Clinical characteristics of intracranial aneurysms in elderly patents over 70 years old: a retrospective observational study

Ruiqi Chen, Dingke Wen, Anqi Xiao, Rui Guo, Chao You and Yi Liu*

Abstract

Background: Although the characteristics of intracranial aneurysms (IAs) in different age groups have been well documented, they remain relatively unclear in elderly patients due to a lack of large sample studies.

Methods: Data from IA patients aged more than 70 years who were treated in our centre from January 2016 to January 2020 were retrospectively collected.

Results: A total of 290 elderly patients (75.9% female) with a mean age of 74.0 ± 4.7 years were analysed. Rupture occurred in 60.7% of patients, 38.6% of whom presented with meningeal irritation, and seizures were noted in 2.3%. A total of 48.9% of the patients with ruptured IAs had initial symptoms presenting with slow development, and the mean delay from ictus was prolonged to 264.2 ± 914.0 hours. In addition, 61.9% of the patients with ruptured IAs had lesions with a maximum diameter of less than 5 mm. A total of 30.3% of the patients had multiple aneurysms, 35.5% had aneurysms with irregular shapes and 54.8% had cerebrovascular atherosclerotic stenosis (CAS). Pulmonary infection (n = 138, 47.6%), hydrocephalus (n = 72, 24.8%), and thrombosis (n = 35, 12.1%) were common complications during hospitalization. By the end of the 1-year follow-up, 22.1% of the patients had unfavourable clinical outcomes, and the mortality rate was 23.4%.

Conclusions: Several characteristics regarding IAs in elderly patients were reported, including an obvious female predominance; mild, slow initial symptom development causing prolonged admission delay; a low incidence of meningeal irritation and seizures due to decreased electrophysiological activity of the neurons; increased percentages of CAS, multiple aneurysms, and aneurysms with daughter sacs causing a high risk of rupture even for small lesions; a high risk of complications during hospitalization; and relatively poor clinical outcomes.

Keywords: Intracranial aneurysms, Elderly patients, Clinical characteristics, Craniotomy treatment, Endovascular treatment

Background

Intracranial aneurysm (IA) is a devastating cerebral vascular disease and a major threat to health worldwide. According to previous epidemiological reports, unruptured IAs are found in approximately 3.2% of the adult population worldwide, with the average annual incidence of IA-related subarachnoid haemorrhage (SAH) among people of all ages ranging from 6 to 27.63 per 100,000 persons [1, 2]. With the general ageing of the population and advances in neuroimaging diagnosis, an increasing number of elderly patients are being diagnosed with IA [3, 4]. A Japanese cohort study on the natural course of cerebral aneurysms reported that 28% of patients with unruptured IAs were older than 70 years [5]. However,
due to a lack of large-sample studies, the overall clinical characteristics of IAs in elderly patients have yet to be fully elucidated. The present study was conducted to clarify the clinical characteristics of elderly patients (more than 70 years old) with IAs, to reveal some new findings and to assist in the choice of best clinical decision for these patients.

Methods

Study design

We performed a retrospective analysis in a single tertiary centre. This study was approved by the institutional review board (IRB) of West China Hospital. We used the hospital information system to collect the related medical data of elderly patients with IAs from January 2016 to January 2020. The inclusion criteria were as follows: (1) IA as identified by discharge diagnoses using the key words “intracranial aneurysm” or “aneurysmal subarachnoid haemorrhage”; IAs were diagnosed through computed tomography angiography (CTA) or digital subtraction angiography (DSA), and SAH was confirmed by the results of initial computerized tomography (CT) scan by experienced neuroimaging specialists; and (2) age more than 70 years. Two experienced neurosurgeons and two neuroradiologists who were blinded to the medical records were invited to confirm the diagnoses of the selected patients. Patients with an incorrect diagnosis or incomplete medical profiles were excluded.

Data collection and grouping

The following patient baseline information was retrieved from the hospital information system: age, sex, smoking, alcohol consumption, history of hypertension (treatment), anaemia, hypoproteinaemia, diabetes mellitus, electrolyte disturbance, coronary disease, hyperlipidaemia, haemorrhagic or ischaemic stroke, encephalatrophy and pulmonary infection, clinical presentations (including symptoms and signs), Glasgow Coma Scale (GCS) score on admission and upon discharge, Hunt-Hess grade, and duration from ictus to admission. Neuroradiological data were collected from the hospital imaging system to evaluate the angiographic metrics of the IAs, including their size (aneurysms with diameters larger than 25 mm were considered giant, and those between 15 and 25 mm were considered large), morphology and location and the presence of multiple aneurysms, based on the results of CTA or DSA. Vascular anomalies were also recorded, mainly including cerebrovascular atherosclerotic stenosis (CAS) and cerebral artery variants in the posterior cerebral artery (PCA), defined as foetal-type posterior cerebral artery (FTP). Irregular aneurysms were defined as the presence of a daughter sac in addition to the main saccular aneurysm. Treatment approaches were documented, and the occurrence of complications was tracked from the daily medical course record during hospitalization. Follow-up information was documented from clinic visits or telephone interviews at discharge and 1 month, 3 months, 6 months and 1 year after discharge. Patient clinical outcomes were measured by GCS upon discharge and modified Rankin scale (mRS) scores during follow-up. GCS scores ≥13 and mRS scores of 0–2 were considered favourable outcomes. The patients were divided into ruptured and unruptured groups, and comparisons were made between the two groups.

