Characteristics and Fate of Systemic Artery Aneurysms caused by Kawasaki Disease

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Presenter Disclosure Information

Shinsuke Hoshino, MD, PhD Characteristics and Fate of Systemic Artery Aneurysm caused by Kawasaki Disease

FINANCIAL DISCLOSURE:

We have no relevant financial relationship.

Introduction

Systemic artery aneurysms (SAA) develop in approximately 2% of untreated patients and 10% of patients with giant coronary aneurysm caused by KD.

Circulation. 1996;94 1379-85 Kato H et al

SAA refers to aneurysms developing anywhere in the arterial system other than the coronary circulation.



The objective of this study is to clarify the characteristics and fate of SAA caused by KD.

Methods

20 pts with SAA

between 1980 and 2013 at NCVC in Japan

- 1. The characteristics of the patients with SAA was analyzed, and the distribution and fate of SAA were also evaluated.
- 2. We analyzed the residual rate of SAA in the late period and the incidence of stenotic lesions in 11 pts with SAA who underwent an initial angiogram less than 4 months after the onset of KD.

Interval from the the onset of KD to respective angiogram



Patients



Treatment of acute KD



Treatment in the late period

n =20

Coumadin with antiplatelets 8 (40%)

Antiplatelets 12 (60%)

Cardiac events

15 pts (75%)

Acute myocardial infarction 7 pts (35%)

Coronary artery bypass grafting 8 pts (40 %)

Death

4 pts (20 %)

Distribution of SAA



Regression of aneurysm in brachial artery

6 months



6 years



Changes of aneurysms in brachial arteries



Regression of aneurysms in bilateral common and internal iliac arteries

20 months



13 years

Changes of aneurysms in bilateral iliac arteries and abdominal aorta

7 months

Abdominal angiogram after surgery

18 years

7 months old

Abdominal aorta aneurysm and bilateral common iliac aneurysms $\geq 30 \text{ mm}$

23 months old

Replacement using **artificial vessels**

Outcome of SAA in 17 pts

Residual rate of SAA in 11 pts (Brachial, common and iliac arteries)

Residual SAA included persistent aneurysms and stenotic lesions.

Cut-off values of the initial diameter leading to residual SAA

	Diameter (mm)	AUC	р
Brachial (n=14)	6.8	0.96	0.0003
Common Iliac (n=16)	9.4	0.75	0.0339
Internal Iliac (n=12)	10.7	0.92	0.0035

pts

- All patients with SAA had had acute KD within 20 months with a median of 6 months.
- All patients had at least one symmetric pair of aneurysms in bilateral peripheral arteries. Sixteen pts (80%) had multiple aneurysms.
- 3. The prevalence of SAA was high in the brachial, common and internal iliac arteries.

4. The incidence of cardiac events was high in patients with SAA.

5. The larger aneurysms persist into the late period, and the acute phase diameter of SAA leading to late stenotic lesions was more than 10.0 mm.

Conclusions

SAA occurred symmetrically and were multiple in younger infants and those with severe acute vasculitis. The fate of SAA resembles that of coronary artery aneurysms, and depends on its acute phase diameter. The larger SAA can lead to stenotic lesions in the late period.