Childhood haemorrhagic stroke – A 7 years single center experience

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Abstract

Background: In recent years, there has been increasing research interest in improving diagnostic and management protocols in childhood arterial ischaemic stroke (AIS). However, childhood stroke comprises, in approximately equal parts, both arterial ischaemic and haemorrhagic stroke (HS).

Objective: The aim of this study was to focus on the aetiology, clinical presentation, treatment and short-term outcome of children with spontaneous intracranial bleeding in a university hospital and elucidate differences to childhood arterial ischaemic stroke.

Design: We performed a retrospective analysis of electronic medical records of children (28 days – 18 years) diagnosed with haemorrhagic stroke between 2010 and 2016.

Results: We included 25 children (male n=11) with a median age of 8y1m. The most common clinical presentations were vomiting (48%), headache (40%) and altered level of consciousness (32%). In more than half of the patients haemorrhagic stroke was caused by vascular malformations. Other risk factors were brain tumour, coagulopathy and miscellaneous severe underlying diseases. Aetiology remained unclear in one child. Therapy was neurosurgical in most children (68%). Two patients died, five patients needed further (rehabilitation) treatment and 18 children could be discharged home.

Conclusions: Haemorrhagic stroke differs from AIS in aetiology (vascular malformations as number 1 risk factor), number of risk factors (“mono-risk” disease), clinical presentation (vomiting, headache and altered level of consciousness) and (emergency) therapy.

1. Introduction

Childhood stroke occurs with an incidence of 1,3-8/100 000 children/year, approximately as often as brain tumours, and is among the top ten of mortality in children [1-4]. Primary strokes may be either arterial ischaemic strokes (AIS) that are caused by a blocked arterial supply or venous (= cerebral venous sinus thrombosis) or haemorrhagic, when they are defined as nontraumatic spontaneous intracranial bleeding. There has been increasing research activity in the field of childhood arterial ischaemic stroke in recent years: Understanding the underlying pathological mechanisms (e.g. focal cerebral arteriopathy), acute treatment options (e.g. iv thrombolysis and mechanical thrombectomy) and reducing the time gap between clinical onset and diagnosis by using appropriate imaging modalities are examples of ongoing research [5-11]. There has been less research interest in and awareness of haemorrhagic stroke (HS), although it accounts for almost half of all childhood strokes.

Therefore, we aimed to describe a paediatric cohort with HS with respect to clinical presentation, aetiology and short-term outcome and point out the differences to childhood AIS. These data could support paediatricians in their clinical assessment and diagnostic investigation of children presenting with possible HS.

2. Method

We performed a retrospective review of electronic medical records of children with haemorrhagic stroke in a tertiary university children`s hospital in Munich, Germany between January 2010 - December 2016. This university children`s hospital provides, together with another university hospital and two other children`s hospitals, acute medical advice for 231.510 children from 0-18 years (overall population of Munich in 2017: 1.456.039)[12] .

Inclusion criteria was the diagnosis of an haemorrhagic stroke defined as spontaneous, non-traumatic intracranial bleeding (intraparenchymal, ventricular or subarachnoid) in children and adolescents (≥28 days of life ≤18 years). As it remains unclear whether children who bleed in a brain tumour are subsumed under the term `haemorrhagic stroke`, we decided only to include those patients with a brain tumour, who bled into brain parenchyma, ventricles or subarachnoid space. Exclusion criteria were perinatal / neonatal haemorrhage (<28 days of life), primary subdural or epidural bleeding, traumatic intracranial bleeding, haemorrhagic transformation of AIS or secondary HS due to a venous infarction.

Patients eligible for inclusion were identified via ICD-10 codes (international classification of diseases) given by the treating paediatrician. In order to ensure as complete data collection as possible, we used a broad spectrum of ICD-10 codes (see table 1).

In total, 290 children were identified applying the abovementioned ICD-10 criteria and their electronic medical records were further reviewed to see whether they met the inclusion - exclusion criteria as shown in figure 1.

Collected information included demographic data, clinical presentation, aetiology, imaging modality, acute treatment and short-term outcome with respect to discharge from hospital.

Ethical approval

The study was approved by the ethics committee and data protection commissioner of the Medical Faculty of the Ludwig-Maximilians-University Munich, Nr 756-16 (06-12-16).

