kit, Cardiovascular diseases, Atherosclerosis, Heart.

Received: 26-04-2024; **Accepted:** 28-10-2024; **Available Online**: 19-08-2025

1. Introduction

Host tissues undergo regeneration and remodelling constantly. They govern morphogenesis, cellular functionality, and molecular integrity. The structural and functional stability of these mechanisms is maintained by complex signalling pathways and systems, some of which are poorly understood and being explored extensively. Telocytes are such cells that are proactively involved in the histogenesis of tissues and are under study and experimentation for their exact role in tissue homeostasis.[1]

Telocytes are crucial component of the stem cell niche and are prime modulators in maintaining the microenvironment responsible for promoting renewal and regeneration[2,3] by orchestrating sequential events mediating cell proliferation, differentiation and de-differentiation, apoptosis, and necrosis thereby pushing mature cells to re-enter the cell cycle and reprogram them to their foetal-like state.[4] Currently, telocytes have been identified in many tissues.[3] In most sites, telocytes express CD34 and platelet-derived growth factor receptor - beta (PGDFR-β).[2] In some sites such as the heart, skeletal muscle, lung, kidney, and blood vessels they express CD117/cytokine receptor tyrosine kinase KIT (c-kit), while telocytes in the synovium, salivary glands, liver, and urinary bladder lack CD117 expression.[2]

The structure of telocytes is a characteristic small spindle-shaped or star-shaped cell with a hyperchromatic nucleus and scant cytoplasm.[5,6] They have up to 5long and

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https://doi.org/10.18231/pjms.v.15.i.2.443-449

thin cytoplasmic processes called telopodes. These are 10 to 1000 µm long and vary from 0.1 to 0.5 µm in thickness.[7,8] They have a beaded appearance forming a moniliform pattern due to alternating podomers and dilated podoms which contain mitochondria and endoplasmic reticulum. [5, 6]. Telocytes are difficult to identify on light microscopy and are routinely studied by immunohistochemistry (IHC), immunofluorescence, or electron microscopy.[7,8]

This study focuses on telocytes in the heart and the blood vessels in their normal and diseased states. The functional role of telocytes in the heart is currently under focus due to their potentially revolutionary status in tissue regeneration in cardiovascular diseases (CVD) primarily associated with ischemia.[5] Telocytes in the heart are present within cardiogenic niches and are closely related to cardiac stem cells.[9] They maintain the cardiac microenvironment by integrating several cellular pathways to re-program the terminally differentiated adult cardiomyocytes to re-enter the cell cycle making them capable of undergoing regeneration.[2]

In the blood vessels, atherosclerosis is an antecedent event for ischemic heart disease (IHD). Studies suggest that in mice, telocytes modulate the immunobiological alterations of atherosclerosis in the coronary arteries however, there is no literature on the role of telocytes in atherosclerotic vessels in human subjects. It is proposed that the modulation of telocyte activity may be a novel therapeutic target in restoring vascular integrity and function in humans.[10,11]

Telocytes remain a promising new entity in regenerative medicine with the prospect of changing the outcome of CVD.[12] However, most of the experimentation has been on animal models, and the true nature and function of telocytes in the heart and blood vessels in humans is not entirely understood[10,11,13] hence, there is a vast knowledge gap on the therapeutic potential and usage of human telocytes.

2. Materials and Methods

This is a retrospective study conducted at a teaching hospital. All adult and foetal post-mortem examinations (PME) of the heart performed between March 2021 and March 2023 were included in the study.

2.1. Case selection

Clinical details of the PME were obtained from the medical records of the hospital. Formalin-fixed paraffin-embedded (FFPE) heart tissue sections stained by haematoxylin and eosin (H&E) were retrieved from the Department of Pathology and were reviewed by light microscopy.