Statistical analysis

SPSS statistical software (version 22.0; SPSS Inc., Chicago, Illinois, USA) was used for all statistical analyses. The mean ± standard deviation (SD) was reported for quantitative data. Categorical data are expressed as frequencies and percentages. Bivariate analyses were conducted using the chi-square test or Fisher's exact tests, Student's t-tests, and Mann–Whitney U-tests as appropriate. Significance was defined as P<0.05, and 95% confidence intervals (CIs) were calculated for each variable.

Results

Baseline characteristics

A total of 2730 patients with IAs were treated in our hospital from January 2016 to January 2020; among them, 290 elderly patients (≥70 years old on admission) (10.6%) were analysed in the current study. The mean age of the patients was 74.0 ± 4.7 years (range 70 to 95 years old); a total of 220 (75.9%) patients were female. Patient demographic information and history of previous illness are shown in Table 1. Compared with patients in the unruptured group (n=114, 39.3%), patients in the ruptured group (n=176, 60.7%) were significantly older (P<0.001) and had a significantly higher percentage of patients with hypoproteinaemia (P<0.001), anaemia (P<0.001), diabetes mellitus (P<0.001), electrolyte disturbance (P=0.001), pulmonary infection (P=0.001), coronary disease (P=0.005), brain atrophy (P=0.032) and ischaemic stroke (P=0.009). The unruptured group had a significantly higher percentage of patients receiving routine treatment for hypertension than the ruptured group (P=0.024).

Clinical presentation

Elderly patients with ruptured aneurysms

For the 176 (60.7%) patients who had ruptured aneurysms, the attacks commonly occurred in the morning after waking (n=49, 27.8%). Common inducements included straining during defecation (n=24, 13.6%), emotional fluctuation (n=15, 8.5%) and sneezing (n=8, 4.5%). Presentations included headache (n=148, 84.1%),
vomiting (n = 118, 67.0%), loss of consciousness (n = 64, 36.4%), dizziness (n = 22, 12.5%) and hemiparesis (n = 20, 11.4%), while only 4 patients (2.3%) presented with seizures. Meningeal irritation (n = 68, 38.6%) was the most common sign, followed by cranial nerve deficits (n = 22, 12.5%). Slow, progressive development of initial symptoms was documented in 86 (48.9%) patients rather than the typical pattern of sudden onset. Consequently, the mean delay from ictus to admission was prolonged to 264.2 ± 914.0 hours. On admission, a total of 130 patients (73.9%) had good clinical performance (Hunt-Hess grade I-III) (Table 2).

Elderly patients with unruptured aneurysms
Among the remaining 114 (39.3%) patients with unruptured aneurysms, dizziness was the most common symptom (n = 42, 36.8%), followed by headache (n = 24, 21.1%), limb weakness (n = 14, 12.3%) and vomiting (n = 6, 5.3%). The most common sign was cranial nerve deficits (n = 28, 24.6%) (Table 3).

Radiological characteristics
Other radiological characteristics of the patients are shown in Table 4. The distribution of modified Fisher grade for the 176 ruptured cases (60.7%) showed that grades 3 and 4 accounted for 60.8% (n = 107) of all ruptured cases. Multiple aneurysms were diagnosed in 88 patients (30.3%). The mean size of the aneurysms (largest diameter) was 5.8 ± 4.6 mm. Notably, of the 176 patients with ruptured lesions, 109 (61.9%) had lesions with a largest diameter of less than 5 mm. In addition, 103

