

# Systemic Artery Aneurysms and Kawasaki Disease

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## abstract

**BACKGROUND:** Coronary artery aneurysms (CAAs) are a well-known complication of Kawasaki disease (KD), but there are no data on incidence or outcomes of systemic artery aneurysms (SAAs) in the current era.

**METHODS:** From April 1, 2016, to March 31, 2019, we screened for SAAs in 162 patients with KD at risk for SAAs with magnetic resonance angiography or peripheral angiography and analyzed incidence and early outcomes of SAAs.

**RESULTS:** Twenty-three patients had SAAs, demonstrating an incidence of 14.2% (23 of 162) in patients who were screened at 1 month after onset. The proportion of patients with SAAs was estimated to be 2% (23 of 1148) of all patients with KD. The median age at onset of KD with SAAs was 5 months. All patients with SAAs had CAAs, with z scores >8. Of patients with giant CAAs, 38.6% (17 of 44) had SAAs. A total of 129 SAAs occurred in 17 different named arteries. The most common sites for SAAs were the axillary (18.6%), common iliac (12.4%), and brachial (11.6%) arteries. During a median follow-up time of 6 months, 92.9% (79 of 85) of SAAs had some degree of regression, with 80% (68 of 85) of SAAs returning to normal. The overall regression rate was higher for medium to large SAAs than for medium to giant CAAs.

**CONCLUSIONS:** Although the incidence of SAAs may not be as dramatically reduced as we expected compared with previous data, SAAs have a high regression rate during short-term follow-up.

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Dr Liu conceptualized and designed the study and finalized the manuscript; Drs Qu-ming Zhao and Chu organized and conducted the project, undertook data management and data analysis, and drafted the report; Drs Wu, Liang, Sun, He, Lu Zhao, Wang, Huang, and Niu critically reviewed and revised the manuscript and contributed important intellectual content; and all authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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**WHAT'S KNOWN ON THIS SUBJECT:** Studies from the 1980s revealed that systemic artery aneurysms (SAAs) may occur in 0.8% to 2% of patients with Kawasaki disease at 3 months after onset. It is speculated that SAAs are now much less common than before.

**WHAT THIS STUDY ADDS:** SAAs occur in 14.2% of patients at risk for SAAs at 1 month after onset, giving an estimated proportion of patients with SAAs as 2% of all patients with Kawasaki disease. SAAs have a high regression rate during short-term follow-up.

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Kawasaki disease (KD) is a self-limiting panarteritis of unknown etiology that predominantly affects medium-sized arteries.<sup>1</sup> Although coronary artery aneurysms (CAAs) are well-recognized in KD, little is known about the features of systemic artery aneurysms (SAAs).<sup>2–4</sup> Since the advent of intravenous immunoglobulin (IVIg) therapy, almost all English-language literature on KD-related SAAs consist of case reports revealing that axillary, subclavian, brachial, iliac, and femoral arteries are common sites of involvement.<sup>5–19</sup> To date, there are only 2 reports from the same Japanese medical center in which authors reported the incidence of SAAs in patients with KD, but, in fact, less than half of their patients with KD were screened for SAAs; it was during their study that they found that only patients with giant CAAs developed SAAs.<sup>3,4</sup> Although it is a reasonable assumption that there is a concomitant decrease in SAA formation with early IVIg therapy, as well as a decrease in CAAs, there are no published data to support this. More importantly, aside from not screening all patients with KD, earlier studies of screening for SAAs at least 3 months after KD onset by angiography may also have underestimated SAA incidence.<sup>3,12</sup>

We should acknowledge that the impracticability of routine screening for SAAs in early-stage KD makes it difficult to obtain a true incidence of SAAs. Nevertheless, on the basis of the current view that SAAs are almost always associated with giant CAAs, and to increase the detection rate of SAAs as much as possible, we clinically identified giant CAAs, progression of coronary artery lesions, and refractory KD that did not respond to 2 doses of IVIg as risk factors for developing SAAs, and we screened patients with these comorbidities. Together with short-term follow-up, we hope to provide a certain reference for the

current incidence and early outcome of SAAs.

