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CASE REPORT

Frosted branch angiitis: a review

S Walker[1], A Iguchi[2] and NP Jones[1]

Abstract

The purpose of this study is to present the first report of a case of primary frosted branch angiitis from the UK and to review the characteristics of this rare disease. Primary frosted branch angiitis causes characteristic florid translucent retinal perivascular sheathing of both arterioles and venules in association with variable uveitis, retinal oedema and visual loss, normally with good recovery. A total of 57 cases have been reported in the world literature. Atypical, typically focal frosted branch angiitis may also occur secondary to other causes of intraocular inflammation, especially cytomegalovirus retinitis. Primary frosted branch angiitis has a characteristic presentation but a variable course, typically affecting children or young adults. The disease is likely to represent a common immune pathway in response to multiple infective agents. The optimal treatment is unclear.
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Keywords: frosted branch angiitis; retinal vasculitis; uveitis; systemic steroid

Case report

A 7-year-old boy presented after 3 days of blurred vision and floaters in both eyes, preceded by an upper respiratory tract infection treated with oral amoxycillin. There was no other significant medical history. On examination, visual acuities were right 6/60 and left 2/36. There was a mild right relative afferent pupillary defect. There was bilateral diffuse conjunctival injection with mild nongranulomatous anterior uveitis and vitritis. Intraocular pressures were right 12 mmHg and left 8 mmHg. Fundal examination (Figure 1) revealed symmetrical widespread retinal vasculitis, including prominent sheathing affecting both venules and arterioles. There was bilateral moderate papillitis, severe macular oedema, and scattered small retinal haemorrhages. The patient had submandibular lymphadenopathy, but no other abnormality on general examination.

Full blood count and differential cell count were normal, as were erythrocyte sedimentation rate, C-reactive protein, chest X-ray, angiotensin-converting enzyme, immunoglobulins, and liver function tests. There was no evidence of cellular or humoral immune deficiency. An auto-antibody screen was negative, as was the Paul Bunnell test. Serology for cytomegalovirus (CMV), herpes simplex, varicella zoster and Epstein–Barr viruses, rubella, and syphilis were negative. Fluorescein angiography (FA, Figure 2) revealed normal blood flow, but late diffuse staining and leakage of the affected vessels and the optic discs. A diagnosis of primary frosted branch angiitis (FBA) was made. Within 24 h visual acuity had deteriorated to counting fingers at 1 m in both eyes. He was treated with oral prednisolone 1 mg/kg/day, with topical steroid and mydriatic. Response was rapid, with resolution of all clinical signs and improvement in visual acuity to 6/9 in both eyes, by day 15. Systemic steroid treatment was tapered and discontinued after 4 weeks.

After 4 weeks, uveitis with mild macular oedema recurred, without angiitis. Topical steroid was recommenced. An MRI scan of orbits and brain was normal, as was electrophysiology. The Goldmann fields showed generalised constriction. The disease course fluctuated in the early stages, accompanied by an inferior punctate retinitis, but subsided 14 months later, leaving fine retinal pigment epithelial changes at both maculae, but visual acuity of better than 6/6 in both eyes.

Discussion and review of the literature

In 1976 Ito[1] reported a 6-year-old Japanese boy with panuveitis and widespread retinal vasculitis. The florid translucent perivascular exudate inspired the descriptive term 'frosted branch angiitis'. The phenomenon is rare and probably a new entity, only 57 cases[2–46] having