2.2. Microscopic examination

Details of cardiovascular disease and/or cases showing cardiovascular disease-associated pathological changes in the heart and coronary arteries were noted. Foetal cases were reviewed for histology. Immunohistochemistry (IHC) was performed to stain cardiac telocytes with CD117/c-KIT using GenomeMe ® Gene Ab ™ Monoclonal Rabbit Anti-Human Antibody (Clone IHC526). Staining was done using standard protocols for IHC. The histopathological examination (HPE)was done by two pathologists by light microscopy using a 40x objective lens (high power) with a field area of 0.255 mm^2. Telocytes were identified by their elongated morphology. Foci of maximum telocytes in the normal and diseased cardiac tissue and blood vessels were identified. Telocytes were counted in 10 consecutive fields per case. Results were recorded as telocytes counted by each pathologist per 10 fields.

2.3. Statistical analyses

Data was expressed as an average of the telocytes counted by each pathologist per 10 fields. The distribution of variables was determined by the Kolmogorov-Smirnov test. Test of significance for normally distributed data was done by independent sample two-tailed t-test, while Mann Whitney U test was used for data that was not normally distributed. A *p*-value of ≤ 0.05 was statistically significant.

All procedures were in accordance with the standard national and international ethical medical practices and with the Helsinki Declaration of 1964 and its later amendments. The study was approved by the Institutional Ethics Committee of the teaching hospital. Standard national guidelines for conducting PME were followed. This study did not require any form of intervention/modification of human subjects and no living tissues were used.

3. Results

A total of 62 heart specimens were included in the study out of which 46 were adult hearts while 16 were foetal hearts. A summary of the heart specimens is shown in **Table 1**.

3.1. Normal heart

Among the adult heart specimens, 11 were found to be histologically unremarkable. HPE of normal specimens showed the endocardium (**Figure 1**a) lined by flattened endothelial cells, the myocardium (**Figure 1**b) composed of cardiomyocytes, and the epicardium (**Figure 1**c) showing adipose tissue. CD117 highlighted 3telocytes per 10 fields in the myocardium (**Figure 1**d). No telocytes were seen in the epicardium and pericardium.

3.2. Ischemia

Ten patients showed early changes of recent myocardial infarction (MI). Gross examination showed a focal pale area of the heart muscle. HPE of the infarcted zone (**Figure 2**a) showed intensely eosinophilic wavy cardiac fibres with focal loss of nuclear details.

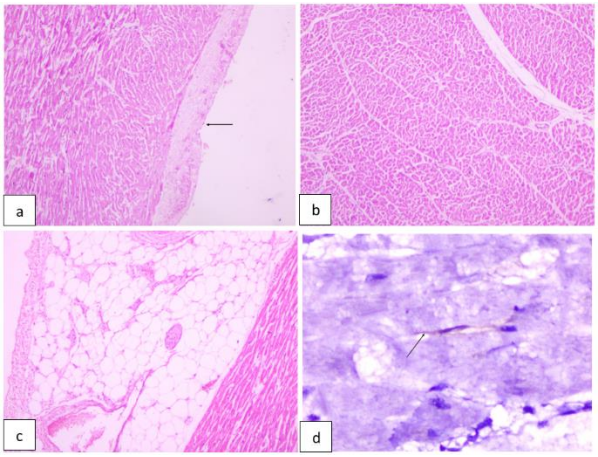

Figure 1: a): Normal adult heart endocardium lined by endothelial cells (arrow) (H&E, 40x); **b):** Normal adult heart myocardium composed of normal cardiomyocytes (H&E, 40x); **c):** Normal adult heart epicardium showing adipose tissue (H&E, 40x); **d):** Normal adult heart showing occasional scattered telocytes in the myocardium (arrow) (CD117, 400x)

