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Vein of Galen malformation

Endovascular management of 43 cases

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Abstract. Since 1984, 43 patients with true vein of Galen aneurysmal malformations have been referred to us and managed according to our patient selection, technique, and follow-up guidelines. Thirty-four were embolized transarterially with bucrilate (isobutyl cyanoacrylate) or enbucrilate (N-butyl cyanoacrylate) embolization. No cutdown or hypotension during or after the embolization was used and no balloon catheter was employed. Forty-seven percent of the children had a completely occluded lesion which was confirmed when the child was at least 6 months of age at the follow-up angiographic examination; 52.9% were found to be completely normal or only to have mild cardiac failure that could be treated medically or moderate macrocephaly without neurological symptoms or mental retardation. In the embolized group 5.8% died as a result of the wrong treatment (1 case) or poor timing of embolization 3 days after ventricular shunting (1 case). The overall mortality (embolized and non-embolized groups) in the neonatal children was 27.7% with a total of 18.6% for all ages. Complete morphological exclusion of the arteriovenous malformation was accomplished in 41.9%; 74.4% of all children referred are now clinically normal or present moderate mental retardation which is diminishing. There was 3% neurological morbidity in the embolized group (only following the venous approach) in 78 sessions and more than 100 arteries embolized. These results compare favorably with surgical or other techniques of arterial embolization (balloon or particles), as well as transvenous (transtorcular or transfemoral) embolization, where the morbidity and mortality are significantly higher and the late clinical evaluation is seldom satisfactory. We believe that presently there is no indication for surgery as a primary form of treatment if a properly trained interventional neuroradiological team is available.

Key words: Vein of Galen malformation – Cardiac failure – Embolization – Neonates

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Since the review of Johnston et al. [13] in 1987, several papers have appeared in the literature dealing mainly with advances in both the neuroradiological diagnosis [9, 32] and endovascular approach [3, 5, 11, 21, 23, 36, 37] to vein of Galen arteriovenous malformations (VGAM). Some surgical data have also been published [19, 20, 25, 30].

Most recent series are either very small (less than ten cases) or include different entities. Most extend over 10 years and do not take into consideration the significant technical advances and better comprehension of the disease that have been achieved over the past few years [6, 10, 27, 31, 35]. No reference to patient selection [15, 17] is given and most of the angioarchitecture [14, 28, 29] is not analyzed in the detail necessary for interpretation and documentation of the results. Vein of Galen aneurysmal dilatations (VGAD) and VGAM [15] are still not differentiated [13, 21] and often treated similarly, sometimes by the transvenous route [22] or with balloons [21].

The purpose of the present paper is to contribute, with our personal series of 43 consecutive children, to a better understanding of the treatment strategy in VGAM. All cases were managed between September 1984 and September 1990. Anatomical description [16, 18], angioarchitectural classification [14], indications and contraindications have already been published [12, 17, 33]. We shall therefore concentrate on the clinical and morphological results obtained with our therapeutic strategy.

Materials and methods

During the past 6 years, 70 patients with the diagnosis of VGAM were referred to one of us (P.L.); among them, only 49 cases (70%) corresponded to the strict definition of VGAM. The remaining 21 consisted of different entities (VGAD with dural or cerebral arteriovenous shunts, venous ectasia, etc.) and will not be considered here. All 49 VGAM cases were children less than 16 years. Five patients were excluded as they were managed differently than recommended at the consultation; and a 6th patient was excluded because we were unable to obtain any data from clinical and morphological follow-up, although the immediate result following arterial embolization, in 1984, was considered excellent.

The remaining 43 children analyzed in the present paper represent all the cases that we have managed in accordance with our guidelines for patient selection, treatment, and follow-up. All cases were followed, both clinically and neuroradiologically (magnetic resonance imaging and angiography), at a frequency dependent on the urgent or emergent need for further active management.

The specific points of our treatment of VGAM can be summarized as follows:

1. The transarterial approach is used a priori in all cases with IBCA or NBCA embolization (isobutyl or *N*-butylcyanoacrylate; Braun, Melsungen, FRG).