3. Results

Patients

The study group consisted of 25 patients. Sex ratio was m:f = 0.8:1. Median age at the time of bleeding was 8 years 1month (range 1 month – 16years 9months) .

Clinical presentation

The presenting features of the children are presented in tables 2 and 3.

Most children presented with vomiting (48%), headache (40%) and altered level of consciousness (32%), followed by seizures (28%) brainstem symptoms (16%), and acute hemiparesis (12%). One child, receiving extracorporeal mebrane oxygenation on the intensive care unit, showed signs of increased intracranial pressure. All three infants (i.e. <1year of age) with intracranial bleeding presented with vomiting. One infant also had seizures.

Three children suffered a “silent” stroke, meaning that the haemorrhage was an incidental finding in otherwise indicated neuroimaging. The reasons for neuroimaging were: (i) routine MRI screening in a child with known multiple cavernoma; (ii) MRI in a child with a suspected transient ischemic attack (clinical symptoms were not explained by the diagnosed cavernoma: localisation of the cavernoma: right frontal lobe; clinical symptoms: speech disturbance and right-sided hemiparesis) and (iii) a CT scan in a child to exclude cerebral oedema after cardiopulmonary resuscitation.

Aetiology

Vascular malformations were the most common underlying aetiology (n=13), but did not occur as haemorrhage in children in the first year of life. We identified six children with

haemorrhage due to arteriovenous malformations (AVM) and seven children with cavernoma, but no patient with bleeding from an intracranial aneurysm. Further characteristics of these patients with vascular malformation are provided in table 3.

Patients with a brain tumour who bled into parenchyma or ventricles represent the second largest group (n=5). Brain tumours were histopathologically classified as glioblastoma, medulloblastoma, diffuse astrocytoma (WHO II), primitive neuroectodermal tumour (PNET) and atypical teratoid/rhabdoid tumour (ATRT).

There were two patients with an underlying coagulopathy: one had idiopathic thrombocytopenic purpura (thrombocyte count 3000/ microliter) and the other had Vitamin K deficiency due to infantile obstructive cholangiopathy. Both patients with coagulopathy were younger than one year.

Other aetiologies included severe acute infection (Herpes simplex virus (HSV) encephalitis) (n=1), chronic lung disease of unknown aetiology with multiorgan failure in the course of a septic shock (n=1) and haemato-oncological diseases (n=2; hepatocellular carcinoma with brain metastases (n=1), acute myeloid leukaemia, pulmonary invasive aspergillosis with need of cardiopulmonary resuscitation (n=1)). In one child, the underlying aetiology for the spontaneous ventricular bleeding remained unclear besides extensive diagnostic work-up including computed tomography (CT), CT angiography, magnetic resonace angiography (MRA), conventional cerebral angiography (CCA) and laboratory testing for coagulation problems and vasculitis.

With respect to number of risk factors, 20 patients had a single risk factor, four patients had a severe underlying disease with in the clinical course presumed multiple risk factors and one child had no known risk factor.

Treatment

Treatment decision was based on the initial presentation of the child and underlying aetiology. 17 patients underwent neurosurgical intervention, one patient endovascular (embolization of an AVM) and seven children conservative treatment.

Short-term Outcome

In our cohort two patients died. Both patients suffered from a severe underlying disease (severe chronic lung disease and infantile obstructive cholangiopathy) with intracranial bleeding as a result of an extremely complicated course of the primary disease. Most patients could be discharged home (n=18). Five children had further in-hospital treatments or were transferred to a rehabilitation clinic.

Neurological examination at time of discharge was normal in 13 children. Neurological deficits in the other patients were hemiparesis ± facial palsy (n=8), ataxia (n=1), speech disturbance (n=1) and impaired short-term memory (n=1). One patient had multiple severe neurological sequelae after a brainstem bleeding. One small infant showed no neurological deficit but had a poor prognosis due to a malignant brain tumour (ATRT) without response to chemotherapy. Our collected data do not provide any information about long-term outcome.

Discussion

Treatment of children with HS is not only the preserve of neurosurgeons and interventional neuroradiologists and should involve paediatricians/paediatric neurologists. It is the paediatrician`s responsibility as the first medical contact to know when to think about HS and what to do as the first diagnostic steps in uncovering the most likely underlying aetiologies.