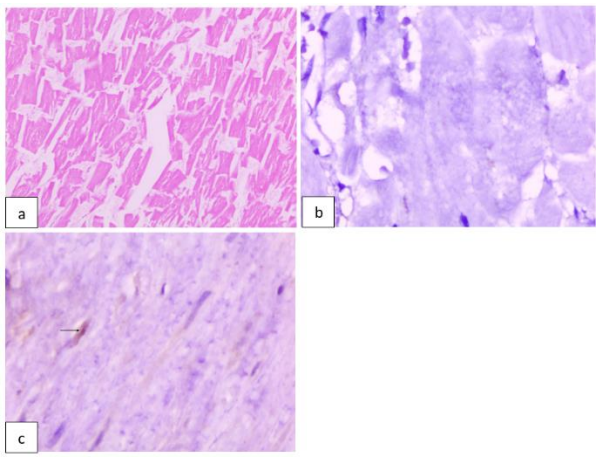

Figure 2: a): Myocardial infarction showing waviness of the cardiac fibres with intense eosinophilia of the cytoplasm and focal loss of nuclear details (H&E, 100x); **b):** Myocardial infarction showing complete loss of telocytes in the necrotic cardiomyocytes (CD117, 400x); **c):** Myocardial infarction showing telocytes (arrow) in the peri-infarct zone (CD117, 400x)

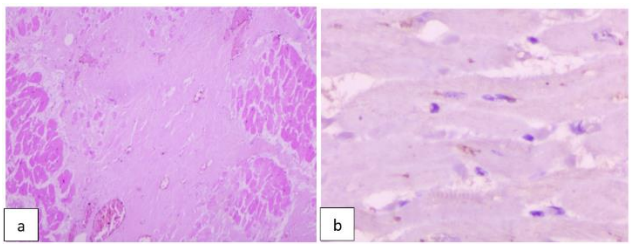

Figure 3: a): Old infarct showing focal fibrosis within the myocardium (H&E, 40x); **b):** Old infarct showing absence of telocytes in many fields (CD117, 400x)

CD117 showed no telocytes in the infarcted zone (**Figure 2**b). In the peri-infarct zone telocytes were increased up to 10 telocytes per 10 fields (**Figure 2**c). This increase in telocytes was statistically significant ($p \leq 0.05$). Eight cases showed old infarcts (**Figure 3**a) evidenced by the presence of focal fibrosis within the myocardium. In these areas, CD117 showed a marked decrease in the telocytes with many cases showing complete absence of staining for telocytes. There was only 1 telocyte per 10 fields (**Figure 3**b). This decrease in telocytes was statistically significant ($p \leq 0.05$).

3.3. Hypertrophy

Ventricular wall thickening was seen in 22 adult heart specimens. HPE showed ventricular hypertrophy (**Figure 4**a) showing myocytes arranged in disarrayed architecture with focal interstitial fibrosis. Cardiomyocytes had increased cell size with mild to moderate nuclear enlargement. CD117 showed telocytes only in 6 cases while there were no telocytes in 16 cases. This gave an average of only 1 telocyte per 10 fields (**Figure 4**b). This decrease was statistically significant ($p \leq 0.05$).

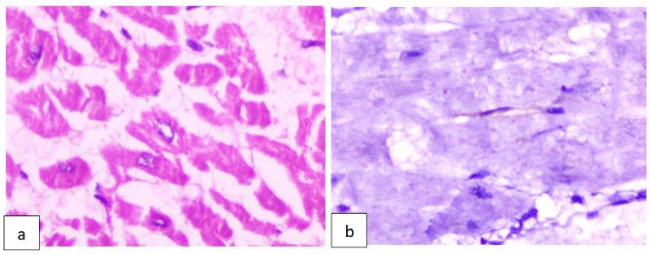

Figure 4: a): Ventricular hypertrophy showing hypertrophied myocytes with disarrayed architecture and focal interstitial fibrosis (H&E, 400x); **b):** Ventricular hypertrophy showing an occasional telocyte in the hypertrophic myocardium (CD117, 400x)

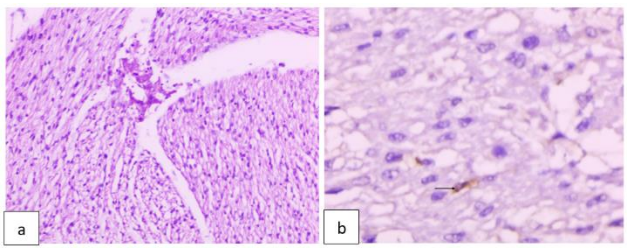

Figure 5: a): Normal foetal heart showing polygonal cardiomyocytes with a prominent central round nucleus (H&E, 40x); **b):** Normal foetal heart showing telocytes (arrow) in the developing myocardium (CD117, 400x)