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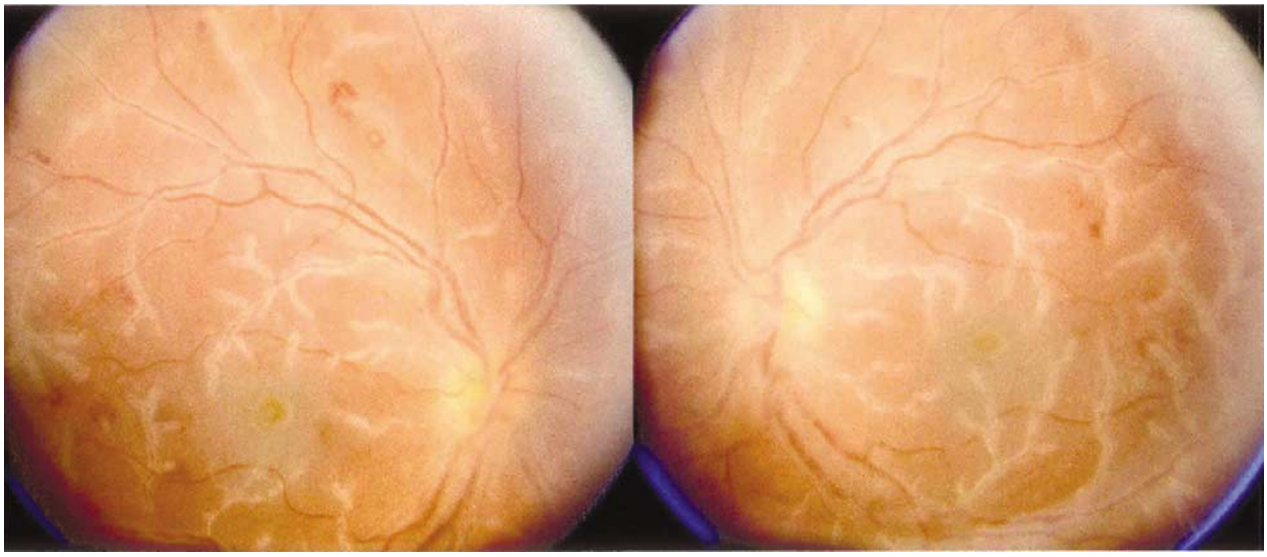

Figure 1 Bilateral primary idiopathic frosted branch angiitis in a 7-year-old boy. There is extensive, translucent perivascular 'exudate', disc and mascular oedema, and scattered intraretinal haemorrhages.

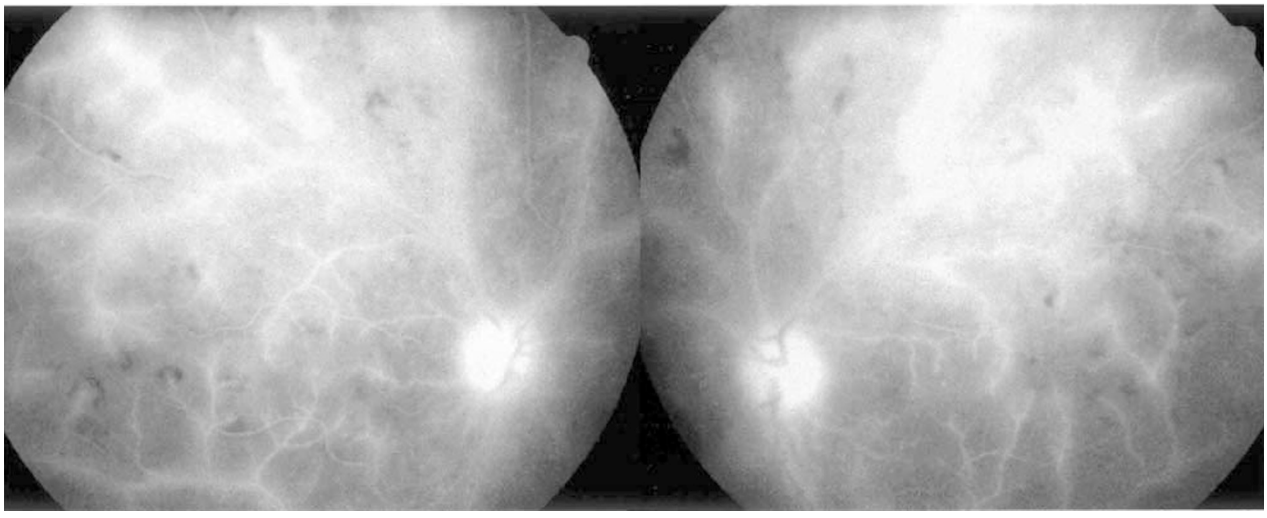

Figure 2 Fluorescein angiography is frosted branch angiitis showing extensive vascular leakage and retinal oedema. The same patient as in Figure 1.

been recorded in the world literature (Table 1). The great majority (75%) of patients are Japanese; indeed, it was not until 1988[13] that any patient outside Japan was reported. A total of 9 cases have subsequently been reported from the USA[13,25,27,32,44] and single cases from Turkey,[37] Korea,[40] India,[41] and Spain.[46] Our patient is the first to be described in the UK.