---

### Table 1  Baseline information of elderly patients with intracranial aneurysms

| Characteristics          | All combined (n = 290) | Ruptured (n = 176) | Unruptured (n = 114) | P value |
|--------------------------|------------------------|--------------------|----------------------|---------|
| Age (SD)/years old       | 74.0 ± 4.7             | 74.5 ± 4.8         | 73.3 ± 4.3           | 0.023   |
| Female patients, (%)     | 220/290 (75.9)         | 132/176 (75.0)     | 88/114 (77.2)        | 0.670   |
| Smoking, (%)             | 25/290 (8.6)           | 15/176 (8.5)       | 10/114 (8.8)         | 0.941   |
| Alcohol consumption, (%) | 15/290 (5.2)           | 6/176 (3.4)        | 9/114 (7.9)          | 0.158   |
| Hypertension, (%)        | 228/290 (78.6)         | 144/176 (81.8)     | 84/114 (73.7)        | 0.133   |
| Regular treatment of hypertension, (%) | 198/228 (86.8)     | 119/144 (82.6)     | 79/84 (94.0)         | 0.024   |
| Hypoproteinaemia, (%)    | 171/290 (59.0)         | 130/176 (73.9)     | 41/114 (36.0)        | <0.001  |
| Anaemia, (%)             | 152/290 (52.4)         | 119/176 (67.6)     | 33/114 (28.8)        | <0.001  |
| Diabetes mellitus, (%)   | 156/290 (53.8)         | 133/176 (75.6)     | 23/114 (20.2)        | <0.001  |
| Electrolyte disturbance, (%) | 143/290 (49.3)   | 113/176 (64.2)     | 30/114 (26.3)        | <0.001  |
| Hyperlipidaemia, (%)     | 36/290 (12.4)          | 17/176 (9.7)       | 10/114 (8.8)         | 0.183   |
| Pulmonary infection, (%) | 110/290 (37.9)         | 80/176 (45.5)      | 30/114 (26.3)        | 0.001   |
| Coronary disease, (%)    | 38/290 (13.1)          | 31/176 (17.6)      | 7/114 (6.1)          | 0.005   |
| Brain atrophy, (%)       | 32/290 (11.0)          | 25/176 (14.2)      | 7/114 (6.1)          | 0.032   |
| Ischaemic stroke, (%)    | 98/290 (33.8)          | 72/176 (40.9)      | 26/114 (22.8)        | 0.009   |
| Haemorrhagic stroke, (%) | 8/290 (2.8)            | 6/176 (3.4)        | 2/114 (1.8)          | 0.636   |

Elderly patients: age ≥ 70 years old; SD standard deviation

---

### Table 2  Clinical presentations of elderly patients with ruptured intracranial aneurysms

| Characteristics                        | All combined | % |
|----------------------------------------|--------------|---|
| Ruptured patients                      | 176/290      | 60.7 |
| Symptoms slow development              | 86/176       | 48.9 |
| Headache                               | 148/176      | 84.1 |
| Unconsciousness                        | 64/176       | 36.4 |
| Vomiting                               | 118/176      | 67.0 |
| Dizziness                              | 22/176       | 12.5 |
| Hemiparesis                            | 20/176       | 11.4 |
| Seizure                                | 4/176        | 2.3 |
| Meningeal irritation                   | 68/176       | 38.6 |
| Cranial nerve deficit                  | 22/176       | 12.5 |
| Admission delay (SD)/hours             | 264.2 ± 914.0| – |
| GCS (SD)                               | 11.9 ± 4.1   | – |
| Good clinical presentation             | 130/176      | 73.9 |

Elderly patients: age ≥ 70 years old; SD standard deviation; GCS Glasgow Coma Score; Good clinical presentation: Hunt-Hess grade I-III

---

### Table 3  Clinical presentations of elderly patients with unruptured intracranial aneurysms

| Characteristics                        | All combined | % |
|----------------------------------------|--------------|---|
| Unruptured patients                    | 114/290      | 39.3 |
| Dizziness                              | 42/114       | 36.8 |
| Headache                               | 24/114       | 21.1 |
| Limb weakness                          | 14/114       | 12.3 |
| Vomiting                               | 6/114        | 5.3 |
| Cranial nerve deficit                  | 28/114       | 24.6 |

Elderly patients: age ≥ 70 years old; SD standard deviation
(35.5%) patients had irregular lesions with daughter sacs. Furthermore, CAS, brain atrophy and FTP were recorded in 159 (54.8%), 32 (11.0%) and 39 (13.4%) patients, respectively.

Compared with the unruptured group, the ruptured group had significantly higher percentages of patients with irregular aneurysms ($P < 0.001$), CAS ($P < 0.001$), and FTP ($P = 0.001$). In addition, the mean lesion diameter in the ruptured group was significantly smaller ($P < 0.001$), and the percentage of patients with small aneurysms was significantly higher than that in the unruptured group ($P < 0.001$).

**Treatment and complications during hospitalization**

The patients’ treatment modalities and incidence of complications are shown in Table 5. A total of 181 patients (62.4%) were treated by surgical intervention, including 91 (31.4%) with craniotomy clipping and 90 (31.0%) with