In our cohort, most children presented with vomiting, headache and an altered level of consciousness. This result is similar to literature where headache is one of the main presenting symptom[13-23]. Headache is very frequently reported in children and adolescents in the general population and is one of the most common reasons for patients seeking medical advice from paediatricians. Sometimes it may be difficult to decide which child with headache needs more extensive investigations because they may have intracranial bleeding. However, as de Ribaupierre et al suggest, every headache that is described as “worst” and/or has a “sudden” onset as well as every headache with concomitant focal neurological deficits should immediately lead to further investigations [17].

In younger children in whom headache may/could not be the leading (communicated) symptom, the clinical presentation of haemorrhagic stroke may be more nonspecific. Presenting symptoms may include vomiting, irritability, reduced level of consciousness, or seizures. It also follows, that well-known stroke recognition tools like the FAST test, featuring the leading symptoms in arterial ischaemic stroke (facial palsy, hemiparesis and speech disturbance) are not applicable in children with haemorrhagic stroke. To increase awareness for both ischaemic and haemorrhagic stroke, the paediatric stroke working group of the Ludwig-Maximilians-University Munich developed a pocket card for paediatricians called

MERCS (= Munich early recognition of childhood stroke) highlighting leading symptoms for both stroke types on the front and back of the pocket card (see supplemental material).

In our cohort, a cranial CT as first imaging was performed in 64% of the children. CT remains the imaging modality of first choice in a clinically unstable child with a reduced level of consciousness. CT is widely available, quick and usually does not require anaesthesia in children. It provides information about the type of bleeding (intraparenchymal vs. ventricular vs. subarachnoid), the size of the haemorrhage and the presence of mass effect and sometimes reveals information about aetiology – all of which is essential information to guide emergency neurosurgical treatment. Outside the emergency situation, an MRI /MRA is required as part of the diagnostic work-up to identify or exclude a vascular malformation as an underlying cause and – with respect to the known insufficient diagnostic yield of non-invasive imaging - in some cases (especially in patients with unknown aetiology) a CCA is necessary [24]. But even a complete imaging and laboratory diagnostic work-up may fail to detect the underlying reason for the bleeding (in our cohort n=1). One reason could be, that the the vascular anomaly is obliterated by the haemorrhage and therefore “invisible” in post-hoc investigations. Diagnostic work-up may also remain incomplete in patients with an unfavourable outcome.

With respect to the underlying risk factors, our study concurs with the published literature: vascular diseases are the predominant risk factor for intracranial bleeding in a paediatric population (52% in our study vs 7-91% in the literature) [13-23]. In our cohort, vascular malformation were more common in elder children and were not present in the first year of life. This is a relevant information with reference to the required diagnostic work up, as

investigation for coagulation problems, infections and malignancies (including brain tumours) then becomes more important in the younger age group. Aneurysms, as one of the three main vascular risk factors besides AVM and cavernoma, were not described in our cohort, probably due to the small cohort size. In some children, intracranial haemorrhage is described as the initial symptom of a brain tumour[18, 20]. The definition of haemorrhagic stroke related to a brain tumour is imprecise and not consistent. We decided to exclude patients who bled into a brain tumour (n=8) and only described patients with a brain tumour who bled into brain parenchyma, the ventricles or subarachnoid space (n=5). One study with high percentages of children with brain tumour as an underlying aetiology included patients who had bled into a brain tumour [18]. One other study did not provide clear information about whether haemorrhage was into or outside the brain tumour, merely describing it as being associated with a brain tumour [20]. Nevertheless, the proportion of children with a brain tumour as underlying disease (20%) in our cohort remains high compared to other studies (up to 15%). As our university hospital is a center for paediatric oncology and haematology, this may be the reason for the higher percentages compared to non-specialized clinics. With respect to the number of risk factors, HS differs from AIS: Whilest childhood AIS is known to be a "multiple risk" disease and requires extensive diagnostic work up, most children with HS show a single risk factor with exception of children with severe underlying multiorgan diseases and complex medical treatment prior the intracranial haemorrhage. Apart from that, the percentage of so called cryptogenic strokes seems lower in HS compared to AIS.

An intracranial haemorrhage is a possible but rare complication in patients with HSV encephalitis [25]. Hypotheses regarding the underlying pathological mechanisms include the

presence of a small vessel vasculitis, raised intracranial pressure or damaged brain tissue being more vulnerable to bleeding or a combination of both altered parenchyma and vessels. It is important to know, that the bleeding may occur even in children treated with antiviral agents and the treating paediatrician should have a bleeding complication in mind in every child who has a secondary neurological deterioration. The patient in our cohort with HSV was a 15 year old boy with an acute clinical deterioration (worst headache, nausea and sinus bradycardia) and large haemorrhage in the temporal lobe on day four after admission and the initiation of antiviral treatment with acyclovir.