2. We use the transarterial Seldinger technique following femoral artery puncture and the 4 French introducer sheath is used, without cutdown.

3. No balloon catheter system is employed.

4. No staged embolization is performed if the occlusion can be achieved in one session.

5. Neonates and infants are kept for 24 h under general anesthesia in the pediatric intensive care unit when complete occlusion has been achieved.

6. The venous approach is only considered when the arterial one has failed.

Our patient selection can be summarized as follows:

1. Aggressive medical management of neonatal systemic manifestations is undertaken to gain as much time as possible [12, 33] (Table 6).

2. An active search is undertaken to identify therapeutic contraindications prior to any treatment [15].

3. Strict periodic neuropsychological testing is performed (Denver and Brunet Lezine).

4. No angiographic exploration is performed unless a therapeutic attempt is contemplated during the same session (to avoid unnecessary femoral punctures) or to confirm the complete exclusion of the lesion 6 months after the last embolization.

The technique, embolic agent used and patient selection have remained the same since the beginning of the series.

Since most patients are referred to us from outside France, initial management is often recommended by telephone, particularly in neonates. If necessary, the child is transferred to the interventional neuroradiology section within the pediatric department of our hospital. In rare cases the child is medically managed in the referring hospital and embolized by one of us (P.L.) in the local institution, if logistically acceptable [34].

Results

We have presented the results in two different ways to outline differences between groups as regards age and type of lesion:

1. Clinical results of overall management in neonates, separate from infants and children since the challenge is significantly different in these groups (Table 1), and

2. Results depending on the anatomical type of lesion, to enhance the prognostic consequences and the predictive value of the anatomical diagnosis on the final outcome (Tables 2, 3).

A comparison between the patients with and without ventricular shunting has also been undertaken with the same material, but will not be presented here [38].

No mortality was directly related to the actual embolization procedure; however, two deaths occurred in the embolized group: one due to multiorgan failure and one to poor timing of the embolization in the immediate postoperative period (3 days) after ventricular shunting associated with bilateral subdural hematomas [15].

No femoral or neurological morbidity was noted with transarterial embolization (74 procedures with more than 100 arteries embolized). Three distal IBCA emboli were noted, none of them symptomatic. Two of these patients are clinically normal after 1 and 4 years of follow-up respectively. The remaining one was secondarily embolized by the venous approach and presented with a postoperative intracranial hemorrhage (ICH) (Fig. 1). The VGAM in all three patients are completely obliterated.

In four patients the transvenous approach (three transtorcular and one transfemoral) was used following

Table 1. Vein of Galen arteriovenous malformations (VGAM) – patients' management and results (Bicêtre 1984–1990)

		No. (100%)	Complete exclusion	Clinically normal	Mild symptoms, neurologically normal[a]	Moderate mental retardation (<20%) or mild neurological symptoms	Significant mental retardation (>20%) or neurological deficit	Death
Embolized patients	Neonatal group	13	4 (30.8%)	3 (23.1%)	1 (7.7%)	6 (46.1%)	2 (15.4%)	1 (7.7%)
	Infants and children	21	12 (57.1%)	8 (38%)	6 (28.6%)	5 (23.8%)	1 (4.8%)	1 (4.8%)
Total		34	16 (47%)	11 (32.35%)	7 (20.6%)	11 (32.35%)	3 (8.8%)	2 (5.8%)
Non-embolized patients	Neonatal group	5	0	1 (20%)	0	0	0	4 (80%)
	Infants and children	4	2	1 (25%)	0	1 (25%)	0	2 (59%)
Total		9	2 (22.2%)	2 (22.22%)	0	1 (11.1%)	0	6 (66.7%)

[a] Moderate cardiac overload stable, or residual macrocephaly (<2 SD)

failure to catheterize safely the arterial feeders to the lesion. One postembolization ICH occurred with incomplete resolution of focal neurological deficit (Fig. 1). One case of early postembolization hydrocephalus occurred in a neonate following 75% occlusion of the malformation with coils in 1986; the child is presently significantly mentally retarded (30% in relation to chronological age). The condition of one child is presently stable; the patient is still under treatment and has mild mental retardation. The remaining child is normal (Fig. 2).

