We are IntechOpen,
the world's leading publisher of
Open Access books
Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

BOOK CITATION INDEX
CLARIVATE ANALYTICS
INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Chapter

Atherosclerosis: A Journey around the Terminology

Oladimeji Adebayo and Abiodun Moshood Adeoye

Abstract

The term atherosclerosis underwent a tedious pathway to arrive at its current status and interpretation. Furthermore, terms such as atherosclerosis, arteriosclerosis and arteriolosclerosis appear similar and are misused interchangeably. This chapter highlighted the various terminologies linked with atherosclerosis. This chapter highlighted how the terminology of atherosclerosis evolved and, also, the various classifications, e.g., atherosclerosis, Monckeberg calcific sclerosis and arteriolosclerosis, and gave mention to the differences among them.

Keywords: atherosclerosis, arteriosclerosis, arteriolosclerosis

1. Introduction

The understanding of atherosclerosis evolved uniquely in terms of terminology, aetiology, structural features or pathophysiology over the last 300 years [1]. Furthermore, the three terms, atherosclerosis, arteriosclerosis and arteriolosclerosis, with lethal implications and similar terminologies affecting the arterial vessels, however, can easily be confused or just used interchangeably indiscriminately [1–5]. There is evidence in the literature that these terms are mistakenly interchanged [3, 4, 6]. This confusion was not helped by the key stakeholders like the pathologists and clinicians who over the years failed to reach a consensus to delineate them [7].

A remarkable example of such confusion is that emanating from the American Heart Association (AHA) who publishes journals titled *Arteriosclerosis, Thrombosis, and Vascular Biology* and *Hypertension*. While the *Arteriosclerosis, Thrombosis, and Vascular Biology* journal publishes mainly articles related to the experimental, clinical and epidemiological facets of atherosclerosis, the *Hypertension* journal has a prominent section on "arteriosclerosis" [6].

Interestingly the confusion around the nomenclature appears to be age-long. Hueper, WC's writing in the *Archives of Pathology and Laboratory Medicine* in 1945 titled "Arteriosclerosis" suggests the term atherosclerosis was extensively used concerning the pathology related to cholesterol metabolism [8]. Rabson a medical doctor while writing a letter to the editor of the American College of Clinical Pathology expressed his alarm at the confusion of the degenerative disease [8, 9]. This alarm was stemmed from the observation of an article titled arteriosclerosis, but the article did not use any of such nomenclature rather than atherosclerosis [9].

As recent as 1995, the AHA Report from the Committee on Vascular Lesions of the Council Arteriosclerosis interchanged arteriosclerosis and atherosclerosis [11].

Not that there are no prominent attempts to delineate these terms, especially for arteriosclerosis and atherosclerosis. As far back as 1963, George Pickering, one of the fathers of modern cardiology, highlighted the similar confusion between arteriosclerosis and atherosclerosis in his lecture at The University of Alabama, published in the article "Arteriosclerosis and Atherosclerosis: The Need for Clear Thinking" which stated the definition in clear terms [6, 7]. He believed the confusion might be as a result of affectation of the arterial system by the two entities although they affect different arterial sizes. He, however, delineated them by defining atherosclerosis as *a complex inflammatory process associated with the presence of oxidized low-density lipoprotein (LDL) cholesterol in the intima and media of the arterial wall* and arteriosclerosis as the *functional depletion of large artery elasticity* [6].

Also, notable observation is that arteriosclerosis [10] serves two functions, an overarching term for other sub-terms and merely a term that can be inter-used with atherosclerosis [2, 11]. Besides, there is poor agreement on the terms related to atherosclerosis.

Also, over the years, other terms such as Mönckeberg medial calcific sclerosis (MCS) and arteriolosclerosis gained popularity [1]. There was a feeble attempt for clear terms and classification as related to atherosclerosis.

The chapter will also highlight recommendations on possible reclassification based on gross and histopathologic features, among others. In summary, the chapter will extensively discuss the key terms related to arteriosclerosis and the historical evolution of these terms and highlight recommendations on possible reclassification as having been previously recommended and published.