## METHODS

### Patients and Methods

We prospectively collected medical records of all patients with KD admitted to the Children's Hospital of Fudan University from April 1, 2016, to March 31, 2019. During this period, we screened for SAAs in patients at risk for SAAs with full-body magnetic resonance angiography (MRA) or peripheral angiography (PA). Within 2 months of onset, MRA was performed if any of the following risk factors were present: (1) development of giant CAAs, (2) progression of coronary artery lesions during hospitalization despite not meeting criteria for giant CAAs, and (3) refractory KD that failed to respond to 2 IVIg doses. Two months after onset, PA was performed at the same time as coronary angiography, which was indicated for those with medium or giant coronary aneurysms or those who exhibited findings of myocardial ischemia. Demographic, clinical, and imaging data of patients with SAAs were collected for analysis. This study was approved by the ethics committee of the Children's Hospital of Fudan University. Written informed consent was obtained from the parents of participating patients.

### Diagnostic Criteria

KD was diagnosed according to Japanese Circulation Society guidelines.<sup>20</sup> A *z* score cut point of  $\geq 2.5$  was used to define CAA.<sup>1</sup> The *z* score was calculated by using the Boston Children's Hospital *z* score calculator (<http://zscore.chboston.org/>). CAA size was classified according to the 2017 American Heart Association proposal: small (*z* score  $\geq 2.5$ – $<5$ ), medium (*z* score  $\geq 5$ – $<10$ ), and giant (*z* score  $\geq 10$  or absolute dimension  $\geq 8$  mm).

SAA was defined as at least a 50% increase in diameter compared with the expected normal diameter, which is the most commonly used and recommended definition.<sup>1,21</sup> Because there is no gold standard for the classification of SAA size, the method we used in this study is similar to that recommended by Japanese guidelines for CAAs,<sup>20</sup> with some modifications: the diameter of small aneurysms was defined as 1.5 to 2.5 times that of an adjacent normal segment, the diameter of medium aneurysms was defined as  $>2.5$  to 4 times that of an adjacent normal segment, and the diameter of large aneurysms was defined as  $>4$  times that of an adjacent normal segment. Meanwhile, rheumatologic, infectious, and hematologic evaluations were conducted to rule out other etiologies of SAAs.

### Short-term Follow-up

Patients with SAAs were followed-up during the convalescence phase by PA; the timing depended on the severity of the coronary artery lesions, but patients were followed-up at least 2 months after onset.

### Statistical Methods

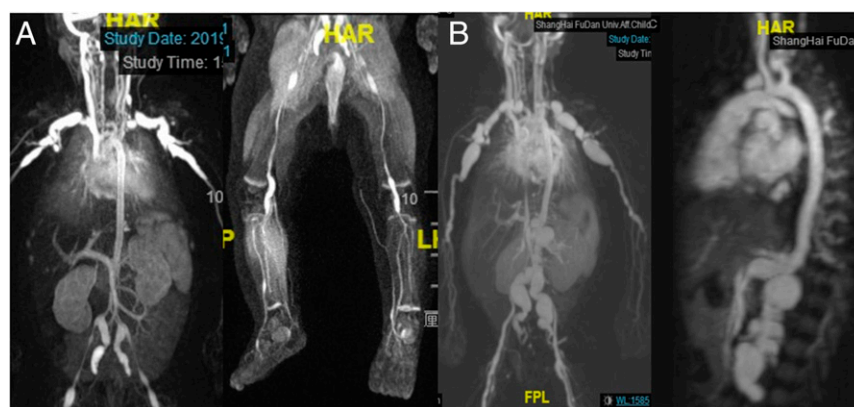
The clinical characteristics were summarized as counts and percentages or medians with ranges, depending on the type of variable. The differences between groups were compared by using the  $\chi^2$  test for categorical variables and the Mann-Whitney *U* test for continuous variables. A probability value of  $P < .05$  was considered significant.