FBA predominantly affects the young and fit. There appears to be a bimodal age distribution (Figure 3), with one peak in childhood and a second in the third decade. The youngest patient reported to date was 2 years[16] and the oldest 42 years.[28] There has been a preponderance of females (61%) to males (39%).

The most common presenting symptoms of FBA are subacute visual loss, floaters or photopsiae. The visual acuity may be substantially reduced (Table 1), even to perception of light.[4,17] Most patients (75%) have bilateral disease.

Typical FBA has a striking fundal appearance (Figures 1 and 2); the retinal vasculitis is widespread and bilateral with a 'frosted' quality to the perivascular exudate. Mild- to moderate iritis with vitritis is common, as is retinal oedema. Intraretinal haemorrhages and punctate hard exudates are only occasionally seen. Papillitis, if present, is usually mild; marked disc oedema has been noted only by us. FA is near normal in the early phase, but there is

Table 1 Reported cases of primary idiopathic frosted branch angiitis 1976–2003

Case no.	*Year, author(s)*	*Age*	*Sex*	*Country of origin*	*Prodrome*	*Side*	*Worst VA*		*Systemic treatment*	*Final VA*		*Recovery time (months)*
							R	*L*		*R*	*L*	
1	1976, Ito *et al*[1]	6	M	Japan	(Triple vaccine)	B	HM	CF	Steroid, isoniazid	6/60	6/12	5
2	1982, Miyazaki *et al*[2]	5	F	Japan	Varicella	R	6/36	6/7.5	Steroid	6/4	6/4	<1
3	1984, Sakanishi *et al*[3]	8	M	Japan	URTI	B	6/60	6/60	Steroid	6/12	6/6	1
4	1984, Sakanishi *et al*[3]	9	F	Japan	URTI	B	6/60	6/15	Steroid	6/5	6/5	12
5	1985, Higuchi *et al*[4]	9	F	Japan	Nil	B	PL	PL	Steroid (-globulin)	6/12	6/9	12
6	1985, Yamane *et al*[5]	8	F	Japan	Varicella	B	CF	CF	Steroid	6/5	6/5	1
7	1986, Kadoya *et al*[6]	23	F	Japan	Nil	B	6/60	6/15	NSAID, aspirin, kallidinogenase	6/5	6/5	1
8	1987, Horiuchi[7]	20	F	Japan	URTI	B	6/60	3/60	Steroid, NSAID	6/6	6/7.5	<1
9	1987, Ichikawa and Majima[8]	9	F	Japan	Nil	B	NK	NK	Steroid	NK	NK	5
10	1987, Watanabe *et al*[9]	16	F	Japan	Nil	B	CF	CF	Steroid	6/9	6/9	5
11	1987, Kubota *et al*[10]	32	M	Japan	Lymphangiitis	B	6/60	CF	Steroid, NSAID	6/6	6/5	1
12	1988, Kodama *et al*[11]	8	F	Japan	Headache	B	6/5	6/9	Aciclovir	6/4	6/4	4
13	1988, Suzuki *et al*[12]	3	M	Japan	Nil	B	6/18	6/18	Steroid	6/12	6/15	8
14	1988, Kleiner *et al*[13]	25	M	USA	Scabies	B	HM	CF	Steroid	3/60	6/6	28
15	1988, Kleiner *et al*[13]	29	F	USA	Nil	B	6/6	6/12	Steroid	6/6	6/6	10
16	1988, Kleiner *et al*[13]	23	F	USA	URTI	B	CF	CF	Steroid, penicillin	6/6	6/6	4
17	1989, Asai *et al*[14]	27	M	Japan	UTI	L	NK	6/9	Steroid	NK	'Imp'	NK
18	1989, Takaseki *et al*[15]	8	F	Japan	Nil	B	6/24	6/60	Steroid	6/6	6/6	6
19	1989, Hikichi *et al*[16]	2	F	Japan	Fever, sepsis	B	NK	NK	Steroid	NK	NK	NK
20	1989, Kanai *et al*[17]	39	F	Japan	Headache, vomiting	B	HM	PL	Steroid, aciclovir	6/5	6/5	10
21	1989, Kijosawa *et al*[18]	24	F	Japan	Nil	B	6/36	6/36	Steroid, NSAID	6/4	6/4	3
22	1989, Terasaki *et al*[19]	21	F	Japan	Fever, arthralgia	B	6/60	6/36	Steroid	6/9	6/7.5	3
23	1990, Yoshida *et al*[20]	13	M	Japan	Nil	B	3/60	3/60	Aspirin, NSAID, kallidinogenase	6/4	6/5	27
24	1990, Narita and Sato[21]	38	M	Japan	Nil	R	6/60	'B1'	Aciclovir, NSAID	6/4	'B1'	2
25	1990, Isobe and Yamamoto[22]	6	F	Japan	Fever	B	6/36	6/60	NSAID	6/6	6/6	3
26	1990, Isobe and Yamamoto[22]	12	M	Japan	Headache	B	6/12	6/15	Steroid	6/6	6/6	<1
27	1990, Mizukami *et al*[23]	5	F	Japan	Fever	B	3/60	2/60	Steroid, aciclovir	6/5	6/9	6
28	1991, Emi *et al*[24]	39	M	Japan	Gout	B	3/60	3/60	Steroid, aciclovir, Urokinase, aspirin	6/5	6/5	1
29	1991, Sugin *et al*[25]	32	M	USA	Viral illness	R	CF	6/6	Steroid	6/6	6/6	2
30	1991, Sugin *et al*[25]	26	M	USA	Nil	R	CF	6/6	Steroid	6/24	6/6	1
31	1991, Mikami *et al*[26]	13	F	Japan	Nil	B	NK	NK	Aciclovir	NK	NK	NK
32	1991, Vander and Masciulli[27]	33	F	USA	Viral illness	B	3/60	3/60	Nil	6/9	6/9	1
33	1992, Ohta *et al*[28]	42	F	Japan	Nil	R	3/60	6/5	Steroid	6/5	6/5	3