This was not an epidemiological study and therefore the patient population studied will have been subject to selection biases that influenced their referral to our hospital. For example, Ludwig-Maximilians-University Hospital is a paediatric onclogy centre and the relatively high incidence of malignancy as an underlying cause of HS probably reflects this selction bias. Other selection biases, like the referral of adolescents to adult stroke units or neurosurgery departments, may be applied, but, we believe that the range of underlying aetiologies that we found reflect the true range even if the proportions may have been different in a population based study.

Our retrospective study only provides data about short-term outcome. At the end of the acute care phase, 52% of the patients had no neurological deficit and 72% could be discharged home. But without using a standardized neurological examination instrument like the Pediatric Stroke Outcome Measure (PSOM) and because of missing short- or long-term follow up data, these results are barely comparable to other studies. Beslow et al found an abnormal neurological examinationa after a median follow up time of 3.5 months in 71% of children after HS [15]. Recently, a rehabilitation center-based retrospective study focused on long-term outcome of 128 children after childhood AIS and HS. In general, they saw better

motor neurological, functional and academic outcomes after HS (versus AIS) at time of discharge from the rehabilitation department [26]. However, at time of discharge (median time since stroke of 4.7 months), 59.3% of children with HS showed some degree of motor deficit and 35.8% required special education after a median follow-up period of 43 months. Further prospective studies or registry based data are required to capture all children with childhood HS in order to describe accurately long-term outcomes and how these outcomes are influenced by underlying aetiology, acute treatment and rehabilitative treatment after discharge from the acute hospital setting.

Conclusion

HS constitutes approximately 40% of all childhood strokes and may lead to significant morbidity and mortality. The knowledge of the leading clinical symptoms, aetiology and necessary diagnostic steps are essential for quick emergency management and professional discussion in a multidisciplinary neurovascular team. As HS differs from AIS in most aspects, the training of caregivers should not be limited to the recognition of AIS and the development of paediatric stroke recognition tools should include basic information about the clinical presentation of children with intracranial bleeding. With respect to further research, a first important step for comparable cohort studies should be a clear definition of HS in the paediatric population and a consistent use of ICD-10 coding.

What is already known on this topic

- Haemorrhagic stroke in children is rare, but constitutes approximately 40% of all childhood strokes and may lead to significant morbidity and mortality
- Vascular malformations are the most common underlying risk factors for spontaneous intracranial bleeding in childhood
- (Interventional) neuroradiology and neurosurgery play a crucial role in the neurovascular treatment team

What this study adds

- Symptoms vary by age (headache in children and seizures and non-specific syptoms in infants)
- Compared to arterial ischaemic stroke, haemorrhagic stroke is a mono-risk disease
- Paediatricians are the first medical contact person for children with headache, vomiting and altered level of conciousness – the most presenting symptoms of haemorrhagic stroke
- The development of pediatric stroke recognition tools should provide both information about ischaemic and haemorrhagic stroke
- Intracranial bleeding due to a vascular malformation is rare in infants and young children and requires thorough diagnostic work up with a paediatric expertise

Contributors

LG and FJO conceptualized and designed the study and FJO supervised all aspects of the work. LG, KB and FJO contributed to analysis and interpretation of the data. RW performed statistical analysis. LG drafted the initial manuscript. FH, AP, FD, MB and SB contributed to

interpretation of the data and reviewed and revised the manuscript. All authors approved the final manuscript as submitted.