Table 1: Telocytes in normal and diseased heart

	Category	Number of cases	Average telocytes in 10 fields (One field area of 0.255 mm^2)	*p*-value
Adults (*n*=46)	Normal adult	11	3	-
	Recent myocardial infarction peri - infarct zone	10	10	<0.0001
	Old infarcts - fibrotic scars	8	1	0.0021
	Hypertrophy	22	1	0.0086
Foetal heart (*n*=16)			5	<0.0001

Table 2: Telocytes in normal and diseased blood vessels

	Category	Number of cases	Average telocytes in 10 fields (One field area of 0.255 mm^2)	*p*-value
Coronary artery (*n*=92)	Normal	27	4	-
	Marked atherosclerosis	33	14	<0.0001
	Mild atherosclerosis	32	Immunohistochemistry was not done	

3.4. Foetal heart

Sixteen foetal heart specimens between 16 to 24 weeks of gestation were examined. HPE of the foetal heart (**Figure 5**a) showed polygonal cardiomyocytes with a prominent central round nucleus. There was scant intervening connective tissue. Myocyte fibre architectural organisation as seen in the adult heart was not seen in foetal histology. CD117 showed 5 telocytes per 10 fields in the myocardium (**Figure 5**b). This increase was statistically significant ($p \leq 0.05$).

A total of 92 coronary arteries (right and left) from 46 adult hearts were examined for atherosclerotic changes. A summary of the blood vessels is shown in **Table 2**.

3.5. Normal blood vessels

27 arteries were histologically unremarkable (**Figure 6**a). CD117 showed 4telocytes per 10 fields (**Figure 6**b).

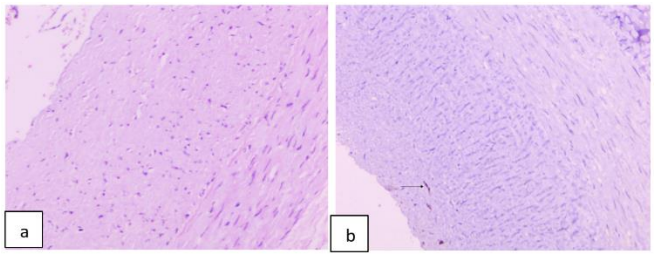

Figure 6: a): Histologically unremarkable coronary artery (H&E, 100x); **b):** Histologically unremarkable coronary artery showing scattered telocytes (arrows) in the vessel wall (CD117, 100x)

3.6. Atherosclerotic blood vessels

HPE of 33 arteries showed marked atherosclerotic changes (**Figure 7**a, b) with a fibrous cap and central necrotic core. Few areas showed the presence of cholesterol cleft and foamy macrophages. Intimal smooth muscle proliferation was present.

CD117 showed 14 telocytes per 10 fields in areas of thickened vessel wall with loss of telocytes in the necrotic foci (**Figure 7**c). This increase was statistically significant ($p \leq 0.05$). Thirty-two arteries showed mild to moderate changes of atherosclerosis. IHC was not done on these.

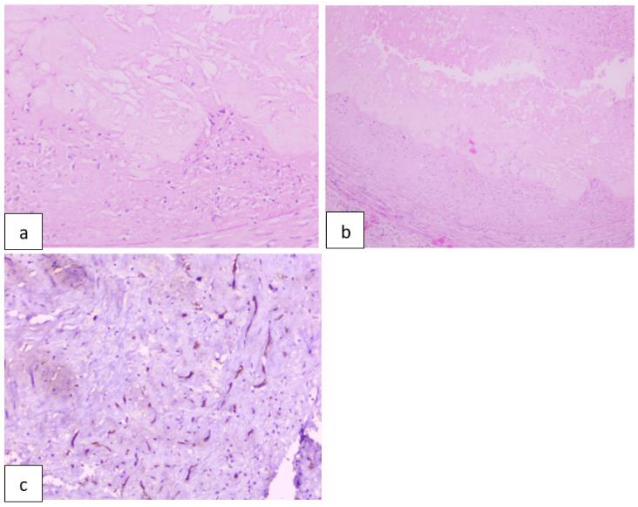

Figure 7: a): Atherosclerotic coronary artery showing necrosis with cholesterol cleft and foamy macrophages (H&E, 100x); **b):** Atherosclerotic coronary artery showing necrosis with intimal smooth muscle proliferation (H&E, 40x); **c):** Atherosclerotic coronary artery showing increased telocytes in areas of the thickened vessel wall (CD117, 400x)