Table 1 *Continued.*

Case no.	*Year, author(s)*	*Age*	*Sex*	*Country of origin*	*Prodrome*	*Side*	*Worst VA*		*Systemic treatment*	*Final VA*		*Recovery time (months)*
							R	*L*		*R*	*L*	
34	1992, Tsue and Maeda[29]	41	M	Japan	Fever	L	NK	HM	Steroid	NK	6/60	NK
35	1992, Suzuki *et al*[30]	24	F	Japan	Nil	B	2/60	6/60	Steroid	6/6	6/6	<1
36	1992, Tachinami *et al*[31]	36	M	Japan	Viral illness	B	6/9	6/12	Steroid	6/6	6/6	<1
37	1992, Hamed *et al*[32]	5	M	USA	Nil	B	CF	CF	Nil	6/9	6/9	2
38	1992, Browning[33]	28	F	USA	Nil	L	6/6	6/6	Nil	6/6	6/6	36
39	1992, Nakai and Saika[34]	3	F	Japan	Nil	B	'moderate'	Steroid	6/7.5	6/7.5	4	
40	1993, Chuman *et al*[35]	4	M	Japan	Fever	B	6/18	6/9	Steroid	6/6	6/6	<1
41	1993, Uenoyama *et al*[36]	30	F	Japan	Nil	B	3/60	6/36	Steroid	6/5	6/4	1
42	1993, Atmaca and Gunduz[37]	30	F	Turkey	Nil	B	3/60	6/12	Steroid	3/60	6/15	6
43	1994, Okinami *et al*[38]	13	M	Japan	Nil	B	6/9	6/9	Steroid	6/5	6/5	2
44	1994, Okinami *et al*[38]	14	F	Japan	Nil	L	6/6	6/5	Steroid	6/5	6/4	36
45	1994, Okinami *et al*[38]	30	M	Japan	Nil	R	6/15	6/6	Steroid	6/36	6/7.5	156
46	1994, Okinami *et al*[38]	28	M	Japan	Renal failure	L	6/7.5	2/60	Haemodialysis	6/5	3/60	2
47	1995, Yamagata *et al*[39]	8	F	Japan	Fever, rash	B	6/7.5	6/60	Steroid	6/6	6/6	2
48	1995, Lee *et al*[40]	5	F	Korea	Pneumonia	B	NK	NK	Steroid, aciclovir	3/60	3/60	NK
49	1996, Biswas *et al*[41]	8	F	India	Fever	B	CF	CF	Steroid	6/12	6/12	2
50	1996, Kawaguchi *et al*[42]	5	F	Japan	Fever	B	6/9	6/9	Steroid	6/5	6/5	2
51	1998, Masuda *et al*[43]	24	F	Japan	Nil	B	6/4	6/4	Steroid	6/4	6/4	3
52	1999, Borkowski and Jampol[44]	8	M	USA	Nil	B	6/60	6/60	Steroid	6/7.5	6/60	7
53	1999, Johkura *et al*[45]	20	F	Japan	Aseptic meningitis	L	NK	NK	Steroid	NK	NK	<1
54	2001, Huerva *et al*[46]	20	M	Spain	Nil	B	6/30	6/60	Steroid	6/7.5	6/7.5	2
55	2001, Kaburaki *et al*[47]	36	F	Japan	Nil	R	1/60	6/5	Steroid, aspirin	PL	NK	NK
56	2001, Kaburaki *et al*[47]	23	F	Japan	Nil	L	6/5	3/60	Steroid	NK	PL	NK
57	2003, Walker	7	M	UK	URTI	B	CF	CF	Steroid	6/9	6/9	<1