2. Arteriosclerosis

Arteriosclerosis is derived from the Greek word arteria, meaning artery, and sclerosis, meaning hardening, and "osis" is a Greek suffix that means a diseased condition [14]. Much literature appears to refer to arteriosclerosis as the overarching term that includes three different lesions, atherosclerosis, arteriolosclerosis and Mönckeberg medial calcific sclerosis (**Figure 1**) [2, 12]. The three lesions are underpinned by the resultant hardening and thickening of the arterial wall [2]. However there appears not to be any consensus document by any significant cardiovascular or pathology organization on such division rather than such classification emanated from classic textbooks of pathology [1]. Many articles also carry this classification [13].

It, however, appears that the term is also used to describe the functional diminution of large artery elasticity marked by pulse wave velocity [14]. It, however, brings to fore another dimension of intermix with atherosclerosis.
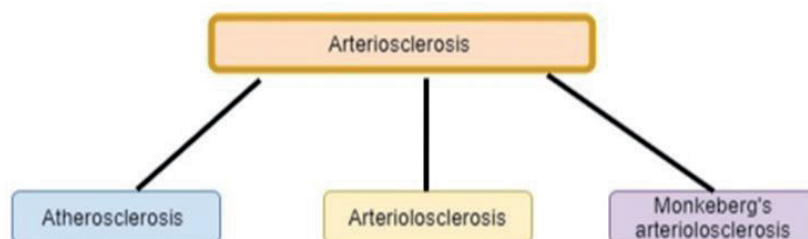
Figure 1.
The classification of arteriosclerosis.

3. Atherosclerosis

Atherosclerosis is derived from the Greek word "athero", meaning gruel or paste, and sclerosis, meaning hardening, and "osis" is a Greek suffix that describes a diseased condition [15]. It merely is the hardening of an artery precisely due to an atheromatous plaque. Atherosclerotic lesions otherwise called atheromata are asymmetric focal

Year	Milestones
1575	Fallopius wrote about "a degeneration of arteries into bone," and anatomists of that era commonly mentioned ossified arteries [2]
1740	Johann Friedrich Crell described this as the hardening of the vessels instead of bony arteries [17]
1755	von Haller described this hardening as thickening as "atheroma" [1, 37, 38]
1833	Frenchman Jean Frederic Martin Lobstein first used the term "arteriosclerosis" to describe calcified arterial lesions [1]
1868	George Johnson limited the term arteriosclerosis to "noncalcified, non-atheromatous stiffening of small vessels" [2]
1881	Jean Frederic Martin Lobstein first used the term arteriosclerosis in the second volume of his book titled *Traité d'Anatomie Pathologique* [17, 39]
1903	Mönckeberg arteriosclerosis was first described and named after Johann Georg Mönckeberg using details from 130 patients. It was also called "Mönckeberg media sclerosis" or "Mönckeberg media calcinosis" [38, 40]
1904	Félix Marchand introduced the term "atherosclerosis" and suggested it is responsible for obstructive processes in the arteries [2, 33, 41]
1908	A.I. Ignatowski demonstrated that rabbits experimentally develop atherosclerosis by feeding on cholesterol-rich food of egg and meat [33, 34]
1910	Adolf Windaus demonstrated that atheromatous plaque has a concentration of cholesterol [33, 42]
1914	Anitschkow described the role of cholesterol accumulation in the development of atherosclerosis [35]
2012	Faber differentiated calcification in the coronary arteries which are atherosclerotic in origin from Mönckeberg sclerosis [4]
1913	Nikolai N. Anichkov showed that cholesterol alone caused the atheromatous changes in the vascular wall [33, 43]
1954	Rabson SM noted that arteriosclerosis lacked specificity, uniformity and consistency. He suggested how the term would be used [2]
1961	Sir George Pickering, one of the fathers of modern cardiology in his lecture at the fourth Tinsley Randolph Harrison lecture at The University of Alabama, clearly differentiated arteriosclerosis and atherosclerosis
1965	Eggen and other workers demonstrated atherosclerotic coronary artery calcification [4]
1971	Russell Ross and Seymour J. Klebanoff using electron microscopy demonstrated that the atherosclerosis lesions are characterized by an accumulation of smooth muscle cells associated with abundant connective tissue matrix [44]
1972	Ross and colleagues showed that vascular smooth muscle cells proliferate and synthesize and secrete all three major constituents of connective tissue: collagen, elastic fibre microfibrils and elastin
1975	Watanabe heritable hyperlipidaemic rabbits were discovered and subsequently used in most experimental settings for lipid disorder and atherosclerosis [45]
1985	Brown and Goldstein discovered the role of low-density lipoprotein (LDL) receptors which won them the 1985 Nobel Prize
1992	The AHA started to release a series to define the intima of human arteries and its atherosclerosis-prone regions
1995	The AHA published the last of report classifying atherosclerosis using histological composition and structure [11]

Table 1.
Key milestone which refined the term atherosclerosis and related terms.