Secondary spontaneous distal (dural sinus) occlusion occurred in four cases following partial or complete transarterial exclusion of the VGAM. One of them presented progressive hydrocephalic manifestations, 3 months after embolization, that resolved after ventricular shunting. The child's VGAM is completely occluded and he is clinically normal [15]. The remaining three cases are asymptomatic after follow-up periods of between 6 months and 2 years.

Table 2. VGAM – choroidal type

27 Patients
20 embolized cases (60 sessions)
No spontaneous closure
7 complete vascular exclusions in 1–6 sessions
4 more are expected to be obtained
6 deaths – all in non-embolized group
15 patients (55%) presented some mental retardation
All neonates that survived (one incidental discovery) presented some mental retardation

Table 3. VGAM – mural type

16 Patients
14 embolized cases (18 sessions)
2 spontaneous closure
9 complete vascular exclusions (1 death) (7 times in 1 session – 2 times in 2 sessions)
2 deaths (1 multiorgan failure, 1 complete exclusion with improper therapeutic timing and management)
The remaining 4 patients are expected to be completely excluded from the circulation by further embolization
7 patients (38.8%) presented some mental retardation

Discussion

Endovascular management is currently the best method of treating VGAM, particularly when compared to other treatment modalities [13, 20, 25].

The transvenous approach, although technically simple, appears unsatisfactory with regard to outcome. Recent series have shown a very high morbidity (30–66%) [4, 5, 11, 23]. Although the number of cases reported in the literature is small, morbidity and mortality are 10–20 times higher than those reported for arterial embolization with NBCA. This corresponds to our experience; in the four cases in which the venous approach had to be employed, significant neurological morbidity occurred (1:4), with only a few normal children on follow-up (Fig. 2).

The transarterial approach, in the present series, shows a high rate of complete obliteration (47%), as well as low morbidity and mortality. Wisoff and Berenstein [37], during the same period (1984–1990), noted similar operative results but a different late clinical outcome: 40% death or disabled patients, as opposed to 14.7% in our series (or 23.2% if non-embolized patients are included). The difference may be related to lack of patient selection in the Berenstein series (Table 4) and to slight differences in NBCA deposition. In our neonatal group we have been able to delay the first embolization procedure for a few weeks in about 30% of cases (Table 5). Management of neonates remains a difficult challenge (Fig. 2) that cannot be simply reduced to a technical one [7, 8, 12, 24]. Different embolic agents have been used by authors, resulting in 57% morbidity with particle embolization [5], as well as disappointing clinical results with either particles or balloons [21].

Our experience appears to indicate that the results and morbidity associated with the transarterial approach and

Table 4. VGAM[a] – natural history and treatment results from Johnston (modified)

Age	Untreated				Treated			
	No.	Dead	Alive impaired	Alive normal	No.	Dead	Alive impaired	Alive normal
Neonate	12	12 (100%)	–	–	58	52 (89.6%)	1	3 (+2)[b] (5%)
Infant	11	8 (72.7%)	1	2	58	22 (37.9%)	17	18 (+1)[b] (31.5%)
1–5 years	7	4 (57.1%)	1	1 (+1)	27	9 (33.3%)	6	10 (+2)[b] (40%)
6–20 years	8	2	–	4 (+2)	10	2	2	3 (+3)[b]
>20 years	8	5	–	– (+3)	13	2	8	2 (+1)[b]