Side B = bilateral, L = left, R = right; URTI = upper respiratory tract infection; UTI = urinary tract infection; NSAID = nonsteroidal anti-inflammatory drug; NK = not known; 'B1' = 'blind'; 'Imp' = 'improved'.

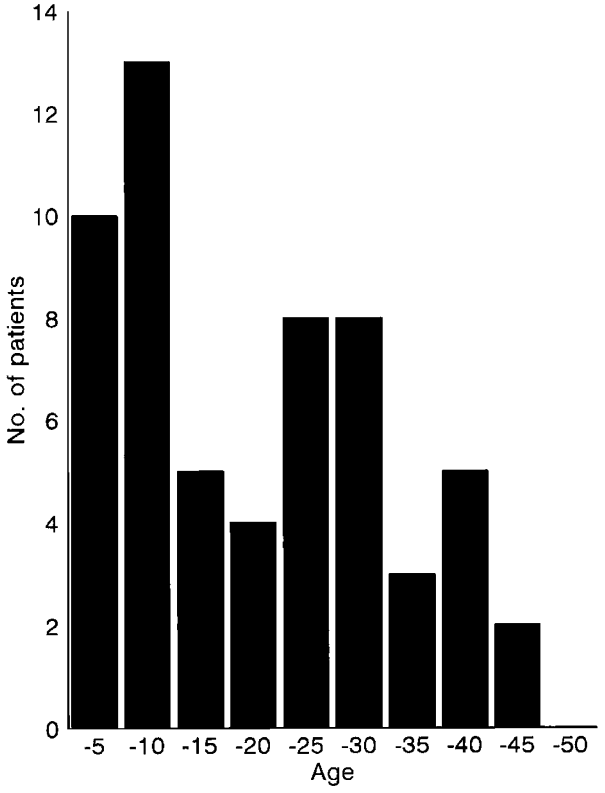

Figure 3 Age distribution at presentation (possibly bimodal) for all published cases of primary frosted branch angiitis.

late leakage from the larger affected retinal vessels. In the recovery phase, microaneurysms have been described.[3] The vasculitis is usually nonocclusive. Visual field analysis reveals constriction or relative central defects that improve after clinical resolution1.[6,7,9,10,19,20,36,37,41,43] The latter are thought to result from macular oedema.[9,37]

Electrophysiology has shown a reduction in the amplitudes of the electroretinogram (ERG), electro-oculogram (EOG), and visually evoked response (VER). This would be consistent with a widespread dysfunction of the retina, pigment epithelium, and optic nerve.[9,47] The EOG and VER may return to normal,[1,4,9] but the ERG changes have generally persisted beyond convalescence, suggesting permanent retinal damage.

Most patients with FBA have been treated with systemic steroids and have rapidly resolved with good visual recovery (Table 1). Less frequently, recovery has taken place over many months. Aciclovir has been used[11,17,21,23,24,26,40] with unknown effect. Recurrence is rare.[4,48]

The prognosis in FBA has not always been favourable. Macular scarring has severely limited vision in 3 patients.[13,25,37] Complications include retinal vein[13,49] or artery occlusion,[24] macular epiretinal membrane formation,[38] diffuse retinal fibrosis,[37] retinal tear formation,[13] vitreous haemorrhage,[50] optic disc atrophy[1] and peripheral atrophic retinal lesions.[1,9,16,30,37] The final visual acuity has been 6/60 or worse in 10% of affected eyes thus far reported (cases 1, 14, 34, 42, 46, 48, 52, 55, 56, Table 1), nine of which received systemic steroid treatment. In contrast, five patients (eight affected eyes) with severe visual loss (cases 7,23,24,32,37, Table 1) did not receive systemic steroid treatment, yet regained high acuity.