## RESULTS

A total of 1148 patients with KD were seen for the first time during the study period, 218 (19%) of whom had CAAs. Within 2 months of onset, 110 patients underwent MRA to screen for SAAs, and another 52 patients who met the criteria for coronary angiography underwent PA at least 2 months after onset. Initial

assessment of SAAs was performed between 10 and 155 days (median 30 days) after onset. Different coronary artery lesions and correspondingly detected SAAs are shown in Fig 1.

Twenty-three patients (16 boys, 7 girls) had SAAs, giving an incidence of 14.2% (23 of 162) in patients who were screened. The proportion of patients with SAAs was estimated to be 2% (23 of 1148) of all patients with KD. Of these patients, 8 had large SAAs, 11 had medium SAAs, and 4 had small SAAs (Fig 1). All these patients underwent physical examination, and only 4 (17.4%) patients were found to have SAA-related abnormalities, one of whom had a weakened radial pulse and 3 of whom had pulsatile masses in the axillae. SAAs in 22 (95.7%) patients were found by MRA (Figs 2 and 3), whereas only 1 patient who was first seen 5 months after onset had SAAs found by PA (Fig 1). All patients with SAAs had concomitant CAAs (17 giant, 6 medium) with  $z$  scores  $>8$  and were anticoagulated with warfarin and aspirin. Of patients with



**FIGURE 2**

Two cases of diffuse SAAs. A, Patient 19, a 2-month-old girl at 1 month after onset of KD who was recovering well. B, Patient 1, a 6-month-old boy 20 days after the onset of KD who died of a ruptured CAA in the hospital, which was confirmed by autopsy.

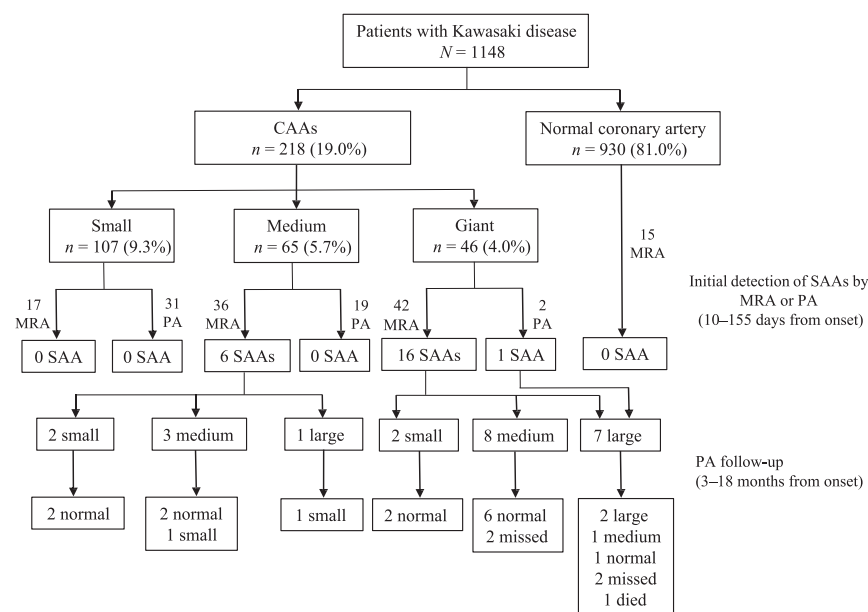
giant CAAs, 38.6% (17 of 44) had SAAs.

Compared with patients with CAAs without SAAs, patients with SAAs had a younger median age at onset (5 months [range 1–20 months] versus 15 months [range 1–160 months];  $P < .001$ ) and a longer duration of fever (12 days [range 7–30 days] versus 8 days [range 5–38 days];  $P < .001$ ), but there were no significant difference in the ratio of boys to girls (16:7 vs 145:50;  $P =$

.621) or in the median time of IVIg initiation (day 7 [range 5–20] versus day 6 [range 4–35];  $P = .294$ ).

