cell expressed FLI-1 and CD31; but were negative for S100-antigen, CD34, CD45, SOX10, CK AE1/AE3, EMA, and desmin. Therefore, the patient was a candidate for radiation therapy.

Cardiac tumors are rare. Patient appropriate treatment for cardiac tumor depends on histological diagnosis and staging. Non-surgical cardiac mass biopsy evaluation can be done by transvascular (artery and vein) and percutaneous approach. Right cardiac biopsies are commonly performed through the venous access. Left cardiac biopsies are most commonly performed through the arterial access. Not all patients with left atrium lesions can undergo diagnostic and therapeutic surgeries.

Percutaneous CT guided cardiac biopsy procedures date back to the early 2000s (2, 3) particularly for the left atrial masses. Percutaneous thoracic imaging guided approach can be indicated in selected cases, with previous multidisciplinary discussions paying attention on the mass anatomical location, characteristics, and extension (1-4). In general, percutaneous transthoracic biopsy procedures may be useful in those cases where traditional approaches or methods have resulted in a possible technical failure or are too complicated. In conclusion, cardiac masses located in the posterior wall of the left atrium can be one of the cases where percutaneous imaging guided biopsy is indicated.

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Shock wave therapy in cardiology: A comment

To the Editor,

Cardiac shock wave therapy (CSWT) was developed based on lithotripsy in that it uses low-intensity shock waves to stimulate angiogenesis (1). Its therapeutic potential was first demonstrated in porcine models of chronic myocardial ischemia, acute infarction, and ischemia-reperfusion injury. Experimental studies have been short-termed, thus unable to provide information on potential late consequences, and only relatively small-scale studies found evidence supporting the clinical application of CSWT (1). Shock waves induce shear stress in tissues (1, 2). Some studies indicate that physical characteristics of CSWT partly overlap with those associated with damage. Shock waves with the flux density 0.09 mJ/mm^2 were used in (1). Approximately one-tenth of the total power used for lithotripsy is usually applied to the heart, which corresponds to the energy flux density ~0.09 mJ/mm^2 and peak pressure of 10 MPa (3). In comparison, shock waves with the peak pressure 10 MPa caused lung bleedings in dogs (4). Histological evidence of damage were observed in murine renal medulla following the shock wave impact with peak pressures 3–5 MPa; severe damage appeared after 15–20 MPa shocks (5). Ultrastructural damage can be histologically invisible. Abnormalities were seen by electron microscopy in rats after a shock wave impact with the energy flux density 0.1 mJ/mm^2; the scores of myocardial ultrastructure damage in the CSWT vs. sham control groups were 2.42 and 1.39 correspondingly (p=0.103) (2). The peak pressure recommended by for the shock wave device used in Moscow has been around 10 MPa (6). In regard to mechanisms, immediate vasodilation has been ascribed to nitric oxide (NO) whose half-life in living tissues is several seconds, which means the NO-mediated action would not last long. The stimulation of angiogenesis is supposed to result from activation of the vascular endothelial growth factor (VEGF), which has an ambiguous role in ischemic heart disease as it can induce proliferation of fibroblasts and myofibroblasts, thus contributing to fibrosis. In coronary arteries, the smooth muscle proliferation due to VEGF may facilitate the growth of atherosclerotic plaques. Presumably, VEGF attracts inflammatory cells into the intima at different stages of atherogenesis (7, 8), which if enhanced might contribute to their instability. In conditions of atherosclerosis, elevated serum VEGF was associated with adverse cardiac events (8). Reported CSWT effects may be transient and reactive in their nature. The placebo effect may partially prompt subjective improvements. Additional impact upon cardiomyocytes, pre-damaged by ischemia, might contribute to apoptosis. Given the limited regeneration capacity, this may result in some degree of interstitial fibrosis. Evaluation of fibrosis by morphometry in the experimental material is technically possible. Other potential late consequences such as enhanced atherogenesis, angiogenesis in plaques, and their instability would be difficult to assess

in experiments. A net harm or benefit can be evaluated in animal studies with comparisons of natural life span between the test and control groups. In the author's opinion, experiments with a longer observation time should be performed prior to large-scale clinical trials.

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