Atherosclerosis [18]	Arteriolosclerosis	Mönckeberg arteriosclerosis
Dyslipidaemia		Dyslipidaemia
Hypertension	Hypertension	
Diabetes mellitus	Diabetes mellitus	Diabetes mellitus
Genetic risk factors	Genetic risk factors such as ABCC9 gene variant	Genetic diseases such as Keutel syndrome
Inflammation		Chronic inflammatory disease such as systemic lupus erythematosus, etc.
Ageing	Ageing	Ageing
		Disturbances of calcium metabolism
Obesity		
Family history of early heart disease or coronary heart disease		
	Chronic kidney disease	Chronic kidney disease
Smoking and other tobacco use		
High level of CRP		
Alcohol		
Sleep apnoea		
Sedentary lifestyle		
Fibrinogen level		
Type A personality type		
Air pollution		
	Blood-brain barrier dysfunctions	

Table 2.
The risk factors associated with atherosclerosis, arteriolosclerosis and Mönckeberg's arteriosclerosis.

	Atherosclerosis	Arteriolosclerosis	Mönckeberg arteriosclerosis
Key processes	Lipid accumulation	Protein accumulation and fibromuscular proliferation of the intima	Calcium deposition
Type of process	Pathologic	Pathologic	Physiology and pathologic
Vessels affected	Large and medium arteries	Concentric media thickening of muscular arteries	
Part of the vessel affected	Intima and underlying smooth muscle	Media	Internal elastic lamina
Pathomorphological	Plaque-forming degenerative changes of the large elastic arteries such as the aorta	Thickening of the vessel walls that narrows the lumen	Patchy calcification of the intima
Effect on vessel dimension	Decrease	Decrease	Nil
Calcification	++		+++

Table 3.
Some key differences in atherosclerosis, arteriolosclerosis and Mönckeberg arteriosclerosis.

thickenings of the intima which is the innermost layer of the artery [16]. While arteriosclerosis is a chronic pathological disease concept which refers to arterial lesions characterized by intimal thickening, stiffening and remodelling of the arterial walls [2]. Atherosclerosis is a generic term used to describe a general term describing a hardening of medium or large arteries [1, 2]. It is also referred to as atherosclerotic vascular disease.

The atheroma principally blocks large- and medium-sized elastic and muscular arteries leading to ischemia in the heart, brain or extremities, thereby causing infarction [17]. The most critical risk factor for atherosclerosis is dyslipidaemia due to high plasma concentrations of cholesterol, especially low-density lipoprotein (LDL) cholesterol or high-density lipoprotein. While dyslipidaemia is a key risk factor, it can be found at age (**Tables 1–3**) [17]. The critical step in atherosclerosis formation includes (i) fatty streak formation, (ii) atheroma formation and (iii) atherosclerotic plaque formation [18].

4. Arteriolosclerosis

Arteriolosclerosis is simply the hardening and loss of the elasticity of the small arteries and arterioles due to the progressive increase in the elastic and muscular components of the wall of those vessels. It is simply the small vessel disease and principally affects the brain and the kidneys [19, 20]. In the brain, it is associated with the lacunar infarcts, vascular cognitive impairment and diffuse white matter lesions [19]. The key predisposing factors include hypertension, ageing, ABCC9 gene variant, diabetes mellitus and blood-brain barrier dysfunctions (**Tables 2** and **3**) [21]. The sub-types include hyaline arteriolosclerosis and hyperplastic arteriolosclerosis.

