

Striated muscles in visceral organs of vertebrates

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Abstract

Pathologists involved in the examination of small rodent tissues are familiar with the presence of striated muscle in walls of intrapulmonary veins. Because these striated muscles express cardiomyocyte markers and are involved in rhythmical contraction during systole, this morphologic arrangement was named “pulmonary myocardium”. Striated cardiac muscles of intrapulmonary veins have also been found in numerous species, including non-human primates. Great attention has been given to animal striated pulmonary veins because in humans, the presence of cardiomyocytes in the pulmonary veins (myocardial sleeves) is a major origin of paroxysmal atrial fibrillation. It is unequivocally suggested that direct extension/migration of cardiomyocytes from the left atrium into pulmonary veins is the origin of “pulmonary myocardium”. This model sounds logical in regard to humans and large mammals because in these cases, striated myocytes extend into pulmonary veins only by 10–30 mm from the left atrium. However, the cardiomyocyte direct extension/migration model becomes less parsimonious when applied to the numerous small intrapulmonary veins with striated muscles in their walls in small mammals. In 1972, Alfred Sherwood Romer, renowned for his contributions to the study of vertebrate evolution, suggested that visceral smooth muscle cells developed a striated phenotype due to the need for more efficient musculature. Romer theorized that the primary anatomical division of the vertebrate muscular system should be not into smooth and striated types but into somatic and visceral systems, because the line of division along the gut between striated and smooth musculature is not a fixed point. Romer’s work offered a parsimonious model for the presence of striated muscle in the walls of the intrapulmonary veins—visceral smooth muscle cells, which are capable of differentiating into striated muscle cells, are always present; whether they differentiate depends on functional demands. From Romer’s hypothesis, it also could be foreseen that striated muscle could appear in visceral compartments other than the blood circulatory system and pharyngeal muscles to facilitate functional needs. Indeed, it was shown that 1) the lymphatic system of many low vertebrates acquired a lymphatic heart with striated muscle cells; 2) some vertebrates also acquire striated muscles in *tunica Muscularis* of the stomach and intestine. The facts above undoubtedly corroborate Prof. Romer’s notion on the evolution of the vertebrate muscular system.

Keywords: Vertebrates, evolution, visceral organs, smooth muscles, striated muscles, intrapulmonary veins, lymphatic hearts, Alfred Sherwood Romer

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The daily experience of any microscopic pathologist (anatomical, clinical, veterinarian, or toxicologic) contains unexpected morphologic findings. They can be pathologic findings in organs that ought to be normal as the organisms have not shown any signs of diseases. We categorize such findings as a “background or accidental” pathology [1]. Other unexpected findings include normal (or altered) structures in an unexpected location. These atypically located structures, termed

“heterotopic”, are usually results of genetic or developmental errors. The above two types of unexpected findings are rare (except in genetically modified animals), and we consider them “structural noise”. However, there is a morphologic phenomenon of a third type, which is more perplexing than unexpected. It is the constant participation of cells/tissues in the formation of normal organs in situations when the participation of these particular cells is puzzling and contradicts common sense. I would like to elaborate on the above notions by providing

the example of the permanent presence of striated muscles in visceral organs.

During my decades of experience in pathology, I have come countless times across the same routine finding: the presence of striated muscle fibers in lung sections. These striated lung muscles, also known as 'pulmonary myocardium', appear in a wall of pulmonary veins of rodents and many other species and are familiar to all morphologists, especially those involved in microscopic analysis of rodent tissues and toxicologic pathology (Figure 1).

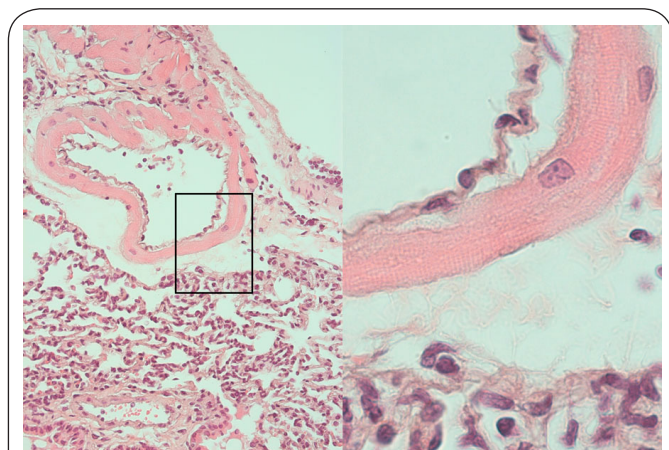


Figure 1. Pulmonary vein of mouse lung, H&E-stained paraffin section. Left: magnification 400x, right: 1250x. Note a visible muscle striation in the right image.