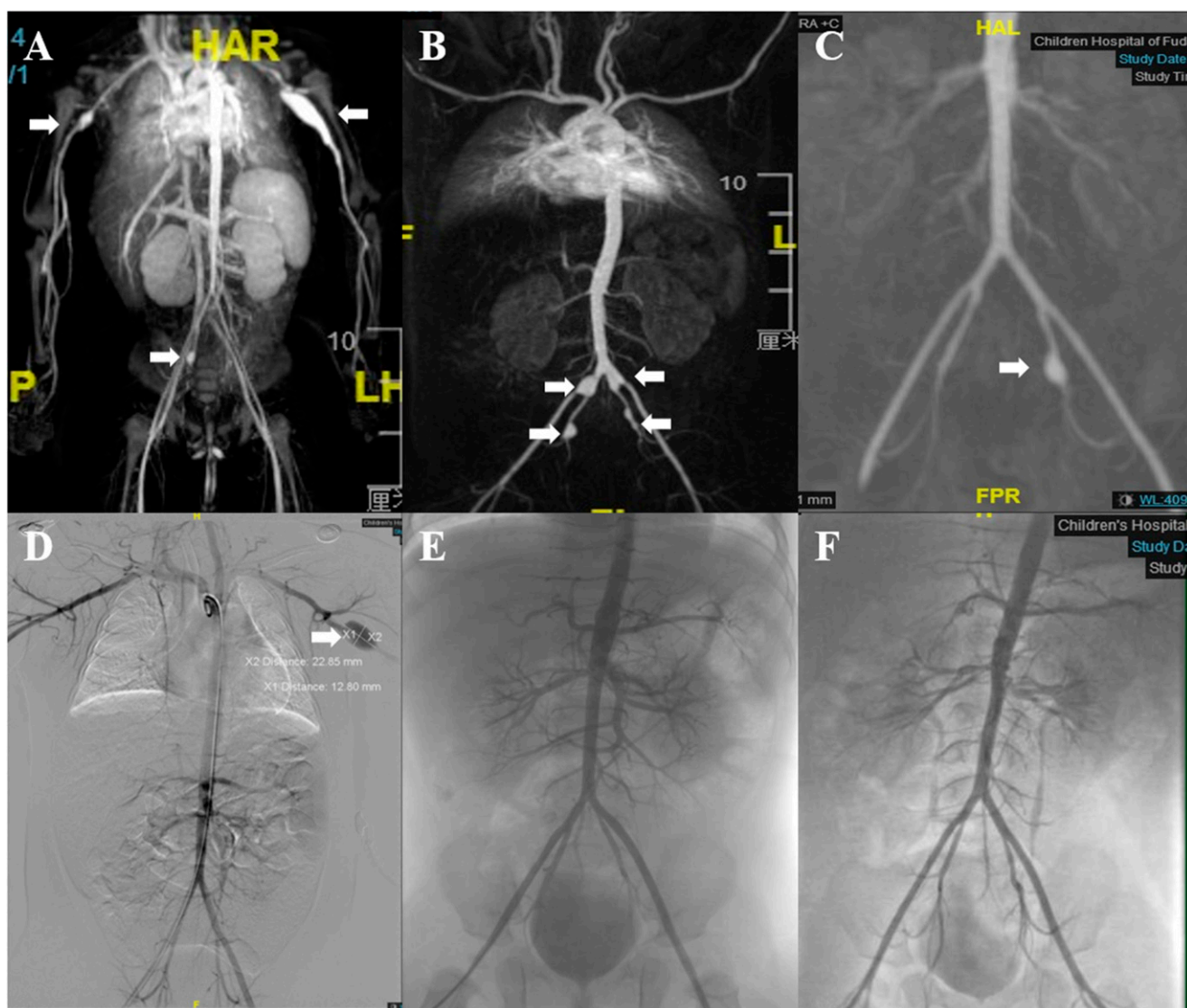
The size and distribution of SAAs in each patient are shown in Table 1. A total of 129 SAAs were found among the 23 patients (mean 5.6 SAAs per patient affected). The SAAs occurred in 17 different named arteries. The 5 most common sites for SAAs were the axillary artery (18.6%), common iliac artery (12.4%), brachial artery (11.6%), internal iliac artery (10.9%), and subclavian artery (10.1%). Similarly, axillary arteries were involved in 13 (56.5%) patients, common and internal iliac arteries were involved in 9 (39.1%) patients each, and subclavian and brachial arteries were involved in 8 (34.8%) patients each. Twenty-one (91.3%) patients had multiple SAAs. SAAs were found bilaterally in 78.3% (47 of 60) of symmetrically paired arteries.