[a] Includes vein of Galen dilatation
[b] (+*n*), Children lost to follow-up

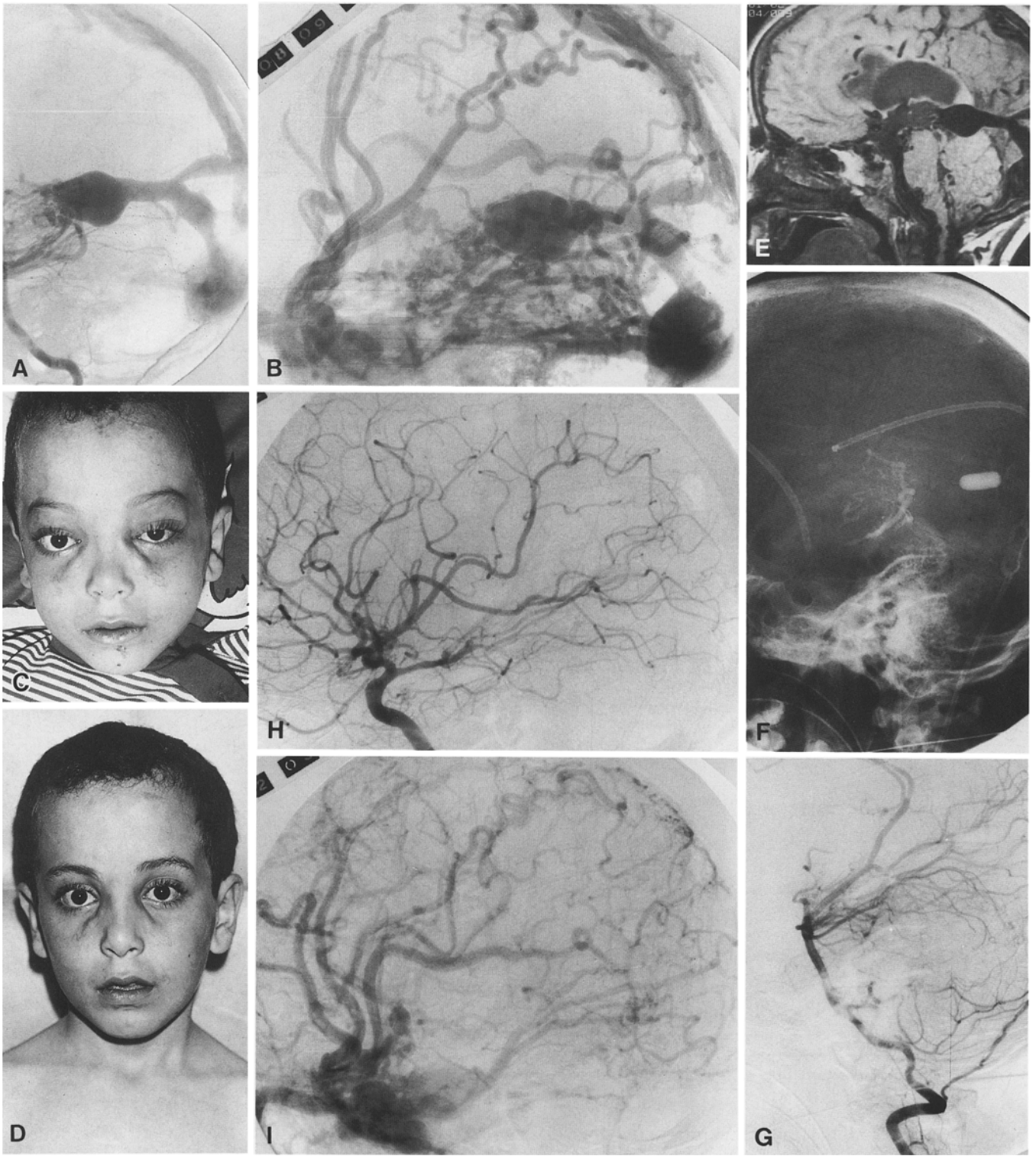

Fig. 1 A–I. Vertebral angiogram in early (**A**) and late (**B**) phase in a case of choroidal type of vein of Galen arteriovenous malformations (VGAM); distal spontaneous sinus occlusion re-routes both the arteriovenous malformation and normal brain venous flow to the facial veins (**C**). Following three arterial embolization sessions, flow reduction is obtained with concomitant improvement of the facial vein circulation (**D**). In view of the remaining retrograde cerebral flow, the venous approach is carried out by transtorcular balloon occlusion of the proximal falcine sinus (**E**) and (**F**). The patient was operated on in the prone position. The procedure was uneventful and lasted 2 h; however, 8 h later, he developed an intracerebral hematoma. He remains hemiparetic 3 months later. Vertebral (**G**) and carotid (**H**, **I**) angiographic control 1 month after embolization shows complete occlusion of the lesion and the residual drainage of the normal cerebral venous system into the facial veins