However, no matter how familiar the finding is, it is always puzzling: Where do these striated muscles come from?

Currently, there is only one explanatory hypothesis of the origin of striated muscles in pulmonary veins. This model includes two non-excluding morphogeneses: 1) extension of sleeves of striated cardiac myocytes from the left atrium to the wall of pulmonary veins during embryogenesis [2]; and 2) migration of atrial myoblasts into pulmonary veins during atrial septation [3]. Therefore, the hypothesis assumes direct extension/migration of myocytes from the left atrium and unequivocally suggests the myocardial origin of striated muscles in pulmonary veins [4]. This model sounds logical in regard to large mammals (including humans) because in these cases striated myocytes extend to pulmonary veins only by 10-30 mm from the left atrium [5-9]. This striated muscle compartment of pulmonary veins in humans is the subject of special clinical attention because it is the source of ectopic beats initiating atrial fibrillation [10-13]. Similar myocardial striated sleeves extending near heart chambers into the beginnings of main vessels were found in the aorta, pulmonary artery [14,15], and caval veins [16].

However, the cardiomyocyte direct extension/migration model becomes less parsimonious when applied to numerous small intrapulmonary veins with striated muscles in their

walls. Although the presence of striated muscle in smaller intrapulmonary veins inversely correlates with body mass (and positively with heart rate) [17], the appearance of striated muscles in distal segments of numerous intrapulmonary veins begs for a model other than direct cardiomyocyte extension/migration from the left atrium. Striated myocytes were found in pulmonary veins with a diameter of 70-250 microns and even in pulmonary veins of 30 microns in diameter in numerous species [18-23]. A quest for an alternative model also appears logical because cardiomyocyte extension/migration to distal pulmonary veins has been never demonstrated; the evidence has shown similarity in gene expression between striated muscle of pulmonary veins and heart myocytes [3,4,24], which neither is a proof of cell migration nor the same developmental origin [25].

Therefore, the question is as follows: How can the presence of striated myocytes in small intrapulmonary veins, which are located very distal to the heart, be explained?

Recently, I realized that this quest was answered long ago.

I was studying (for an unrelated reason) the famous work of Alfred Sherwood Romer 'The Vertebrate as a Dual Animal – Somatic and Visceral', when I came across the following notes on striated visceral muscle of mesenchyme origin:

"Histologically, vertebrate musculature can be divided into the striated type, which makes up the "flesh" of the body, and smooth musculature, with simpler fibers, which is mainly confined to the gut tube, although with "outliers" (particularly in higher vertebrates) in the vascular system and so forth (it is generally agreed that heart muscle is a derivative of the smooth muscle type). (p.122, [26]; and further:

"It seems clear that the pharyngeal group of muscles are a specialized part of a set of visceral muscles, associated with the gut, arising in a fashion similar to the smooth musculature found more posteriorly, but developing as striated fibers in connection with the functional need for more efficient musculature in the mouth and pharynx region." [26] (p.125).

The above notions carry two theoretical implications. Considering that the development of a striation phenotype in mesenchymal smooth muscle fibers is a reflection of functional needs and selection, it could be foreseen that:

1) this phenomenon could appear in the blood circulatory system in more than one place (i.e., heart); and 2) the striated-muscle phenotype also could appear in visceral compartments other than the blood vascular system and the pharyngeal group of muscles if functional needs required it.

I decided to check whether there are available facts that support these theoretical implications, and the results of this simple inquiry stunned me.

Implication #1

This phenomenon could appear in the blood vascular system in more than one place (i.e., heart):

While I read 'The Vertebrate as a Dual Animal – Somatic and Visceral' long ago, only recently did I realize that the puzzling

morphology of distal intrapulmonary veins constitutes the same phenomenon that Romer described as “the functional need for more efficient musculature” [26] because pulmonary veins rhythmically contract and propel blood to the left atrium [3,18-20].