A total of 18 patients with 85 SAAs were managed for a median time of 6 months (range 3–18 months) (Figs 1 and 4), and 92.9% (79 of 85) of SAAs had some degree of regression, with 80% (68 of 85) regressing to normal (Figs 3 and 4). Similarly, aneurysm diameters returned to normal in all patients with small SAAs. The regression rates of varying degrees of medium and large SAAs



**FIGURE 1**

Study profile. Two patients with giant CAAs declined to be screened for SAAs.



**FIGURE 3**

Short-term follow-up of patients with SAAs. A and D, Patient 11: only the left axillary artery aneurysm was visible 4 months after onset. B and E, Patient 6: all SAAs returned to normal. C and F, Patient 7: the left internal iliac aneurysm regressed to normal.

were 100% (9 of 9) and 60% (3 of 5), respectively (Fig 3). During the same period, only 2 of the 5 patients with medium CAAs had their diameters normalized; of the remaining 3, 1 had a reduction and 2 had no change. Of the 13 patients with giant CAAs, the diameter was unchanged in 9 and reduced in 4. The overall regression rate was higher for medium to large SAAs than for medium to giant CAAs (12 of 14 vs 7 of 18;  $\chi^2 = 7.158$ ;  $P = .007$ ). No angiographically visible stenosis was noted during follow-up.

## DISCUSSION

Although the characteristics and natural history of CAAs after KD are well-documented in many studies, little literature has been published on the distribution and frequency of SAAs. Studies on the incidence of SAAs after KD have been limited to the pre-IVIg treatment era, when Japanese researchers reported a 0.8% to 2% incidence of angiographically detected SAAs 3 months after onset.<sup>3,4</sup> Of 217 consecutive patients with KD who were screened by PA in

the first 6 years of their study period, only 3 (1.4%) had SAAs, and all of them had giant CAAs. Thus, PA was performed only in patients with giant CAAs during the subsequent 5 years, and 10 (2.6%) patients out of all 377 patients with KD were found to have SAAs. Even on the basis of their results, it is still not possible to assert that SAAs do not occur in patients with normal coronary arteries. However, many case reports in the current era have also revealed that SAAs are almost always associated with giant CAAs.<sup>5-7,11,14,16,22,23</sup> With



