Swiss Medical Weekly

Formerly: Schweizerische Medizinische Wochenschrift An open access, online journal • www.smw.ch

Technical comment | Published 20 November 2017 | doi:10.4414/smw.2017.14561 Cite this as: Swiss Med Wkly. 2017;147:w14561

Technical comment on: Gawinecka et al. Acute aortic dissection: pathogenesis, risk factors and diagnosis

Aortic dissections: time for revising classifications

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I read with great interest the review recently published by J. Gawinecka et al. [1]: "Acute aortic dissection: pathogenesis, risk factors and diagnosis". The authors report an update that summarises the current knowledge on aortic dissection. I regret, however, that two major issues, matters of debate and controversy, were not discussed.

Firstly, the reported classifications, the DeBakey [2] and the Stanford [3] classifications, are of course widely used to characterise aortic dissection. In the DeBakey classification, described in 1965, type I is defined as involvement of the ascending, aortic arch and descending aorta, in type II only the ascending aorta is involved, and type III is confined to the descending aorta. In the Stanford system, proposed in 1970, the extension of the membrane to the ascending aorta defines the type A, whereas type B is defined by a membrane limited to the thoracic aorta distal to the left subclavian artery. Both systems drive the decision-making process of patient management well, in the context of open surgery or conservative medical treatment. Advances in diagnostic imaging, mainly from computed tomography (CT) angiography, provide more refinement in the features of this pleomorphic disease. A remaining configuration where the dissection is extended or initiated at the aortic arch and not involving the ascending aorta, that some called "non-A non-B aortic dissection" is not covered by these systems [4]. This configuration is routinely observed. Whether management should be similar to that of type A or type B or should be specific is matter of discussion. Appropriately describing this condition helps in understanding how this differs from other types, in evaluating the natural history and, in consequence, in establishing strategies for appropriate management, including modern endovascular approaches. A simple proposal, based on the Stanford classification, might be a modified Stanford classification, in which the non-A non-B aortic dissection described above is identified as type C:

Correspondence: Salah Dine Qanadli, MD, PhD, FCIRSE, CHUV, BH10-107, Bugnon 46, CH-1011 Lausanne, Salah.Qanadlifat]chuv.ch type A – involvement of the ascending aorta irrespective to the extension;

type B – involvement of the descending aorta exclusively; type C – involvement of the aortic arch without involvement of the ascending aorta. To drive further invasive actions (stentgrafts or stents), subtypes according to the absence (a) or presence (b) of a malperfusion syndrome (peripheral or visceral) could be added to types B and C (e.g., type Bb corresponds to type B dissection with a malperfusion syndrome).

The second issue is related to the continuum between the most common acute aortic syndromes: dissection, intramural haematoma (IMH) and penetrating aortic ulcer. From the pathogenesis point of view, the relationship between these entities should be highlighted. In routine practice, many patients who presented with aortic dissection in an anatomic segment of the aorta had an IMH in a different segment of their aorta at the initial imaging work-up (e.g., aortic dissection of the ascending aorta and IMH in the descending aorta). Furthermore, IMH is considered as precursor of aortic dissection [5]. At least one third of patients with intramural haematoma will have a transformation to aortic dissection over a matter of weeks. Similarly, a penetrating aortic ulcer is considered to be a precursor of IMH. Understanding this pathological continuum might help in identifying and classifying correctly acute aortic syndromes.

I would conclude that the current classification systems are outdated. They are clearly not sufficient to characterise all observed aortic dissection patterns. The clinical impact of misclassifying acute aortic syndromes is not negligible, particularly in the era of advanced minimally invasive transcatheter therapies. A modification is needed to include the so-called "non-A non-B dissection" and integrate malperfusion syndromes. Coexistence of aortic dissection and IMH is not rare and should be integrated into diagnosis, extension and classification of aortic dissections.

Disclosure statement

No financial support and no other potential conflict of interest relevant to this article was reported.

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