5. Mönckeberg medial calcific sclerosis (MCS)

Monckeberg medial calcific sclerosis (MCS), Mönckeberg arteriosclerosis, or Mönckeberg sclerosis is a degenerative and noninflammatory disease in which the media of medium-sized and small muscular arteries becomes calcified independent of atherosclerosis [22]. It is also considered as atherosclerotic medial calcification [4]. It is a ringlike calcification of the vascular media of small- to medium-sized vessels without associated intimal thickening [22, 23]. It is a form of arteriosclerosis or vessel hardening, where calcium deposits are in the muscular middle layer of the walls of arteries (the tunica media). It usually causes damage to the kidney and heart.

Mönckeberg arteriosclerosis is commonly mixed up with calciphylaxis and probably is the most controversial type of arteriosclerosis [4, 24].

6. Historical background of atherosclerosis

Atherosclerosis or other related nomenclatures are not new disease entities (**Tables 2** and **3**). There was, however, some emerging insight into what was later termed as atherosclerosis. Hippocrates (469–377 BC), the father of modern medicine, described the sudden cardiac death, while approximately 300 BC, Erasistratus described the typical claudication intermittent symptoms of peripheral arterial disease [25].

Egyptian mummies were noted to have evidence of atherosclerosis; although this was not yet named atherosclerosis [8, 26–28]. In the earlier times, due to the lack of sophisticated equipment to help the understanding, degenerative and nondegenerative arteriopathy was also lumped together and poorly delineated. However, the understanding of atherosclerosis increased exponentially in the last seven decades,

even though it was earlier thought to be a mere accomplishment of ageing and dodged for many years with controversies surrounding its link with cholesterol [27].

Albrecht von Haller, a Swiss biologist and the father of experimental physiology, used the Latin term "atheroma," in 1755, to describe the plaque deposited on the innermost layer of systemic artery walls [29]. However it appears that Celsius may have used the same word about 2000 years ago while describing a fatty tumour [30].

In 1575, Fallopius wrote about "a degeneration of arteries into bone" suggestive of the presence of calcified atherosclerotic lesions in the arteries. Furthermore, ossified arteries were commonly mentioned by anatomists of that era [2]. By the eighteenth century, it was apparent that some progress has been made in understanding albeit rudimentary on what we call atherosclerosis today. For example, in 1799, Parry suggested the relationship between coronary lesions and the symptoms of angina pectoris [25].

Also, in 1815, J. Hodgson defined the fatty arterial degeneration as atheromatosis [30].

Frenchman Jean Frederic Martin Lobstein first used the term "arteriosclerosis" to describe calcified arterial lesions in his main body of work, a four-volume work on pathological anatomy written in French titled *Traité d'Anatomie Pathologique (Treatise on Pathological Anatomy)*, based upon his vast personal experience [1, 25]. The work was unfinished at the time of his death in 1835.

The first mention of nondegenerative arteriopathy in the textbook was by Maurice Raynaud's in *De l'Asphyxie Locale et de la Gangrene Symetrique des Extremites* (1852) and Leo Buerger's "Thromboangiitis obliterans" [31]. While in 1879, more than 80 years later, Potain interpreted the relationship Parry found to arise from myocardial ischemia [25].

From 1900 onwards, the most significant progress in the understanding of atherosclerosis was made. At the beginning of the twentieth century, Aschoff introduced atherosis and atherosclerosis to describe morphologically different intimal lipid deposits of children and adults as early and late stages, respectively [32].

By 1904, Félix Marchand suggested that "atherosclerosis" should be better instead of "atheroma" as earlier described by Haller [29]. He had combined two Greek root words: *athéré, which* meant gruel or porridge, and *sclerosis*, which signifies hardening [29]. He did not only gave a nomenclature currently in use, but he also did justice to the pathologic process involving the association of fatty degeneration and vessel stiffening. He believed that atherosclerosis was responsible for almost all obstructive processes in the arteries [33]. In 1910, Adolf Windaus demonstrated that aortic "atheromatous lesions" contained six times as much as free cholesterol and 20-fold of esterified cholesterol compared to normal aortic wall [27, 33]. A.I. Ignatowski demonstrated that rabbits experimentally develop atherosclerosis by feeding on cholesterol-rich food of egg and meat [33, 34].