**TABLE 1** The Distribution and Size of SAAs

No.	Sex	Age at Onset, mo	Initial Assessment After Onset, d <sup>a</sup>	Subclavian		Axillary		Brachial		Radial		Ulnar		Common Carotid		Renal		Common Iliac		Internal Iliac		External Iliac		Femoral		Popliteal		Anterior Tibial		BCT	AA	SM	CT	
				Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left					
1	Male	5.1	27	L	L	L	L	L	L	S	S	—	—	L	M	M	M	L	L	L	L	M	M	S	S	M	M	—	—	M	M	L	L	
2	Male	17.2	30	—	—	—	—	—	—	—	—	—	—	—	—	S	M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
3	Male	3.6	155	—	—	L	L	—	—	—	—	—	—	—	—	—	—	—	—	L	—	—	—	—	—	—	—	—	—	—	—	—	—	
4	Male	6.9	19	—	—	—	—	—	—	—	—	—	—	—	—	—	—	M	M	L	S	—	—	—	—	—	—	—	—	—	—	—	—	
5	Male	11.7	30	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	M	—	—	—	—	—	—	—	—	—	—	—	—	—	
6	Male	10.4	61	—	—	M	L	—	—	—	—	—	—	—	—	—	—	M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
7	Female	1.8	19	—	—	—	M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
8	Male	6.4	10	—	—	—	—	—	—	—	—	—	—	—	—	—	—	L	M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
9	Male	5.1	30	—	—	—	L	M	—	—	—	—	—	—	—	—	—	—	—	M	—	—	—	—	—	—	—	—	—	—	—	—	—	
10	Female	6.0	35	—	—	—	—	S	S	—	—	—	—	—	—	—	—	M	M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
11	Female	2.0	30	M	M	M	M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	S	—	S	S	
12	Female	2.4	18	—	—	M	M	—	—	—	S	S	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
13	Male	6.5	15	—	—	M	—	—	—	—	—	—	—	—	—	—	—	—	—	M	M	—	—	—	—	—	—	—	—	—	—	—	—	
14	Male	4.1	45	S	S	M	M	S	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
15	Male	4.0	30	—	—	M	M	—	—	—	—	—	—	—	—	—	—	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
16	Male	2.5	23	M	M	M	M	—	—	—	—	—	—	—	—	S	S	L	L	L	L	—	—	—	—	—	—	—	—	—	—	—	—	
17	Female	1.0	35	M	M	L	L	M	L	—	—	—	—	M	M	S	S	L	L	L	L	S	S	M	S	M	M	S	S	S	S	S		
18	Male	15.8	35	—	—	—	—	—	—	—	—	—	—	—	—	—	—	M	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
19	Male	20.7	14	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
20	Female	2.3	20	S	—	—	—	S	—	—	—	—	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
21	Female	14.4	35	—	—	—	—	S	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
22	Male	3.5	22	—	S	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
23	Male	8.0	22	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
Total SAAs				13	24	15	15	4	3	4	8	16	14	—	4	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	3	5	3	3

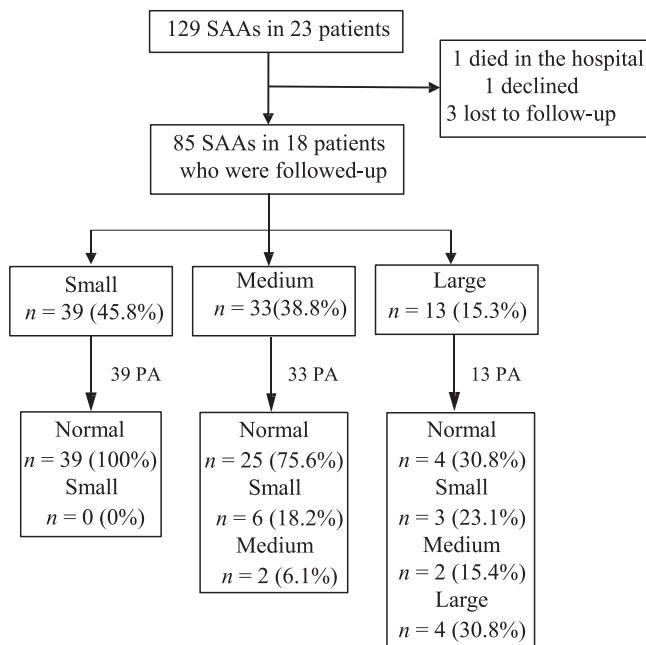
AA, abdominal aorta; BCT, brachiocephalic trunk; CT, celiac trunk; L, large aneurysm; M, medium aneurysm; S, small aneurysm; SM, superior mesenteric; —, not applicable.

<sup>a</sup> MRA was used as an initial evaluation in all patients except patient 3, who underwent direct PA because he was in the convalescence phase.

these data, it is reasonable to assume that even screening patients with giant CAAs may in part reflect the incidence of SAAs in all patients with KD.

