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## Aortic dissection and aneurysm are hypertensive target organ damages and should be listed in the guidelines



### To the Editor:

The significance of aortic (Ao) diseases derives from the prevalence, a dramatic clinical picture and a very high mortality rate in acute forms. Moreover, misdiagnosis and the economic burden are also not to be neglected.

The prevalence of abdominal aortic aneurysm (AAA) in men over 60 years old is about 5% and it doubles in men over the age of 80 years [1,2]. Aneurysms of thoracic aorta represent a 1/5 to a 1/4 of all Ao aneurysms [3]. In the US, AAA has been reported to be in the 13th place among the most important causes of death and in 3rd place in men who are older than 60 years as far as sudden death is concerned [1]. AAA (particularly a large one) is prone to rupture and if this occurs, every third patient will die before reaching the hospital. Moreover, the total mortality rate approaches 60–80% [4,5].

Aortic dissection (AoD) incidence is relatively low: 3–6 patients per 100,000 individuals annually [5,6], but the clinical picture is dramatic and the prognosis is grave. Mortality of AoD has been “exceedingly high” [7] and AoD has been among the deadliest diseases in emergency departments (ED) [6]. A significant number of AoD patients die prior to being admitted into hospital [8]. More than 1/4 of patients can die while in hospital even though they received appropriate treatment [6]. Moreover, Oxford Vascular Study reported that only 52.6% of patients with AoD who were hospitalized had managed to survive 30 days [8]. Therefore, it is very important to prevent such dangerous diseases.

Systemic arterial hypertension (HTN) has been a very important risk factor for Ao diseases. An overall prevalence of 0.3% has been identified during the screening for AAA in Japan in 1591 individuals and no less than 7.7% in patients with HTN [9]. In addition to smoking, male gender

and advanced age, HTN is one of the crucial factors for AAA etiopathogenesis. Additionally, HTN increases the risk of rupture by 30% for each 10 mm Hg mean BP elevation [10]. Consequently, HTN is a well-known risk factor for AAA formation, enlargement and rupture [10,11]. During the screening of >3,000,000 people for AAA risk factors, insufficiently controlled BP had been measured in more than half of the patients with diagnosed HTN [12].

The most prevalent risk factor for AoD is HTN, which has been repeatedly found in 2/3 to 3/4 of patients [5,7]. In the majority of such AoD patients, BP has been insufficiently controlled [7]. Therefore, aneurysm and dissection of Ao represent very important target organ damages (TOD) of HTN.

Accordingly, *aorta should be listed among target organs* in the relevant guidelines (having in mind their importance). To our surprise, this has not been the case. Although the current guidelines for HTN are very good and despite the obvious significance of Ao diseases (such as Ao aneurysm and AoD), as well as the evidence-based importance of HTN in their genesis, Ao complications of HTN have not been listed in contemporary HTN guidelines properly [13–17]. For example, the Joint National Committee (JNC-8) guidelines focused on HTN treatment and in the JNC-7 AoD was not mentioned as TOD of HTN [15,18,19]. There is no obvious explanation to cite the peripheral arterial disease as TOD and to omit the central arterial (aortic) disease (e.g., Table 6 in JNC-7) [15].

In the European Society of Cardiology (ESC) and European Society of Hypertension (ESH) 2013 HTN guidelines, the peripheral arteries are also listed in signs of TOD but Ao is not. In another place the authors recommend the ultrasound examination of the ascending Ao. Moreover, concerning HTN complications, there is a suggestion to search for Ao stiffness but not directly for Ao dilatation, aneurysm and (clinically unrecognized) dissection. For example, in Table 8.4 “History and symptoms of organ damage and cardiovascular disease”, Ao consequences of HTN were not mentioned. Moreover, in Table 9, under the subheading: “Signs of organ damage”, “Peripheral arteries” are mentioned but “Central arteries” (or „aorta”) are not. In 2018 Canadian guidelines for HTN, in the Examples of TOD (4th Supplemental Table) the authors listed „Peripheral artery disease” but Ao diseases were not mentioned [16]. In the pragmatic National Institute for Health and Care Excellence (NICE) 2011 HTN guidelines Ao complications were also not cited among TOD [17].

As prevention is better than cure, the HTN - AoD relationship, most probably a causal one, ought to be mentioned in the HTN guidelines. Regarding the diseases with a causal relationship such as HTN (usually the cause) and AoD (consequence), it is important to write about this in the guidelines for both diseases. It is even more important to write about it in the guidelines for HTN (cause), in order that we may become more aware that a preventive effort is required. Hopefully the new 2018 ESC ESH hypertension guidelines will correct these shortcomings.