Conflicts of interest

None

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References

1. Fullerton HJ, Wu YW, Zhao S and Johnston SC. Risk of stroke in children: ethnic and gender disparities. *Neurology*. 2003; 61: 189-94.
2. Giroud M, Lemesle M, Gouyon JB, Nivelon JL, Milan C and Dumas R. Cerebrovascular disease in children under 16 years of age in the city of Dijon, France: a study of incidence and clinical features from 1985 to 1993. *J Clin Epidemiol.* 1995; 48: 1343-8.
3. Lynch JK, Hirtz DG, DeVeber G and Nelson KB. Report of the National Institute of Neurological Disorders and Stroke workshop on perinatal and childhood stroke. *Pediatrics*. 2002; 109: 116-23.
4. Mallick AA, Ganesan V, Kirkham FJ, et al. Childhood arterial ischaemic stroke incidence, presenting features, and risk factors: a prospective population-based study. *Lancet Neurol*. 2014; 13: 35-43.
5. Fullerton HJ, Hills NK, Elkind MS, et al. Infection, vaccination, and childhood arterial ischemic stroke: Results of the VIPS study. *Neurology*. 2015; 85: 1459-66.
6. Gerstl L, Olivieri M, Heinen F, et al. Successful mechanical thrombectomy in a three-year-old boy with cardioembolic occlusion of both the basilar artery and the left middle cerebral artery. *Eur J Paediatr Neurol.* 2016; 20: 962-5
7. Kirton A and deVeber G. Paediatric stroke: pressing issues and promising directions. *Lancet Neurol*. 2015; 14: 92-102.
8. Mallick AA, Ganesan V, Kirkham FJ, et al. Diagnostic delays in paediatric stroke. *J Neurol Neurosurg Psychiatry*. 2015; 86: 917-21.
9. Rafay MF, Pontigon AM, Chiang J, et al. Delay to diagnosis in acute pediatric arterial ischemic stroke. *Stroke*. 2009; 40: 58-64.
10. Rivkin MJ, deVeber G, Ichord RN, et al. Thrombolysis in pediatric stroke study. *Stroke*. 2015; 46: 880-5.
11. Steinlin M and Mackay MT. Emergency management of ischemic stroke in children. *Curr Treat Options Neurol*. 2015; 17: 349.

12. Bayerisches Landesamt für Statistik. *https://wwwstatistikbayernde/statistik/gebiet_bevoelkerung/bevoelkerungsstand/indexhtml - link_2*. 2017.
13. Abbas Q, Merchant QU, Nasir B, Haque AU, Salam B and Javed G. Spectrum of Intracerebral Hemorrhage in Children: A Report from PICU of a Resource Limited Country. *Crit Care Res Pract*. 2016; 2016: 9124245.
14. Aydinli N, Tatli B, Caliskan M, et al. Stroke in childhood: experience in Istanbul, Turkey. *J Trop Pediatr*. 2006; 52: 158-62.
15. Beslow LA, Licht DJ, Smith SE, et al. Predictors of outcome in childhood intracerebral hemorrhage: a prospective consecutive cohort study. *Stroke*. 2010; 41: 313-8.
16. Blom I, De Schryver EL, Kappelle LJ, Rinkel GJ, Jennekens-Schinkel A and Peters AC. Prognosis of haemorrhagic stroke in childhood: a long-term follow-up study. *Dev Med Child Neurol*. 2003; 45: 233-9.
17. de Ribaupierre S, Rilliet B, Cotting J and Regli L. A 10-year experience in paediatric spontaneous cerebral hemorrhage: which children with headache need more than a clinical examination? *Swiss Med Wkly*. 2008; 138: 59-69.
18. Lanthier S, Carmant L, David M, Larbrisseau A and de Veber G. Stroke in children: the coexistence of multiple risk factors predicts poor outcome. *Neurology*. 2000; 54: 371-8.
19. Laugesaar R, Kolk A, Uustalu U, et al. Epidemiology of childhood stroke in Estonia. *Pediatr Neurol*. 2010; 42: 93-100.
20. Lo WD, Lee J, Rusin J, Perkins E and Roach ES. Intracranial hemorrhage in children: an evolving spectrum. *Arch Neurol*. 2008; 65: 1629-33.
21. Meyer-Heim AD and Boltshauser E. Spontaneous intracranial haemorrhage in children: aetiology, presentation and outcome. *Brain Dev*. 2003; 25: 416-21.
22. Paonessa A, Limbucci N, Tozzi E, Splendiani A and Gallucci M. Radiological strategy in acute stroke in children. *Eur J Radiol*. 2010; 74: 77-85.
23. Yock-Corrales A, Varela-Bulgarelli F, Barboza C, Gutierrez-Mata A, Mackay MT and Babl F. Presentation of Acute Childhood Stroke in a Tertiary Pediatric Emergency Department. *Pediatr Emerg Care*. 2016.
24. Liu AC, Segaren N, Cox TS, et al. Is there a role for magnetic resonance imaging in the evaluation of non-traumatic intraparenchymal haemorrhage in children? *Pediatr Radiol*. 2006; 36: 940-6.
25. ElShimy G, Mariyam Joy C, Berlin F and Lashin W. Intracranial Hemorrhage Complicating Herpes Simplex Encephalitis on Antiviral Therapy: A Case Report and Review of the Literature. *Case Rep Infect Dis*. 2017; 2017: 6038146.
26. Yvon E, Lamotte D, Tiberghien A, et al. Long-term motor, functional, and academic outcome following childhood ischemic and hemorrhagic stroke: A large rehabilitation center-based retrospective study. *Dev Neurorehabil*. 2018; 21: 83-90.