4. Discussion

Telocytes were first described in the muscle layer of the gut about a century ago. These cells resembled neurons due to their cytoplasmic projections and were called *'interstitial neurons'* by neuropathologist Santiago Ramon y Cajal in 1911.[14] In 1977 these cells were re-examined by Faussone-Pellegrini et al and it was found that they were not neurons, and were renamed as *'interstitial cells of Cajal'*.[15] In 1982 Thuneberg found them to be pacemaker cells of the

gastrointestinal tract.[16] By 1996 Lecoin et al reported that these interstitial cells of Cajal had a mesenchymal origin and expressed gene encoding c-kit.[17] In 2005 Popescu et al found cells in the exocrine pancreas that resembled the interstitial cells of Cajal and named them *'interstitial Cajal-like cells'*.[12,18] Following this discovery, these cells were found in many organs and tissues including the heart, and in 2010 they were re-named as *telocytes.*[19] From 2012 onwards there have been several experiments on animal models, but only a few studies on human subjects, to determine the exact function of telocytes.[3]

4.1. Normal heart

Under physiological conditions in experimental mice, telocytes are distributed longitudinally and in cross-network patterns in the heart.[5] They are found in all three cardiac layers.[5,19] In the current study, an average of 3 telocytes per 10 fields were identified in the histologically unremarkable adult myocardium. No telocytes were identified in the epicardium and pericardium.

4.2. Ischemia

Ischemic events cause damage to the cardiac tissue leading to replacement by connective tissue scar with irreversible focal loss of cardiac contractility and function.[7,20] In experimental animal models, there is an absence of telocytes in the necrotic infarcted zone however, an increase is seen in the number of telocytes in the peri-infarct zone signifying attempted regeneration starting at the peripheries.[13,21] Human experimentation in this area of research lacks remarkably and there is very little information available on the pattern and expression of telocytes in the infarcted cardiac tissue. Even though research in experimental animal models is markedly different from human studies,[5,13] the current study showed similar results. Cases of MI showed up to 10 telocytes per 10 fields in the peri-infarct zone. There was a complete absence of telocytes in the old connective tissue scars. These increased telocytes in the peri-infarct areas can be key modulators in post-infarction regeneration. It is suggested that transplantation of telocytes at the site of infarction in humans will increase angiogenesis and decrease fibrosis as seen in this study.[5,9]

4.3. Hypertrophy

The role of telocytes in cardiac hypertrophy is under experimentation in animal models. A study conducted on rats subjected to a week of exercise protocols showed a significant increase in the number of telocytes in test rats as compared to the controls,[9] signifying exercise training promoted the formation of new telocytes.[22] In this study out of the 22 cases of hypertrophy, telocytes were absent in the hypertrophied cardiac wall area in 16 cases, providing an average of only 1 telocyte per 10 fields. This decrease was statistically significant ($p \leq 0.05$). However, the 6 cases that showed telocytes had normal to slightly increased telocytes with up to 4 telocytes per 10 fields. This difference in the number of telocytes may be related to the activity status of the patient. In animal model studies cardiac tissue was examined after a period of active exercise regimen which caused changes in cardiac tissue as a result of an ongoing stimulus at the time of the study, hence an increase in telocytes was noted. Similarly in this study, the increase in telocytes in 6 cases may have been from patients with an active lifestyle with an ongoing stimulus for cardiac hypertrophy. However, a major limitation to getting patients details was the retrospective nature of this study. The study was conducted on PM samples and the physical exercise/activity status of the cases and the duration of hypertrophy were largely unknown, giving varied result among the cases.