| Characteristics       | All combined ($n = 290$) | Ruptured ($n = 176$) | Unruptured ($n = 114$) | $P$ value |
|-----------------------|--------------------------|---------------------|------------------------|-----------|
| Craniotomy, (%)       | 91/290 (31.4)            | 75/176 (42.6)       | 16/114 (14.0)          | <0.0001   |
| Endovascular, (%)     | 90/290 (31.0)            | 33/176 (18.8)       | 57/114 (50.0)          | <0.0001   |
| Conservative, (%)     | 109/290 (37.6)           | 68/176 (38.6)       | 41/114 (36.0)          | 0.646     |
| Pulmonary infection, (%)| 138/290 (47.6)          | 83/176 (47.2)       | 55/114 (48.2)          | 0.856     |
| Hydrocephalus, (%)    | 72/290 (24.8)            | 43/176 (24.4)       | 29/114 (25.4)          | 0.846     |
| Thrombosis, (%)       | 24/290 (8.2)             | 20/176 (11.4)       | 4/114 (3.5)            | 0.031     |
| Rebleeding, (%)       | 23/290 (7.9)             | 14/176 (8.0)        | 9/114 (7.9)            | 0.985     |
| Gastrointestinal bleeding, (%)| 16/290 (5.5) | 8/176 (4.5)    | 8/114 (7.0)           | 0.368     |
| Seizure, (%)          | 5/290 (1.7)              | 2/176 (1.1)         | 3/114 (2.6)            | 0.622     |
| Blood vasospasm, (%)  | 8/290 (2.8)              | 4/176 (2.3)         | 4/114 (3.5)            | 0.794     |
| Intracranial infection, (%) | 15/290 (5.2)  | 9/176 (5.1)       | 6/114 (5.3)            | 0.955     |

Elderly patients: age $\geq$ 70 years old

**Table 4** Radiological characteristics of elderly patients with intracranial aneurysms

| Characteristics       | All combined ($n = 290$) | Ruptured ($n = 176$) | Unruptured ($n = 114$) | $P$ value |
|-----------------------|--------------------------|---------------------|------------------------|-----------|
| SAH + ICH, (%)        | –                        | 29/176 (16.5)       | –                      | –         |
| Modified Fisher grade I-II, (%) | –         | 69/176 (39.2)       | –                      | –         |
| Modified Fisher grade III-IV, (%) | –              | 107/176 (60.8)      | –                      | –         |
| With irregular aneurysms, (%) | 103/290 (35.5) | 88/176 (50.0)       | 15/114 (13.2)          | <0.0001   |
| CAS, (%)              | 159/290 (54.8)           | 121/176 (68.8)      | 38/114 (33.3)          | <0.0001   |
| FTP, (%)              | 39/290 (13.4)            | 33/176 (18.8)       | 6/114 (5.3)            | 0.001     |
| With multiple aneurysms, (%) | 88/290 (30.3)  | 51/176 (29.0)       | 37/114 (32.5)          | 0.529     |
| Dissecting aneurysms, (%) | 12/290 (4.1)       | 7/176 (4.0)         | 5/114 (4.4)            | 0.896     |
| Lesion diameter (SD)/mm | 5.8 ± 4.6              | 5.1 ± 3.3           | 6.8 ± 6.0              | <0.0001   |
| Small-size aneurysm, (%) | 158/290 (54.5)       | 113/176 (64.2)      | 45/114 (39.5)          | <0.0001   |
| Anterior circulation, (%) | 280/290 (95.6)   | 172/176 (97.7)      | 108/114 (94.7)         | 0.301     |
| ICA, (%)              | 204/290 (70.3)           | 124/176 (70.5)      | 80/114 (70.2)          | 0.959     |
| MCA, (%)              | 58/290 (20.0)            | 38/176 (21.6)       | 20/114 (17.5)          | 0.400     |
| ACA, (%)              | 24/290 (8.3)             | 18/176 (10.2)       | 6/114 (5.3)            | 0.134     |
| Acom, (%)             | 32/290 (11.0)            | 18/176 (10.2)       | 14/114 (12.3)          | 0.586     |
| Posterior circulation, (%) | 24/290 (8.3)    | 10/176 (5.7)        | 14/114 (12.3)          | 0.076     |
| PCA, (%)              | 4/290 (1.4)              | 2/176 (1.1)         | 2/114 (1.8)            | 0.940     |
| PICA, (%)             | 2/290 (0.7)              | 2/176 (1.1)         | 0/114 (0)              | 0.678     |
| VA, (%)               | 14/290 (4.8)             | 5/176 (2.8)         | 9/114 (7.9)            | 0.093     |
| BA, (%)               | 4/290 (1.4)              | 1/176 (0.6)         | 3/114 (2.6)            | 0.339     |

Elderly patients: age $\geq$ 70 years old; SAH subarachnoid haemorrhage; ICH intracerebral haemorrhage; CAS cerebrovascular atherosclerotic stenosis; FTP foetal-type posterior cerebral artery; SD standard deviation; ICA internal carotid artery; MCA middle cerebral artery; ACA anterior cerebral artery; Acom anterior communicating artery; PCA posterior cerebral artery; PICA posterior inferior cerebellar artery; VA vertebral artery; BA basilar artery
an endovascular approach; the remaining 109 patients (37.6%) received conservative treatment. Pulmonary infection \( (n = 138, 47.6\%) \), hydrocephalus \( (n = 72, 24.8\%) \), and thrombosis \( (n = 35, 12.1\%) \) were common complications during hospitalization. Notably, some commonly seen complications in other age groups, such as seizures (1.7%) and vasospasm (2.8%), were not obvious in our series.