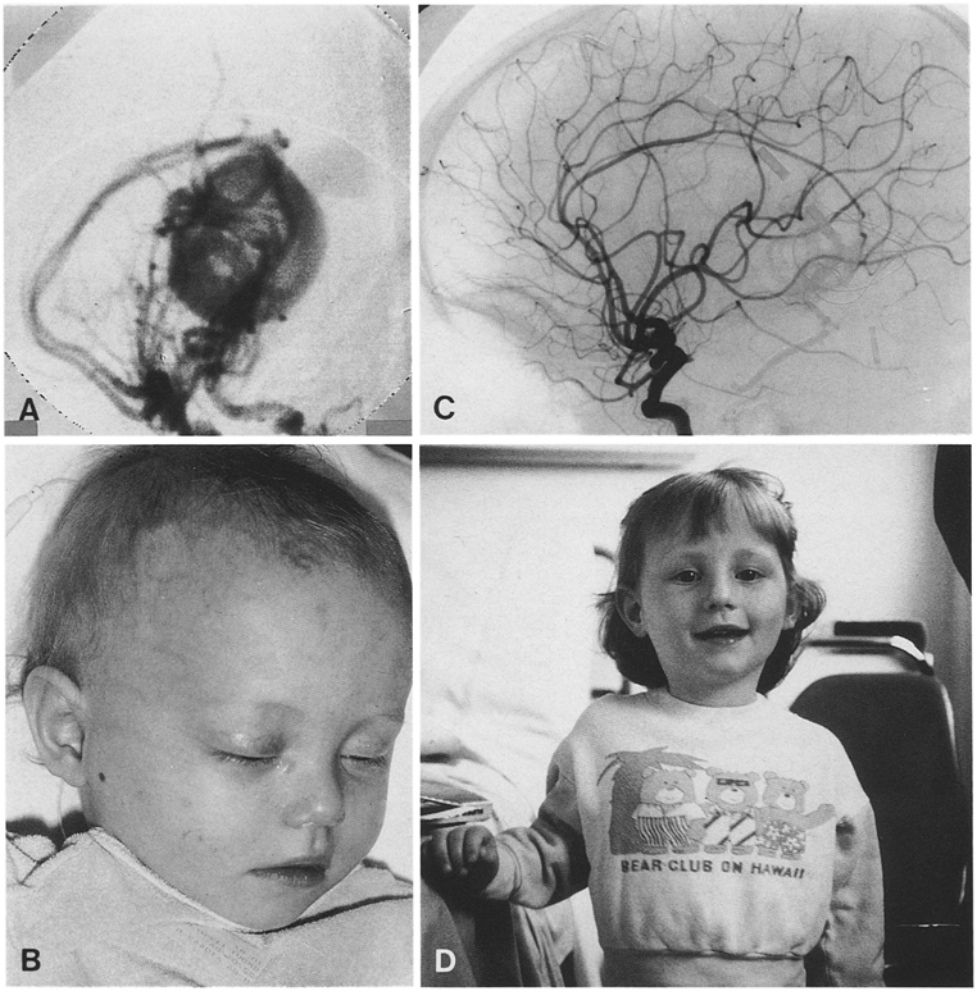

Fig. 2. **A** Typical choroidal form of VGAM in a neonate with severe cardiac failure. **B** Following two arterial embolizations in the neonatal period age, the cardiac failure was controlled; then she progressively developed macrocephaly in infancy. **C** After two arterial, one venous (femoral) and one additional arterial approach, complete occlusion was obtained. **D** Five years later, she is clinically normal, free of any systemic or neurological symptoms; her head circumference is normal