Within Romer’s model, the puzzling and paradoxical striated muscle phenotype of distal pulmonary veins becomes explainable and expected.

Because the occurrence of the striated muscle phenotype in the heart is consequential to functional needs and selection, this logic is applicable to any blood-conducting compartment under similar demands. We do not have to infer a complicated model based on the myocardial origin of pulmonary striated muscles and cell migration along each developing intrapulmonary vein up to 30-50 microns in diameter. There is a parsimonious model-visceral smooth muscle cells, which are capable of differentiating into striated muscle cells, are always present; whether they do differentiate into cardiac phenotype is just a matter of the functional demands. Therefore, the myocytes of intrapulmonary veins acquired the striation phenotype independently of heart myocytes due to the demand for “the functional need for more efficient musculature” [26].

In clinical pathology, the finding of striated muscle fibers in lung parenchyma, not in conjunction with vasculature, is not a rare observation [27-33]. It is usually attributed to trapping fragments of foregut tissues (i.e., pharyngeal muscles) into lung parenchyma before separation of the pulmonary bud from esophagus tissues in embryogenesis [28]. However, it was long ago suggested by R.A. Willis that striated muscles may develop from the lung mesenchyme itself as heteroplasia [34] (page 345). Sequestration of foregut cells into developing lung for striated muscle morphogenesis also seems unnecessary because many genes directing the striated muscle phenotype appeared activated in developing lung parenchyma [35]. It also was shown that visceral smooth muscle cells are able to develop the striated muscle phenotype well after commitment and differentiation [36], and cell migration is not necessary for the phenotype switch [37]. Furthermore, there is a known gene expression program that facilitates the spontaneous switch from a smooth to a skeletal muscle phenotype [38], and this switch can be bifunctional [39].

Implication #2

Striated muscle could appear in visceral compartments other than the blood circulatory system and pharyngeal group of muscles to facilitate functional needs.

In vertebrates, a lymphatic system has to facilitate movement of lymph in one direction—from the periphery to the central venous system. Lymph propulsion is facilitated by voluntary muscle contraction, which surrounds conducting lymphatic vessels (mostly in the extremities), by the thrust of pulsing arteries (deep collecting lymphatics usually lying contiguous to the arteries in confines channels) [40] and by coordinated contraction of smooth muscle cells in lymph-collecting vessels [41]. Directional movement of the lymph

is facilitated by lymphatic endothelial flaps, or valves, which prevent retrograde flow [42]; the contracting segment of a lymphatic vessel with two valves is known as a lymphangion [43].

What is less well known is that in addition to the above recognized mechanisms facilitating lymph movement, the lymphatic system of many vertebrates acquires a specific contractile organ named the lymphatic heart [44-57]. It was documented by many investigations that the main structural component of lymphatic hearts is striated muscle cells [e.g., [45,46,48,50-52,54], with only one report on smooth muscle morphology of the lymphatic heart [49].

Apart from blood and lymphatic conducting systems, and visceral musculature in the mouth and pharynx regions, some vertebrates also acquire striated muscles in *tunica Muscularis* of the stomach [58,59] and intestine [60,61].

The facts above undoubtedly corroborate Prof. Romer’s hypothesis on the design of the vertebrate muscular system:

“Further evidence that the primary anatomical division of the vertebrate muscular system should be not into smooth and striated types but into somatic and visceral systems is afforded by the fact that the line of division along the gut between striated and smooth musculature is not a fixed point.” [26] (p.125).

In addition to having great theoretical importance, the above statement on Romer’s work highlights the feasibility of a very practical biomedical goal—the creation of an additional heart. The heart muscle is a derivative of the smooth muscle lineage, and differentiation of cardiac striated muscular compartments from smooth muscle cells or their progenitors is a physiologic event. Such organogenesis, as Prof. Romer noted, is the result of selection complying with “the functional need for more efficient musculature” [26]. Romer’s insights together with the presence of myocardial compartments with autonomous contractility in visceral organs of vertebrates (pulmonary artery and lymphatic system) and recent scientific discoveries (e.g., [62-67]) should encourage funding of research aiming to construct of additional vascular compartments to propel blood in systemic circulation. The creation of “additional heart” is an enormous practical challenge but not a theoretical barrier.

Competing interests

The author declares that he has no competing interests.

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