In 1914, Anitschkow, influenced by Ignatowski's work, gave the first description of the role of cholesterol accumulation in the development of atherosclerosis while emphasizing the cardinal role of cholesterol in atheromatous changes in the vascular wall [35]. He had used a cholesterol-fed rabbit model while working at the Military Medical Academy in St. Petersburg to demonstrate that the extent of atherosclerosis was proportional to the absolute amount of and length of exposure to high blood cholesterol [27, 35]. His discovery was a defining moment in the study of this disease entity, although not without scepticism. While the rabbit model develops atherosclerosis after being fed high amounts of meat, eggs and milk, the dog and rat model did not [33]. This finding by later workers almost torpedoed this discovery attributable to Anitschkow [27]. It was also a blow to the lipid hypothesis due to these later species relative resistance to diet-induced hypercholesterolemia. Other factors that may have eroded the most unequivocal link of

cholesterol and atherosclerosis as enunciated by Anitschkow was the prevailing senescence hypothesis as a plausible reason for the development of atherosclerosis when he made the discovery [27]. Anitschkow also first described the *cholester-inesterphagozyten*, which today commonly is known as foam cells, derived from macrophages [25, 35].

The discussion of lipid hypothesis generally in the first 50 years of the twentieth century was also dodged with controversy, particularly the role of high blood cholesterol levels to the causal relationship of atherosclerosis and coronary heart disease, and that atherosclerosis was a reward of ageing as espoused by senescence hypothesis [27, 36].

With the improvement in morphological, immunohistological and molecular methods and advanced investigative techniques, LDL receptor and its roles in the development of atherosclerosis were discovered by Brown and Goldstein [25]. By 1958, there was enough information to make classification of atherosclerosis by the World Health Organization and described the sequences as a fatty streak, atheroma, fibrous plaque and complicated lesions [32]. The American Heart Association (AHA) starting from 1992 proposed a new morphological classification based on eight lesion types designated by Roman numerals which indicate the usual sequence of lesion progression [32].

Jian-Jun Li and Chun-Hong Fang's writing in *Medical Hypotheses* journal suggests that atheroscleritis is a more rational term for atherosclerosis [29].

We now know the pathological processes that underpin atherosclerosis, which entails (1) endothelial injury, (2) intimal cholesterol accumulation and monocyte invasion with subsequent foam cell formation, (3) migration and proliferation of smooth muscle cells with expression of extracellular matrix, (4) local thrombus formation with secondary organization, (5) calcification and/or plaque rupture and (6) final occlusion due to plaque rupture/thrombus formation [25].

7. Challenges with terms

Gregory et al. pointed out the difficulty with terms which they notice: (i) it has an inconsistent naming convention, (ii) it fails to use terms that accurately describe the lesions, (iii) significant sclerotic arterial lesions are absent from the classification, and (iv) interchangeability of arteriosclerosis and atherosclerosis [2]:

1. **Naming convention.** The three authors noticed that the arteriosclerosis, atherosclerosis and Mönckeberg medial sclerosis are defined by their gross and histopathologic attributes while arteriolosclerosis was defined by vessel dimension [2]. Furthermore, "arteriolo" is a descriptive pathologic term.

2. **Description of lesion.** This term poorly describes the terms [2]. The Mönckeberg medial sclerosis as described by Mönckeberg, ironically, may not be described, and the lesion may involve the arterial intima rather than the arterial media [2]. Furthermore, arteriolosclerosis, which is a hardening of the vessel, does not describe the lesion since the process involves both protein accumulation and fibromuscular proliferation of the intima [1].

3. **Absence of crucial lesion from the classifications.** Restenosis lesions after balloon angioplasty and stenting, transplant arteriopathy, intimal nonatherosclerotic proliferative lesions in arterial vessels larger than arterioles, and a variety of disorders associated with vascular calcification are not yet included in the current classification.

Although Gregory et al. highlighted the problems with terms, they retained the classifications which do not have all the necessary inclusion [2]. They suggested atherosclerosis, primary arterial calcification, fibromuscular intimal hyperplasia, hyalinosis, and miscellaneous categories which include amyloidosis and oxalosis, among others [1].

8. Conclusion

Atherosclerosis, arteriosclerosis and arteriolosclerosis are not only confused; they are also associated with some controversies. However, while the current classifications and description of terms have been developed over the centuries, there are possible better ways to classify the terms.