Table 5. NYU series (courtesy of A. Berenstein): VGAM – pediatric population <16 years (1984–1990).VGC, Choroidal type of VGAM; VGM, mural type of VGAM

	No.	Permanent anatomic exclusion	Clinically normally	Resid. manif. but neurologically normal	Neurologically impaired[a]	Disabled	Dead
Embolized patients (VGC and VGM)	20 (100%)	4 (20%)	5 (25%)	2 (10%)	5 (25%)	2 (10%)	6 (30%)
Non-embolized patients	5	3	2	–	1	–	2
Total patients treated and non-treated	25 (100%)	7 (28%)	7 (28%)	2 (8%)	6 (24%)	2 (8%)	8 (32%)

[a] Includes mild mental retardation

bucrilate embolization are significantly better than for any other treatment strategy for patients with VGAM.

Short-term results include correction of mental retardation, macrocephaly, magnetic resonance imaging (MRI) evaluation of brain maturation and shrinkage of the pouch ([16], Fig. 3). These represent additional features to be credited to the transarterial route, and have so far not been reported in approaches used by other authors.

The high frequency of mental retardation was remarkable in our series (51%, but 62% in children that survived the neonatal period). Many infants, although referred to us as neurologically normal, already had mild or severe retardation when included in our series. This symptom is rarely documented in the literature (10%) [3, 13, 26]. Following embolization of 80% of the lesion, correction of mild retardation (<20% in relation to chronological age) is rapidly obtained. Mental retardation is different from developmental delay, since thriving is usually preserved in infants. This retardation is probably related to cerebral fluid disorders and will be discussed with the cerebrospinal fluid manifestations [38].

So far we have not felt the need in the postembolization follow-up for the use of Doppler ultrasonography [9,

