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Commentary

Commentary on "Outcomes of Endovascular Stent Graft Repair for Penetrating Aortic Ulcers with or without Intramural Hematoma"

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ABSTRACT

Penetrating Aortic Ulcer (PAU) is a kind of Acute Aortic Syndrome (AAS). Compared with PAUs without IMH, PAUs with Intramural Hematoma (IMH) presented significant differences in history of acute coronary syndrome, pleural effusion, emergency admitted, aortic diameter at the PAU level, and the occurrence of Stent-Induced New Entry (SINE). The cumulative survival rates of patients undergoing endovascular repair during the first hospitalization were significantly higher than those having delayed repair during the follow-up.

Keywords: Penetrating aortic ulcer; Intramural hematoma; Endovascular therapy

DESCRIPTION

Penetrating Aortic Ulcer (PAU) is a subset of Acute Aortic Syndrome (AAS), defined as the rupture of atherosclerotic lesions through the internal elastic lamina of the aortic wall with subsequent hematoma formation between the media and the adventitia. Apart from PAU, AAS also included aortic dissection (AD) and intramural hematoma (IMH). Although the majority of PAUs are asymptomatic, the rupture rate for PAU presenting as AAS is higher than that for aortic dissection, with a reporting rupture rate as high as 38% [1, 2]. Meanwhile, Harris, et al. determined that 67% of PAU patients were associated with IMH [3], which was significantly associated with a progressive disease course and should be treated aggressively [4]. According to the guideline, endovascular therapy is the first-line therapy for these patients [5]. However, the difference between PAUs with and without Intramural Hematoma (IMH) with respect to presentations, clinical features, and outcomes after endovascular repair requires further elucidation.

In the context, we conducted this study aiming at demonstrating the long-term results of PAUs with or without IMH after endovascular repair. Among 1542 patients with AAS undergoing endovascular repair in our center during the past decade, 138 (8.9%) were PAUs. 58 (42.0%) of them were implicated with IMH. Indications for endovascular repair included persistent or recurrent pain (n=85), disease progression (n=43), aortic contained rupture (n=5), aortic free rupture (n=3), and embolic event (n=2). Meanwhile, compared with PAUs without IMH, PAUs with IMH presented significant differences in history of acute coronary syndrome, pleural effusion, emergency admitted, aortic diameter at the PAU level, and the occurrence of Stent-Induced New Entry (SINE). And SINE was defined as the new tear caused by the stent graft and excluding those arising from natural disease progression or iatrogenic injury from the endovascular manipulation [6]. During an average of 51.4 ± 36.5 months of follow-up, in-hospital mortality was 0, and 5-year survival rate was 90.3%. Previous studies found asymptomatic patients were less likely to progress or rupture, but with poorer prognosis once PAU progressed during the follow-up [3, 6]. In this study, we confirmed the cumulative survival rates of patients undergoing endovascular repair during the first hospitalization were significantly higher than those having delayed repair during the follow-up. There were no significant differences in mortality and freedom from reintervention between PAUs with and without IMH [7].

Despite all this, this study suffers from several limitations needs to be mentioned. First, this is a retrospective study with limited patients, which causes the conclusions less convincing. For PAUs, the most important issue was to identify those patients with high risk of progression and rupture. On one hand, for patients who are at high risk of progression, intervention could be performed promptly. Because the prognosis could be poorer after endovascular therapy once dissection or aneurysm developed. On the other hand, for patients who pose stable course undergo endovascular therapy, the complications are

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higher. Second, there are still some patients who have met the indication of repair but continue to be treated conservatively due to different reasons. What's more, we don't know how many PAUs never be treated or the proportion of all PAU cases that do need intervention. Hence, a contemporaneous control arm including the above patients receive conservative treatment is lacking, which would made us unable to confirm the benefits of the endovascular repair or the nature history of PAUs. In the future, studies concerning PAU should focus on identifying risk factors for progression and achieve individualized treatment.

In sum, this is a relatively large sample size demonstrating encouraging results in PAU patients treated by endovascular repair. However, prospective randomized controlled trials with appropriate follow-up are required to clarify the nature history of PAUs and indications of surgery due to aforementioned limitations.

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