Our study reveals that incidence of SAAs in the patients who were screened was 14.2% (23 of 162). Even when taking the number of all patients with KD as the denominator, the incidence can be as high as 2% (23 of 1148). The latter at least suggests that improved treatment of KD may be able to reduce the incidence of SAAs but perhaps not as dramatically as we suspected.<sup>14</sup> We also demonstrated that all SAAs in the patients who were screened were associated with CAAs with z scores >8, 73.9% (17 of 23) of which were giant CAAs. Moreover, larger CAAs indicated a higher risk of developing SAAs, and, similarly, larger SAAs were also associated with a higher proportion of larger CAAs (Fig 1). We confirmed that most of the SAAs were detected during the first year of life,<sup>3</sup> whereas the patients in this group were even younger, with a median age of 5 months. Previous studies have suggested SAAs most commonly develop in the subclavian, brachial, axillary, and iliac arteries,<sup>3,4,12</sup> which is similar to our results. We also found that SAAs occurred in the radial, ulnar, popliteal, and anterior tibial arteries, which have not been reported in the past. In addition, most SAAs in this study were multiple and had a symmetrical and bilateral distribution.

Literature on the outcome of SAAs is even rarer. The only study reported on the long-term outcome of SAAs revealed a 51% regression rate of SAAs at 20 years after onset.<sup>12</sup> However, the inclusion criteria were defined by the presence of SAAs on angiography at 3 months after onset, so it is likely that some SAAs would have resolved by this time, thus underestimating the true regression rate. More importantly, the outcome of SAAs may differ in the current era



**FIGURE 4**  
Follow-up of SAAs.

of IVIg therapy. In this study, we confirm the previous suspicion that 85% (68 of 80) of SAAs regress to normal at 6 months after onset. If the 12 patients with a median follow-up time of 3 months were analyzed alone, up to 81.3% (39 of 48) of SAAs would have also regressed to normal. In addition, we found that, similar to CAAs, the regression rate of SAAs correlated with the size of SAAs (Fig 2) but was higher than that of CAAs.

Although only 23 patients with SAAs were included in this study (because of the rarity of the disease), it is still the largest sample size we are aware of, and may, in part, reflect the current incidence and early outcome of SAAs. Early screening of SAAs by full-body MRA in patients with medium or giant CAAs can detect clinically significant SAAs more promptly than PA,<sup>6,8,24</sup> which is essential for the management of patients with KD. Careful physical examination for pulsatile masses by palpation of the axillary, brachial, abdominal, and inguinal regions is also important.<sup>6</sup>

This study has some important limitations. First, as mentioned before, in reality, it is difficult to screen all patients with KD for SAAs to obtain true incidence. Secondly, the high incidence of CAAs (19%) in this study may be related, in part, to the fact that this was the statistical result in the acute phase of KD (1–2 weeks after onset) but may also be due to the fact that our center is a tertiary referral hospital. Thus, the incidence results of SAAs may not be fully representative of other institutions. Finally, patients in this study were only followed-up for a short period of time; long-term follow-up is necessary to assess the risk of and risk factors for SAAs progression to obstructive lesions or to cause ischemic symptoms.

## CONCLUSIONS

With this study, we are the first to report the incidence and early outcomes of SAAs in the current IVIg era, showing that although the incidence of SAAs may not be as dramatically reduced as we expected,

SAAs have a relatively high regression rate, which correlated with the size of SAAs and was higher than that of CAAs during short-term follow-up. Early screening for SAAs by noninvasive imaging modalities should be considered in patients with KD with medium or giant CAAs.

## ABBREVIATIONS

CAA: coronary artery aneurysm  
IVIg: intravenous immunoglobulin  
KD: Kawasaki disease  
MRA: magnetic resonance angiography  
PA: peripheral angiography  
SAA: systemic artery aneurysm

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