Tables:

ICD-10 code	
I61.X (I61.0-I61.9)	intracerebral haemorrhage
I60.X (I60.0-I60.9)	subarachnoid haemorrhage
I62.9	Intracranial haemorrhage (non-traumatic), unspecified
I64	stroke, not specified as haemorrhage or infarction
I67.5	Moyamoya disease
I67.1	cerebral aneurysm, non-ruptured
Q28.2	Arteriovenous malformation of cerebral vessels
Q28.3	Other malformations of cerebral vessels
D18.0	haemangioma, intracranial
D43.X + R58	Neoplasm of uncertain or unknown behaviour of brain and central nervous system + haemorrhage, not elsewhere classified
C71.X + R58	malignant neoplasm of brain + haemorrhage, not elsewhere classified

Table 1: ICD-10 codes for case identification

Table 2: Basic demographic and clinical data (n=25)	
Variable	**n (%)**
Sex (male)	11 (44%)
Age (years)	
<1	3 (12%)
1-5	4 (16%)
6-18	18 (72%)
First event	23 (92%)
Symptoms	
vomiting	12 (48%)
headache	10 (40%)
altered level of conciousness	8 (32%)
brainstem symptoms	4 (16%)
seizure	7 (28%)
hemiparesis	3 (12%)
ataxia	2 (8%)
facial palsy	1 (4%)
other cranial nerve symptoms	2 (8%)
speech deficit	1 (4%)
irritability	1 (4%)
fever	1 (4%)
signs of increased intracranial pressure	1 (4%)
no symptoms, incidental finding	3 (12%)
First Imaging	
cCT/cCTA	16 (64)
cMRI/cMRA	7 (28%)
Cranial ultrasound	1 (4%)
Unknown	1 (4%)
Aetiology	
AVM	6 (24%)
cavernoma	7 (28%)
brain tumor	5 (20%)
others (coagulopathy, infection, medication, malignancy)	7 (28%)
Treatment	
conservative	7 (28%)
enodovascular	1 (4%)
neurosurgery	17 (68%)
Discharge	
further rehabilitation/treatment in a hospital	5 (20%)
home	18 (72%)
died	2 (8%)

Table 2: basic demographic and clinical data in n=25 children with HS

Table 3: Vascular diseases (n=13)		
	AVM (n=6)	**Cavernoma (n=7)**
	n (%)	**n (%)**
Sex (male)	1 (17%)	4 (57%)
Age (years)		
< 1	-	-
1-5	2 (33%)	1 (14%)
6-18	4 (67%)	6 (86%)
Symptoms		
headache	3 (50%)	2 (29%)
seizure	2 (40%)	4 (57%)
altered level of conciousness	3 (50%)	-
hemiparesis	2 (40%)	-
facial palsy	1 (17%)	-
other cranial nerve symptoms	2 (40%)	-
brainstem symptoms	1 (17%)	-
vomiting	4 (67%)	-
speech deficit	1 (17%)	-
no symptoms, incidental finding	-	2 (29%)
First imaging		
cCT/cCTA	4 (67%)	1 (14%)
cMRI/cMRA	2 (33%)	5 (71%)
unknown	-	1 (14%)
Conventional angiography	4 (67%)	1 (14%)
Emergency Treatment		
neurosurgery/craniectomy	2 (33%)	-
Treatment		
Neurosurgery	5 (83%)	5 (71%)
endovascular	1 (17%)	-
conservative	-	2 (29%)
Familiar	-	2 (29%)
Discharge		
further rehabilitation/treatment in a hospital	1 (17%)	-
discharged home	5 (83%)	7 (100%)

Table 3: Characteristics of n=13 children with vascular diseases