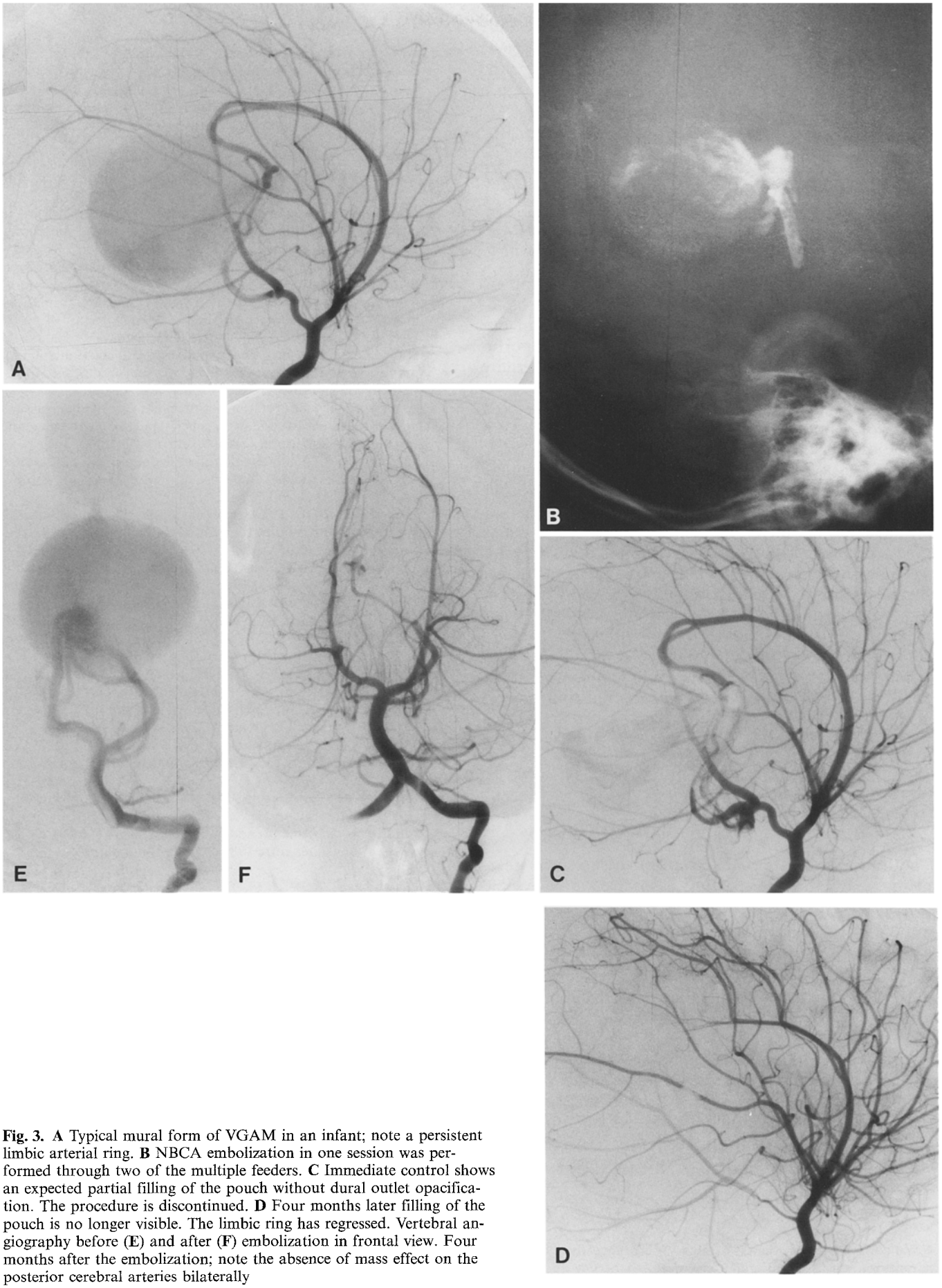

Fig. 3. **A** Typical mural form of VGAM in an infant; note a persistent limbic arterial ring. **B** NBCA embolization in one session was performed through two of the multiple feeders. **C** Immediate control shows an expected partial filling of the pouch without dural outlet opacification. The procedure is discontinued. **D** Four months later filling of the pouch is no longer visible. The limbic ring has regressed. Vertebral angiography before (**E**) and after (**F**) embolization in frontal view. Four months after the embolization; note the absence of mass effect on the posterior cerebral arteries bilaterally

Table 6. VGAM – age at referral and treatment (1984–1990)

Age at referral	Age at embolization
18 Neonates (<1 month)	5 Neonates
14 Infants (<1 year)	16 Infants[a]
11 Children (<16 years)	13 Children
43 Patients	34 Patients

[a] Five neonates were embolized in infancy

32]. Most of the indications for further treatment are related to clinical findings and MRI provides a satisfactory morphological control for decision making, in our opinion. Obviously, ultrasonography offers an opportunity to collect additional information that may require further anatomical and clinical correlation.

Late postembolization venous occlusions have been observed in four cases with no consequences. Similar observations have been made without any form of treatment ([2, 14], Fig. 1). It seems that pre-existing stenotic jugular bulbs remain patent due to the VGAM flow. The normal cerebral veins are already draining into the cavernous sinus and then into the ophthalmic vein or pterygoid venous plexuses. Thus, following reduction or exclusion of the VGAM shunt, the stenotic segment further thromboses without clinical consequence and does not require additional medical or urgent endo-vascular treatment. Conversely, incompletely embolized lesions, with venous drainage re-routed into the cerebral venous circulation, would in most instances require an immediate attempt to further reduce or exclude the remaining shunt (Fig. 1); in that situation the predictable cerebral risk is higher than for dural AV shunts with cortical venous drainage.

Conclusion

The transarterial approach as a primary form of treatment in our series of 43 patients has proved to be feasible in 95% of cases. Our present overall strategy for embolized and non-embolized patients gives satisfactory clinical results with 46.5% normal or almost normal patients 41.8% of them with an already excluded lesion. Half of the remaining patients are expected either to be completely normal or at least to have completely occluded VGAM.

Using other embolic materials and the venous approach, a significantly higher morbidity results. Since arterial embolization corrects the hydrodynamic disorders, we believe that there is presently no indication for surgery as a primary form of treatment if a properly trained interventional neuroradiological team is available.

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