For the comparison between the two groups, the ruptured group had significantly higher percentages of patients who underwent craniotomy surgery \( (P < 0.001) \), while the unruptured group had a significantly higher ratio of cases undergoing the endovascular approach \( (P < 0.001) \). Regarding complications, the ruptured group had a significantly higher percentage of patients with thrombosis than the unruptured group \( (P = 0.031) \).

**Clinical outcomes**

Patient clinical outcomes are shown in Table 6. The mean length of hospital stay was 9.5 ± 10.7 days, ranging from 1 to 74 days. By the time of discharge, favourable clinical outcomes (GCS score ≥ 13) were seen in 199 (68.6%) patients. A total of 35 patients (12.1%) died during their stay in the hospital. By the end of the 1-year follow-up, 158 patients (54.5%) demonstrated favourable outcomes (mRS score 0–2), while 33 additional patients had died, resulting in a total mortality rate of 23.4% \( (n = 68) \). The remaining 64 (22.1%) patients demonstrated unfavourable outcomes (mRS score 3–5).

The clinical outcomes showed significant differences between the ruptured and unruptured groups. Patients in the ruptured group had a significantly longer length of hospital stay than those in the unruptured group \( (P = 0.037) \). Additionally, the ruptured group had significantly better clinical outcomes at different follow-up time points, presenting as significantly higher percentages of unfavourable outcomes \( (P < 0.05) \) and death \( (P < 0.05) \) and significantly lower rates of patients who had favourable outcomes \( (P < 0.001) \).

**Discussion**

IAs in the elderly population are not very well documented due to a lack of large-sample studies. In addition, existing studies have mainly focused on patients with unruptured aneurysms [6–9]. Subsequently, the overall characteristics of IAs in elderly patients remain unclear. In the present study, several clinical characteristics of elderly IAs were reported. The findings of our study will be helpful for obtaining a better understanding of IAs in elderly age subgroups and will be beneficial for medical professionals in making the best clinical decision.

**Female predominance**

A significant female predominance (75.9%) was noted in our cohort. This result is consistent with some previous studies [8, 10, 11], but ours reported an even higher sex imbalance. Interestingly, this female predominance was not significant according to data from our younger aneurysm patient cohort, and paediatric IA patients even showed a reverse sex predominance [12]. One explanation for this discrepancy is differences in the quantity of oestrogen receptors after menopause, which might result in deterioration of vascular biology and reduced fibrillar collagen in the cerebral arteries [13–15]. This difference is also reported to be responsible for the greater propensity for aneurysm rupture [16]. Admittedly, the generally

| Table 6 | Clinical outcomes of elderly patients with intracranial aneurysms |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| **Characteristics** | **All combined (n = 290)** | **Ruptured (n = 176)** | **Unruptured (n = 114)** | **P value** |
| Mean length of stay (SD)/day | | | | |
| Discharge | 9.5 ± 10.7 | 10.5 ± 11.1 | 7.8 ± 9.9 | 0.037 |
| Favourable, (%) | 199/290 (68.6) | 97/176 (55.1) | 102/114 (89.5) | <0.001 |
| Unfavourable, (%) | 56/290 (19.3) | 49/176 (27.8) | 7/114 (6.1) | <0.001 |
| Death, (%) | 35/290 (12.1) | 30/176 (17.0) | 5/114 (4.4) | 0.002 |
| 3 month | | | | |
| Favourable, (%) | 187/290 (64.5) | 87/176 (49.4) | 100/114 (87.7) | <0.001 |
| Unfavourable, (%) | 61/290 (21.0) | 53/176 (30.1) | 8/114 (7.0) | <0.001 |
| Death, (%) | 42/290 (14.5) | 36/176 (20.5) | 6/114 (5.3) | <0.001 |
| 6 month | | | | |
| Favourable, (%) | 164/290 (56.6) | 75/176 (42.6) | 89/114 (78.1) | <0.001 |
| Unfavourable, (%) | 71/290 (24.5) | 56/176 (31.8) | 15/114 (13.2) | <0.001 |
| Death, (%) | 55/290 (19.0) | 45/176 (25.6) | 10/114 (8.8) | <0.001 |
| 1-year follow-up | | | | |
| Favourable, (%) outcomes | 158/290 (54.5) | 72/176 (40.9) | 86/114 (75.4) | <0.001 |
| Unfavourable, (%) | 64/290 (22.1) | 46/176 (26.1) | 18/114 (15.8) | 0.038 |
| Death, (%) | 68/290 (23.4) | 58/176 (33.0) | 10/114 (8.8) | <0.001 |

Elderly patients: age ≥ 70 years old; SD standard deviation; GCS Glasgow Coma Score; Favourable outcomes: GCS score ≥ 13 at discharge and Modified Rankin Scale of 0–2 during follow-up; Unfavourable outcomes: GCS score < 13 at discharge and Modified Rankin Scale of 3–6 during follow-up
longer life span in women could also contribute to this observation [17], yet further evidence is needed to validate this assertion.