4.4. Foetal heart

Among embryonic stages of mouse myocardium telocytes are in close proximity to the immature components of the developing heart indicating that telocytes modulate cardiogenesis.[23] In primary cultures derived from mice, cardiac telocytes express embryonic stem cell marker Nanog, and myocardial stem cell marker Stem Cell Antigen-1 (Sca-1).[24] Telocytes, in addition, also produce anti-apoptotic factors beneficial for tissue growth and remodelling.[25] There is, however, no data/literature available on telocytes in the foetal human heart. In the current study, scattered telocytes up to 5 per 10 fields were seen in the foetal myocardium. This increase was statistically significant ($p \leq 0.05$) signifying their unarguable role as stem cells during embryogenesis.

4.5. Blood vessels

The most significant antecedent event to IHD is atherosclerosis. It begins with marked endothelial dysfunction and lipid infiltration into the vessel wall followed by a production of pro-inflammatory cytokines and growth factors which exacerbate unsuitable vessel wall remodelling by smooth muscle proliferation. Experimentation on animals shows during atherosclerosis some telocytes retain their function and structure while some undergo several cytopathic changes at the ultrastructural level such as nuclear chromatin atrophy, cytoplasmic liposomes, and decreased endoplasmic reticulum and mitochondria.[10] In the current study, atherosclerotic blood vessels showed increased telocytes up to 14 per 10 fields in the wall of the blood vessel contributing to the process of atherosclerosis. This increase was statistically significant ($p \leq 0.05$) indicating that telocytes play a significant role in the pathogenesis of atherosclerosis in humans. Drugs and techniques that can target telocytes and modulate and/or inhibit their function appropriately, can prevent the progression of atherosclerosis by altering the pathogenetic mechanisms involved in atherosclerosis.

5. Limitation of Study

The current study was a retrospective study with a small sample size based entirely on PME findings. In some cases complete clinical details of cardiac diseases, antecedent

events, and ongoing treatments were unavailable. A more organised and focused multicentric approach to studying telocytes in the adult and foetal heart and blood vessels for changing the course of disease will greatly enhance the current scenario in regenerative cardiac medicine.

6. Conclusion

Telocytes have tremendous unexplored functionality in cardiac tissue. Normal adult and foetal cardiac tissue have telocytes, with a higher number in the foetal tissue indicating their definite role in cardiogenesis.

In IHD, there is loss of telocytes in the infarcted necrotic zone with an increase in telocytes around the zone of infarction promoting tissue regenerative activity beginning at the peripheries of the infarct. No telocytes are seen in areas of old connective tissue scars indicating destruction of telocytes causes loss of regeneration and leads to fibrosis. The current unfavourable outcome of a diseased heart can likely be modulated with the help of a better insight into telocyte function. Transplantation of cardiac telocytes in the zone of infarction can be experimented with as a novel therapeutic measure to salvage dysfunctional cardiac tissue.

In hypertrophic cardiac muscle, the timing of biopsy is important. Telocytes may increase only when there is a potentially high workload in the heart as a result of an active ongoing event creating a stimulus for the activation of telocytes in the myocytes.

In the atherosclerotic blood vessels, there is a marked increase in telocytes in the vessel wall causing vessel wall remodelling. Inhibition/suppression of telocytes in studies on human subjects may shed light on the status of its use as a therapeutic agent.

Future perspectives in 2020, telocytes were successfully isolated from the human skin by Romano et al using an immunomagnetic microbead-based technique. Such experiments have not been performed on the human heart.[26] Fully understanding the molecular heterogeneity of telocytes requires robust profiling of these cells. Standardized protocols for isolation, cultivation, and transplantation of telocytes may be a game-changer in cardiac homeostasis for the management of cardiac diseases.

7. Ethical Approval

This study was approved by Institute Ethical approval committee with ref. no. RRMCH-IEC/225/2023.

8. Source of Funding

None.

9. Conflict of Interest

None.

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Cite this article: Harrison A, Ananthraj J, Karishma K, Vejay Viknesh AM, Jahnavi C, Choudhary R, Prabha A. Expression of telocytes in cardiovascular diseases. *Panacea J Med Sci*. 2025;15(2):443-449.