Author details

Oladimeji Adebayo[1*] and Abiodun Moshood Adeoye[1,2,3]

1 Cardiology Unit, Department of Medicine, University College Hospital, Ibadan, Nigeria

2 Department of Medicine, University of Ibadan, Ibadan, Nigeria

3 Cardiovascular Genetics and Genomic Research Unit, Institute of Cardiovascular Diseases, Faculty of Clinical Sciences, College of Medicine, University of Ibadan, Ibadan, Nigeria

*Address all correspondence to: doctorladi@gmail.com

IntechOpen

References

[1] Fishbein MC, Fishbein GA. Arteriosclerosis: Facts and fancy. Cardiovascular Pathology. 2015;**24**(6):335-342

[2] Fishbein GA, Fishbein MC. Arteriosclerosis: Rethinking the current classification. Archives of Pathology & Laboratory Medicine. 2009;**133**(8):1309-1316

[3] Schwartz C, Mitchell J. The morphology, terminology and pathogenesis of arterial plaques. Postgraduate Medical Journal. 1962;**38**(435):25

[4] McCullough PA et al. Accelerated atherosclerotic calcification and Mönckeberg's sclerosis: A continuum of advanced vascular pathology in chronic kidney disease. Clinical Journal of the American Society of Nephrology. 2008;**3**(6):1585-1598

[5] Schaar JA et al. Terminology for high-risk and vulnerable coronary artery plaques. European Heart Journal. 2004;**25**(12):1077-1082

[6] Lotufo PA. New findings about atherosclerosis in Brazil from the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil). Sao Paulo Medical Journal. 2016;**134**(3):185-186

[7] Pickering SG. Arteriosclerosis and atherosclerosis: The need for clear thinking. The American Journal of Medicine. 1963;**34**(1):7-18

[8] Thomas GS et al. Why did ancient people have atherosclerosis?: From autopsies to computed tomography to potential causes. Global Heart. 2014;**9**(2):229-237

[9] Rabson SM. Arteriosclerosis: Definitions. American Journal of Clinical Pathology. 1954;**24**(4):472-473

[10] Hueper WJ. Arteriosclerosis. Archives of Pathology. 1945;**39**:187-216

[11] Stary HC et al. A definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis. Circulation. 1995;**92**:1355-1374

[12] Sakata N. AGEs and atherosclerosis. Anti-Aging Medicine. 2012;**9**(3):89-95. Department of Pathology, Faculty of Medicine, Fukuoka University

[13] Fishbein GA, Fishbein MC. Arteriosclerosis. Archives of Pathology & Laboratory Medicine. 2009;**133**(8)

[14] Wilkinson IB, McEniery CM, Cockcroft JR. Arteriosclerosis and atherosclerosis: Guilty by association. American Heart Association. 2009;**54**:1213-1215

[15] Soltero-Perez I. Toward a new definition of atherosclerosis including hypertension: A proposal. Journal of Human Hypertension. 2002;**16**(S1):S23

[16] Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. New England Journal of Medicine. 2005;**352**(16):1685-1695

[17] Ross R. Atherosclerosis—An inflammatory disease. New England Journal of Medicine. 1999;**340**(2):115-126

[18] Rafieian-Kopaei M et al. Atherosclerosis: Process, indicators, risk factors and new hopes. International Journal of Preventive Medicine. 2014;**5**(8):927-946

[19] Bridges LR et al. Blood-brain barrier dysfunction and cerebral small vessel disease (arteriolosclerosis) in brains of older people. Journal of Neuropathology

& Experimental Neurology. 2014;**73**(11):1026-1033

[20] Kryscio RJ et al. The effect of vascular neuropathology on late-life cognition: Results from the SMART project. The Journal of Prevention of Alzheimer's Disease. 2016;**3**(2):85-91

[21] Ighodaro ET et al. Risk factors and global cognitive status related to brain arteriolosclerosis in elderly individuals. Journal of Cerebral Blood Flow Metabolism. 2017;**37**(1):201-216

[22] Amann K. Media calcification and intima calcification are distinct entities in chronic kidney disease. Clinical Journal of the American Society of Nephrology. 2008;**3**(6):1599-1605

[23] Saxena A et al. Monckeberg medial calcific sclerosis mimicking malignant calcification pattern at mammography. Journal of Clinical Pathology. 2005;**58**(4):447-448