Small lesions carry a high risk of rupture
It has been widely reported that the risk of rupture increases with the size of aneurysms [18]. However, in the present study, it is worth noting that of the 176 patients with ruptured lesions, 109 (61.9%) had lesions with a largest diameter of less than 5mm, which is smaller than the commonly recognized risk factors for rupture (>7 mm) [19] and the surgical indication (>5 mm) [20] based on the study of different age groups. Several factors, particularly in this age group, may contribute to this result. 1) The percentage of patients with a previous disease history, such as hypertension and diabetes mellitus, was higher than that in previous studies on other IA age groups. These comorbidities are reported to be risk factors for the rupture of small IAs [21]. 2) In morphological analysis, we noted a higher percentage of multiple aneurysms and lesions with daughter sacs. These factors have been reported to be associated with a propensity for future rupture [22, 23]. 3) Our present data showed an elevated percentage of CAS and cerebral variants (FTP) in elderly patients, which has been suggested as a possible factor for the development and even rupture of IAs [24, 25]. Together, structural abnormalities related to congenital vascular variants and multiple lesions, vessel injury caused by comorbidities and particular morphological characteristics are very likely to contribute to the formation and rupture of IAs in later stages of life, even for small lesions. Based on our results, more attention should be given to elderly patients with multiple unruptured aneurysms, with daughter sacs and with CAS and FTP, even for lesions measuring less than 0.5 cm. Typical radiological images of ruptured small lesions with diffuse SAH in our elderly patient cohort are shown in Fig. 1.

Slow development of initial symptoms caused a prolonged admission delay
In our study, we noted that the initial clinical symptoms for elderly patients with IAs were generally milder. Even for patients with ruptured IAs, headache and unconsciousness were more likely to present with slow development. Typical thunderclap headache symptoms observed in other age groups were relatively uncommon [26]; consequently, much of the delay in admission might have resulted from ignorance of early mild symptoms and overlooking the importance of early treatment. A previous study clearly specified that the delay in admission for elderly patients contributed to a poorer prognosis among the patients [27]. We suggest that medical professionals pay more attention to neurological symptoms such as moderate or even mild headache in this cohort. Possible aneurysm rupture should be considered, and a radiological exam should be arranged in a timely manner.

Complications during hospitalization
Decreased electrophysiological activity of neurons causes a low incidence of seizures, blood vasospasm and meningeal irritation
Previous studies based on different age groups noted that seizure onset was commonly associated with rupture of an IA, mainly due to the stimulation of bleeding to the cerebral cortex as well as vasospasm and subsequent ischaemic events [28, 29]. According to our previous study, the incidence of seizures was much higher in paediatric patients who carry an IA located distal to the circle of Willis [30]. For our elderly IA patient cohort, however, the incidence of seizures upon admission was obviously lower than that in previous studies on IAs in different age groups. In addition, we noticed a high modified Fisher grade in our ruptured cases due to widespread, thick haemorrhage in the expanded subarachnoid space due to obvious brain atrophy; in contrast, however, the occurrence of vasospasm was low. Some studies have suggested that arterial ageing significantly influences normal vasoconstriction, and the cellular response towards haemodynamic changes in aged arteries is reduced [31]. Additionally, it is presumed that regional metabolic dysfunction leads to the disrupted release of excitotoxic neurotransmitters [32]. This evidence indicates that ageing and brain atrophy significantly affect the normal regulation of the electrophysiological activity of neurons and arterial component cells, which was also supported by the low incidence of meningeal irritation revealed in our study.

High risk of pulmonary infection and thrombosis contribute to poor prognosis
Regardless of the presence of seizures and meningeal irritations, we noticed that the risk of pulmonary infection and thrombosis in our elderly IA patients was greater than that in previous IA studies on different age groups [33, 34]. For elderly patients, slow recovery after treatment, a prolonged length of stay in the hospital and excess time spent lying in bed may contribute to this result. It is worth noting that a certain number of deaths were caused by these complications rather than rupture of the aneurysm itself. Therefore, medical professionals should pay more attention to taking measures to prevent or address these complications during hospitalization.