The only guidelines that have specifically addressed Ao diseases are the 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guidelines [19]. There has been half a page dedicated to drug treatment of patients who have the Ao disease in addition to HTN. The authors correctly stated that studies of such patients had been missing and that target BP and the optimal antihypertensive medications have been unknown. They have recommended beta blockers [19]. Nevertheless, a lot of information about the aortic consequences of HTN, including the significance of HTN for Ao aneurysm and AoD, classification of Ao aneurysms and AoD as hypertensive TOD, recommendation when and how to screen for Ao aneurysms, as well as (scarce) information about the HTN characteristics that are risk markers for hypertensive Ao complications are missing.

Conclusion: As opposed to peripheral arterial disease, acute and chronic aortic complications of HTN have not been adequately listed in all the hypertension guidelines among the useful information in medical history, symptoms, hypertensive target organ damages and suggestions about which hypertensive lesion to look for. Having in mind the significance of the guidelines for the management of such an important

disease as HTN, we believe that an improvement in the elaboration of aortic hypertensive complications in the future guidelines about HTN will help better prevention.

### Acknowledgement

This work has been supported by the Serbian Ministry of Education and Science, grant No. III41018.

The author would like to thank Vesna Milovanovic, MD, Canterbury, UK, for the scientific edit of the manuscript.

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18 June 2018

<https://doi.org/10.1016/j.ajem.2018.06.047>

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### Penile fracture: Surgical vs. conservative treatment



Penile fracture is a relatively uncommon form of urologic trauma, and is defined as blunt direct trauma to the fully erect or semi-erect penis resulting in rupture of the tunica albuginea of the corpora cavernosa [1, 2]. Due to a sudden increase in the intracorporeal pressure, over-stretching of the tunica albuginea occurs, causing rupture. Involvement of or injury to the corpus spongiosum, urethra, dorsal nerve and vessels may also be present [3]. Patients typically present with symptoms including sharp penile pain, hearing a 'cracking' sound, rapid detumescence and swelling with or without ecchymosis of the penile shaft [4]. The penis also may be bent or angled toward the side contrary the injury, and ecchymosis may extend in to the scrotum and perineum, as well as the suprapubic and inguinal regions [4]. Treatments of penile fracture have ranged from conservative, including compression bandages, anti-inflammatory agents, and ice, to the more invasive and complicated surgical repair, and all involve a risk of complications [5]. Currently, immediate surgery for a fracture is recommended, and has been shown to be superior to non-operative treatment, however if it is an uncomplicated case without extensive hematoma or concomitant urethral injury, conservative therapy has been shown to yield equally good outcomes [6]. The purpose of this study was to review the injury patterns and treatment of penile fracture in a cohort of patients who presented to the emergency department (ED).

We conducted a retrospective cohort analysis of males presenting to the EDs of seven affiliated hospitals in West Michigan with a diagnosis of penile fracture. All eligible cases were seen between January 2005 and July 2017 (150 months). Patient demographics, presenting complaints, co-morbidities, radiographic studies, treatment in the ED, final disposition, and complications were recorded using a standardized abstraction forms. Operative notes were reviewed to define the extent of penile injuries, and time and type of definitive treatment. Descriptive statistics including frequency tables and confidence intervals were used to summarize the data.

During the study period, 32 patients presented to the ED with a penile fracture. Average age was  $37.2 \pm 12.1$  years with a range of 14 to 59 years. Average duration of symptoms was  $21.7 \pm 33.9$  h; range 1 to 120 h. Mechanism of injury included sexual maneuvers (66%), masturbation (13%), manipulation of erect penis (9%), rolling over (6%), and fall onto erect penis (6%). Characteristically, patients heard a cracking sound associated with sharp pain followed by immediate loss of the erection, deformity, discoloration and swelling of the soft tissues. The penis often took on a bizarre shape, with deviation of the penile shaft, usually to the side opposite the tear. The penis may be bent (88%) as well as have ecchymosis confined to Buck's fascia, resulting in massive edema as seen in 15 patients (46%). Additionally, injured fascial compartments lead to an extension of the ecchymosis into the scrotum, perineum, the suprapubic area and the inguinal regions. The ED diagnosis of penile fracture was made clinically in all cases, without the need for