The cause of FBA is unknown. The typical onset of FBA after a multifactorial prodromal illness has led to the suggestion of a hypersensitivity reaction to various infective agents,[23] which may initiate FBA via a common pathway, possibly of immune-complex deposition.[13,38] This would support the use of systemic corticosteroids for those with substantial visual loss. However, the visual results with or without systemic steroid treatment thus far provide no clear guidance on treatment.

Other causes of retinal vasculitis should be excluded, especially viral retinitis, sarcoidosis, multiple sclerosis, toxoplasmosis, syphilis, and Behçet's disease. Infiltrative causes such as lymphoma and leukaemia may occasionally mimic FBA. However, the distinctive clinical appearance is usually pathognomonic. Tentative suggestions[31] of a link to HLA-A2 are inadequately supported. A presumed viral illness has preceded the onset in 33% of cases including our own.[2–4,5,7,13,17,19,22,23,25,27,29,31,35,39,41,42] No consistent aetiological agent has been identified, but positive serology results have been reported for: herpes simplex virus (HSV),[11,13,26,34,35,41] varicella zoster virus (VZV),[2,5,11,13,21] tuberculoprotein,[1,5,34] antistreptolysin O,[4,13,39] Epstein-Barr virus,[25] CMV,[34] Coxsackie virus A10,[31] adenovirus,[34] measles[34] and rubella.[41]

The cases listed in Table 1 and referred to above appear, with limited variation, to adhere to Ito's original description of a primary idiopathic FBA. However, other cases of FBA, often localised, have been described, which appear either to be secondary to intraocular infection by CMV,[51–58] HSV (confirmed by aqueous PCR),[59,60] herpesvirus hominis,[61] toxoplasmosis[62] or *Fusarium dimerium*,[63] or in association with systemic lupus erythematosus,[64] Crohn's disease,[65] lymphoma,[66] leukaemia[67] and Behçet's disease (our own unpublished case, Figure 4).

In summary, Frosted branch angiitis in typical form, unassociated with preexisting intraocular inflammation, is likely to be a primary immunological process in response to a number of provoking antigens. As discussed by Kleiner,[13,68] an apparently different form exists, which is secondary to concurrent inflammation, most commonly in association with CMV retinitis.[51–58] Kleiner classifies the form seen in association with intraocular lymphoma or leukaemia differently, which

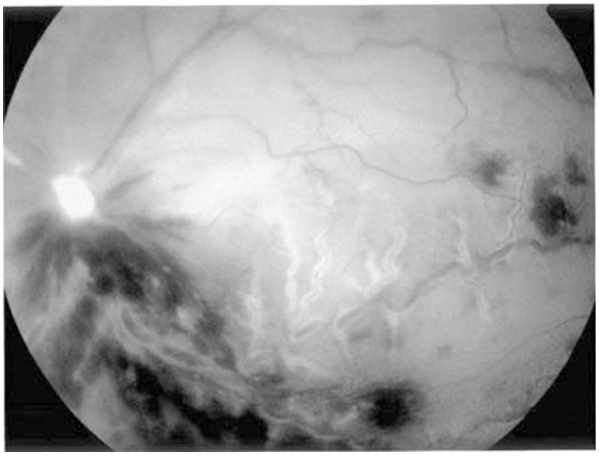

Figure 4 Occlusive hemispheric retinal vasculitis in a 22-year-old woman with Behçet's disease. In addition to widespread retinal haemorrhage, venous tortuosity, and disc oedema, there is a frosted branch appearance to much of the involved venous tree.

may mimic FBA but is probably an infiltrative process. Our review of the world literature to date suggests that this proposal is sound. However, in order to distinguish classical FBA, as originally described by Ito[1] from secondary forms and masquerades, we propose the descriptive term 'primary idiopathic frosted branch angiitis'.

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