Limitations
To the best of our knowledge, the present study is a relatively large single-centre retrospective study that
has validated the clinical characteristics and surgical outcomes of elderly patients with IAs to date. The limitations of this study include its nonrandomized, retrospective, observational and single-centre design, as the database only had hospitalized patients, and those with small unruptured lesions or who did not wish to have surgical treatment were not included in the analysis, which may have led to case selection bias. In addition, the characteristics included for analysis were limited due to the scope of the database, and some important factors, such as fluctuation of blood pressure, treatment of hypertension (i.e., type of medication, duration) and correlation with occurrence of rupture, were not included in the present study. Therefore, additional well-designed studies are required to obtain more robust evidence and verify the results presented in this study. In addition, although we performed bivariate analysis to explore differences in the clinical characteristics between the ruptured and unruptured groups, further multiple logistic regression analysis should be

Fig. 1 Small ruptured aneurysms with a high modified Fisher grade due to widespread, thick haemorrhage in the expanded subarachnoid space caused by obvious brain atrophy
Conducted to reveal independent factors related to the rupture of IAs in this age group.

Conclusions
Several findings regarding IAs in elderly patients were reported in this study, including an obvious female predominance; relatively mild initial symptoms causing prolonged admission delay; a low incidence of meningeval irritation, seizures and vasospasm due to decreased electrophysiological activity of neurons; increased percentages of CAS, multiple aneurysms, and aneurysms with daughter sacs causing a high risk of rupture even for small lesions; a high risk of complications during hospitalization; and relatively poor clinical outcomes.

Abbreviations
IA: Intracranial aneurysm; SAH: Subarachnoid hemorrhage; BA: Basilar artery; GCS: Glasgow Coma Scale; CTA: Computed tomography angiography; SD: Standard deviation; DSA: Digital subtraction angiography; CAS: Cerebrovascular atherosclerotic stenosis; PICA: Posterior cerebral artery; FTP: Fetal-type posterior cerebral artery; mRS: Modified Rankin scale; CI: Confidence interval; CT: Computed tomography; ICH: Intracerebral hemorrhage; ICA: Internal carotid artery; MCA: Middle cerebral artery; ACA: Anterior cerebral artery; Acom: Anterior communicating artery; VA: Vertebral artery; PICA: Posterior inferior cerebellar artery.

Acknowledgements
Not applicable.

Authors’ contributions
RC and DW analyzed and interpreted the patient data and were major contributors in writing and revising the manuscript. The author(s) read and approved the final manuscript.

Availability of data and materials
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations
Ethics approval and consent to participate
The study was approved by the institutional review board (IRB) of West China Hospital (No.2017217). A written informed consent was obtained from all individual participants included in the study.

Consent for publication
Not Applicable.

Competing interests
The authors declare that they have no competing interests.