[24] Micheletti RG et al. Mönckeberg sclerosis revisited: A clarification of the histologic definition of Mönckeberg sclerosis. Archives of Pathology & Laboratory Medicine. 2008;**132**(1):43-47

[25] Hanke H, Lenz C, Finking G. The discovery of the pathophysiological aspects of atherosclerosis—A review. Acta Chirurgica Belgica. 2001;**101**(4):162-169

[26] Widmer L, Da Silva A. Historical perspectives and the Basle study. In: Epidemiology of Peripheral Vascular Disease. Springer; 1991. pp. 69-83

[27] Steinberg D. Thematic review series: the pathogenesis of atherosclerosis. An interpretive history of the cholesterol controversy: Part I. Journal of Lipid Research. 2004;**45**(9):1583-1593

[28] Hart GD et al. Lessons learned from the autopsy of an Egyptian mummy. Canadian Medical Association Journal. 1977;**117**(5):415

[29] Li J-J, Fang C-H. Atheroscleritis is a more rational term for the pathological entity currently known as atherosclerosis. Medical Hypotheses. 2004;**63**(1):100-102

[30] Cottet J, Lenoir M. Two thousand years of historical study on the words atheroma, atheromatosis, atherosclerosis, arteriosclerosis. Bulletin de l'Académie Nationale de Médecine. 1992;**176**(9):1385-1390. discussion 1390-1

[31] Widmer LK, Da Silva A. Historical perspectives and the basle study. In: Fowkes FGR, editor. Epidemiology of Peripheral Vascular Disease. London: Springer London; 1991. pp. 69-83

[32] Gaudio E et al. Morphological aspects of atherosclerosis lesion: Past and present. La Clinica Terapeutica. 2006;**157**(2):135-142

[33] Konstantinov IE, Mejevoi N, Anichkov NM, Nikolai N, et al. Anichkov and his theory of atherosclerosis. Texas Heart Institute Journal. 2006;**33**(4):417

[34] Ignatowski A. Über die Wirkung des tierischen Eiweisses auf die Aorta und die parenchymatösen Organe der Kaninchen. Virchows Archiv für Pathologische Anatomie und Physiologie und für Klinische Medizin. 1909;**198**(2):248-270

[35] Enas EA, Kuruvila AT, Lulla S. Retracing the heroic steps fromlipid hypothesis to aggressive treatment of blood cholesterol—A revolution in preventive cardiology. In: Choptra HK, Nanda Navin C, editors. Textbook of Cardiology. New Delhi: Jaypee Brothers Medical Publishers(P) Ltd; 2013. pp. 180-194

[36] Steinberg D. Thematic review series: The pathogenesis of atherosclerosis. An interpretive history of the cholesterol controversy, part V: the discovery of the statins and the end of the

controversy. Journal of Lipid Research. 2006;**47**(7):1339-1351

[37] Blumenthal HT. Cowdry's Arteriosclerosis: A Survey of the Problem. Thomas; 1967

[38] Drüeke TB. Arterial intima and media calcification: Distinct entities with different pathogenesis or all the same? Clinical Journal of the American Society of Nephrology. 2008;**3**(6):1583-1584

[39] André É, Lobstein J-F. Artériosclérose et ostéoporose. Société Française D'histoire De La Médecine. 2018;**52**:197

[40] Mönckeberg JG. Über die reine Mediaverkalkung der Extremitätenarterien und ihr Verhalten zur Arteriosklerose. Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin. 1903;**171**:141-167

[41] Marchand F. Ueber Atherosclerosis. Verhandlungen der Kongresse fuer Innere Medizin. 1904;**21**

[42] Windaus A. Über den Gehalt normaler und atheromatöser Aorten an Cholesterin und Cholesterinestern. Hoppe-Seyler´s Zeitschrift für physiologische. Chemie. 1910;**67**(2): 174-176

[43] Anitschkow N. On experimental cholesterin steatosis and its significance in the origin of some pathological processes (1913). Arteriosclerosis. 1983;**3**:178-182

[44] Ross R, Klebanoff SJ. The smooth muscle cell: I. In vivo synthesis of connective tissue proteins. The Journal of Cell Biology. 1971;**50**(1):159-171

[45] Kondo T, Watanabe Y. A heritable hyperlipidemic rabbit. Jikken Dobutsu. 1975;**24**(3):89-94