The interplay of three factors is generally held accountable for the pathogenesis of most cases of aortic dissection – an abnormality or weakening of the aortic media (as may occur with ageing, certain congenital cardiovascular anomalies, and the fibrillinopathies), an agent of intimal injury or tear (as occurs with atherosclerosis or hypertension) and hemodynamic factors (systemic blood pressure) responsible for a pressure head that drives blood to dissect the aortic wall. It is difficult to conjecture the contribution of sildenafil to these three factors. The strong temporal relationship between sildenafil intake and the onset of dissection is the most potent argument. This was true in the cases reported so far [4]. Sildenafil has an antiproliferative effect on vascular smooth muscle cells in the pulmonary circulation [4]; possibly, similar effects on the aorta may lead to non-inflammatory smooth muscle cell loss in the aortic media rendering it more vulnerable to dissection.

On the other hand, all three reported cases of aortic dissection involving the use of sildenafil had other well established predisposing factors – old age and hypertension [4], cocaine abuse [5], and bicuspid aortic valve with ascending aortic aneurysm [1]. In addition, these patients had been taking sildenafil for months until the dissection. It may be speculated that chronic sildenafil use induces some changes in the aortic wall that ultimately makes dissection a possibility. Nevertheless, until more evidence is accumulated, it appears the role of sildenafil in the causation of aortic dissection shall remain speculative.

References