References
1. Thompson BG, Brown RD Jr, Amin-Hanjani S, et al. Guidelines for the Management of Patients with Unruptured Intracranial Aneurysms: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2015;46(8):2368–400.
2. Ikawa F, Morita A, Nakayama T, et al. A registra-based SAH study in Japan: high incidence rate and recent decline trend based on lifestyle. J Neurosurg. 2020;134(3):983–91.
3. Huang H, O’Neill AH, Chandra RV, Lai LT. Asymptomatic intracranial aneurysms in the elderly: long-term clinical and radiologic follow-up of 193 consecutive patients. World Neurosurg. 2020;133:e660–8.
4. Brinjikji W, Rabinstein AA, Lanzino G, Kallmes DF, Cloft HJ. Effect of age on outcomes of treatment of unruptured cerebral aneurysms: a study of the National Inpatient Sample 2001-2008. Stroke. 2011;42(5):1320–4.
5. Investigators UJ, Morita A, Kinro T, et al. The natural course of unruptured cerebral aneurysms in a Japanese cohort. N Engl J Med. 2012;366(26):2474–82.
6. Lee SH, Lee SU, Kwon OK, et al. Clinical outcomes of clipping and coil in elderly patients with Unruptured cerebral aneurysms: a National Cohort Study in Korea. J Korean Med Sci. 2021;36(26):e178.
7. Bekelis K, Gottlieb DJ, Su Y, et al. Comparison of clipping and coiling in elderly patients with unruptured cerebral aneurysms. J Neurosurg. 2017;126(3):811–8.
8. Kubo Y, Koji T, Kashimura H, Otawara Y, Ogawa A, Ogasaawa K. Female sex as a risk factor for the growth of asymptomatic unruptured cerebral saccular aneurysms in elderly patients. J Neurosurg. 2014;121(3):599–604.
9. Cheikh A, Kasinathan S, Yasuhito Y, Kawase T, Kato Y. Surgical Management of Unruptured Cerebral Aneurysms in the elderly: an institution experience. Asian J Neurosurg. 2019;14(3):730–6.
10. Yang H, Jiang H, Ni W, et al. Treatment strategy for Unruptured intracranial aneurysm in elderly patients: coiling, clipping, or conservative? Cell Transplant. 2019;28(6):767–74.
11. Hishikawa T, Date I, Tokunaga K, et al. Risk of rupture of unruptured cerebral aneurysms in elderly patients. Neurology. 2015;85(21):1879–85.
12. Chen R, Zhang S, You C, Guo R, Ma L. Pediatric intracranial aneurysms: changes from previous studies. Childs Nerv Syst. 2018.
13. Mata KM, Li W, Reslan OM, Siddiqui WT, Siddiqui LA, Khalil RA. Adaptive increases in expression and vasodilator activity of estrogen receptor subtypes in a blood vessel-specific pattern during pregnancy. Am J Physiol Heart Circ Physiol. 2015;309(10):H1679–96.
14. Arnal JF, Laurell H, Fontaine C, et al. Estrogen receptor actions on vascular biology and inflammation: implications in vascular pathophysiology. Climacteric. 2009;12(Suppl 1):12–7.
15. Ouchi Y, Akishita M, Urao T. The role of estrogen in the regulation of blood vessel function. Nihon Rinsho. 2005;63(Suppl):S575–84.
16. Maekawa H, Tada Y, Yagi K, et al. Bazedoxifene, a selective estrogen receptor modulator, reduces cerebral aneurysm rupture in Ovariectomized rats. J Neuroinflammation. 2017;14(1):197.
17. Zrubka Z, Kincses T, Calusci L, Kovacs L, Pentek M. Subjective expectations concerning life expectancy and age-related health burden. J Psychiatr Res. 2021;162(3):911–23.
18. Rinkel GJ, Dijbuti M, Algra A, van Gijn J. Prevalence and risk of rupture of intracranial aneurysms: a systematic review. Stroke. 1998;29(1):251–6.
19. Koja M, Lehto H, Juvela S. Lifelong rupture risk of intracranial aneurysms depends on risk factors: a prospective Finnish cohort study. Stroke. 2014;45(7):1958–63.
20. Komotar RJ, Mocco J, Solomon RA. Guidelines for the surgical treatment of Unruptured intracranial aneurysms: the first annual J. Lawrence pool memorial research symposium—controversies in the management of cerebral aneurysms. Neurosurgery. 2008;62(1):183–93 discussion 193–184.
21. Ikawa F, Morita A, Tomiyan S, et al. Rupture risk of small unruptured cerebral aneurysms. J Neurosurg. 2019;1-10.
22. Jung KH. New pathophysiological considerations on cerebral aneurysms. Neurointervention. 2018;13(2):73–83.
23. Bor AS, Tiel Groenestege AT, terBrugge KG, et al. Clinical, radiological, and flow-related risk factors for growth of untreated, unruptured intracranial aneurysms. Stroke. 2015;46(1):42–8.
24. Feng X, Qi P, Wang L, et al. Relationship between cerebrovascular atherosclerotic stenosis and rupture risk of unruptured intracranial aneurysm: a single-center retrospective study. Clin Neurol Neurosurg. 2019;186:105543.
25. He Z, Wan Y. Is fetal-type posterior cerebral artery a risk factor for intracranial aneurysm as analyzed by multislice CT angiography? Exp Ther Med. 2018;15(4):838–46.
26. Schwiedt TJ. Thunderclap headaches: a focus on etiology and diagnostic evaluation. Headache. 2013;53(3):563–9.
27. Goertz L, Pflaeging M, Hamsch C, et al. Delayed hospital admission of patients with aneurysmal subarachnoid hemorrhage: clinical presentation, treatment strategies, and outcome. J Neurosurg. 2020;134(3):1182–9.
28. Choi KS, Chun HJ, Yi HJ, Ko Y, Kim YS, Kim JM. Seizures and epilepsy following aneurysmal subarachnoid hemorrhage: incidence and risk factors. J Korean Neurosurg Soc. 2009;46(2):93–8.
29. Lin CL, Dumont AS, Lieu AS, et al. Characterization of perioperative seizures and epilepsy following aneurysmal subarachnoid hemorrhage. J Neurosurg. 2003;99(6):978–85.
30. Chen R, Zhang S, Guo R, Ma L, You C. Pediatric intracranial distal arterial aneurysms: report of 35 cases. Acta Neurochir. 2018.
31. De Silva TM, Modrick ML, Dabertrand F, Faraci FM. Changes in cerebral arteries and parenchymal arterioles with aging: role of rho kinase 2 and impact of genetic background. Hypertension. 2018;71(5):921–7.
32. Davalos A, Shuaib A, Wahlgren NG. Neurotransmitters and pathophysiology of stroke: evidence for the release of glutamate and other transmitters/mediators in animals and humans. J Stroke Cerebrovasc Dis. 2000;9(6 Pt 2):2–8.
33. Geraldini F, De Cassai A, Correale C, et al. Predictors of deep-vein thrombosis in subarachnoid hemorrhage: a retrospective analysis. Acta Neurochir. 2020;162(9):2295–301.
34. Cavallo C, Safavi-Abbasi S, Kalani MYS, et al. Pulmonary complications after spontaneous aneurysmal subarachnoid hemorrhage: experience from Barrow neurological institute. World Neurosurg. 2018;119:e366–73.

Publisher’s Note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.