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Solitary intraretinal macular hemorrhage

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Abstract. Thirty patients presented with a solitary intraretinal macular hemorrhage (SIMH) without clinical evidence of associated retinal or choroidal vascular disease. The hemorrhages originated from the perifoveal capillary network and often extended into the central fovea. They were solitary in all cases, usually small and situated beneath the internal limiting membrane. Fluorescein angiograms showed no abnormalities except for blockage of normal fluorescence of retinal and choroidal vessels underlying the hemorrhage. Resolution of the hemorrhage mostly occurred within 1–2 months, with complete recovery of visual acuity. Possible pathogenetic mechanisms such as clotting disorders or Valsalva maneuvers could be found in only a few cases, while in the majority of cases SIMH seemed to be idiopathic.

Introduction

Solitary intraretinal macular hemorrhages (SIMH) have been previously reported as Valsalva retinopathy (Gass 1977), small unilateral foveal hemorrhages in young adults (Pitta et al. 1980), and microhemorrhagic maculopathy (Pruett et al. 1981), among other descriptive terms (Duke-Elder and Dobree 1967; Walsh and Hoyt 1969; Wise et al. 1971; Raspiller et al. 1975). These hemorrhages were not associated with retinal vascular diseases such as hypertensive and diabetic retinopathy, or with subretinal neovascular membranes such as in presumed histoplasmosis and senile macular degeneration. Records of 30 patients with a solitary intraretinal macular hemorrhage (SIMH), not associated with retinal or choroidal vascular disease, have been reviewed for this study, in which the delineate the clinical and angiographic features of SIMH and discuss possible pathogenetic mechanisms as well as predisposing conditions.

Patients and methods

Criteria for case selection were the presence of a SIMH with no ophthalmoscopic evidence of associated retinal or choroidal disease. All patients had a complete ophthalmologic examination at one of our institutions and were seen over a period of 15 years, between 1966 and 1981. Seventeen patients had fluorescein angiograms. Additional medical work-up to exclude hematologic disorders was performed in 15 patients. Nineteen patients were followed up several weeks to several months, mostly until complete resolution of the hemorrhage was observed.

Report of cases

A total of 30 patients with the above criteria were included in the study. There were 12 females and 18 males. Most often affected were young and middle-aged adults with a median age of 34 years (range 17–70 years). The age distribution is illustrated graphically in Fig. 1. All patients initially had complaints suggestive of positive central or paracentral scotomas with visual acuities ranging from 20/20 to 20/200. Twenty-five of the affected eyes were emmetropic (including minor refractive errors with spherical equivalents from −1.0 to +1.0 diopters). Four patients were myopic and one patient was hyperopic with spherical equivalents ranging between −3.0 and +3.5 diopters. All patients were phakic and affected unilaterally.

Biomicroscopically the hemorrhages were usually located beneath the internal limiting membrane, and typically there was a central reflex over the top of the hemorrhage (Fig. 2). The hemorrhages were solitary in all cases with shapes ranging from small dots to drop-like configurations of $\frac{1}{2}$ disc diameter. The internal limiting membrane was sometimes markedly elevated with a ring of radial stress folds, caused by the intraretinal drop of blood. The hemorrhage seemed to arise from the perifoveal capillary network.

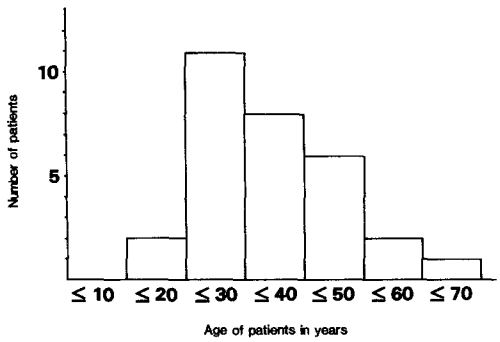

Fig. 1. Age distribution of 30 patients with solitary intraretinal macular hemorrhage (SIMH)

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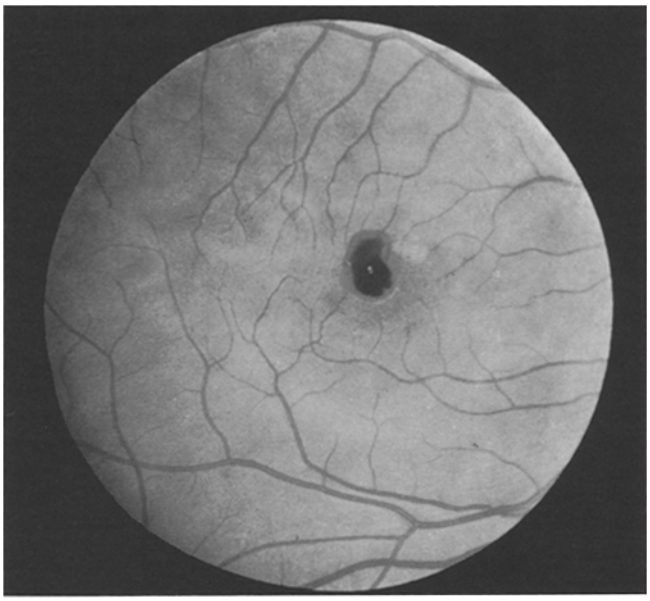

Fig. 2. SIMH arising from suprafoveolar capillary network and extending into fovea. Note light reflex over the center of the drop-shaped hemorrhage. Vision was 20/40

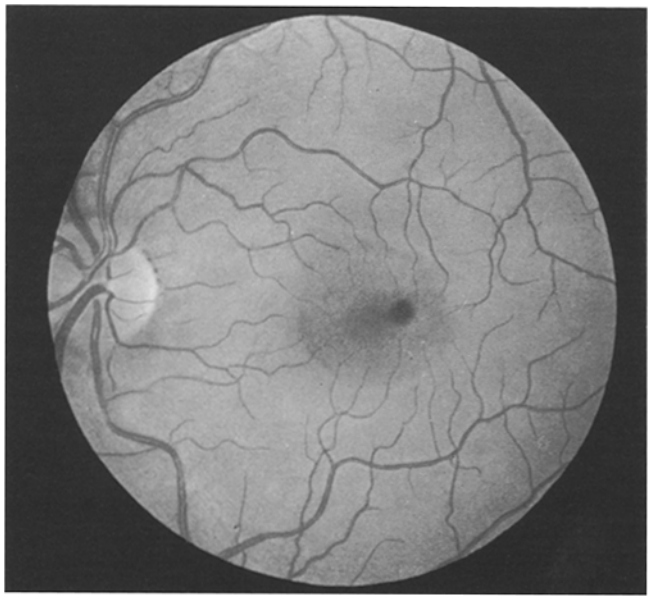

Fig. 3. Paracentral intraretinal macular hemorrhage not affecting visual acuity. Vision was 20/20 and hemorrhage resorbed within 4 weeks

While sometimes located paracentrally (Fig. 3) the hemorrhage often extended into the central fovea (Fig. 4). In one case, part of the hemorrhage had broken through the internal limiting membrane and caused a discrete reddish haze in the overlying vitreous. In two other cases, a minor part of the hemorrhage extended into the subretinal space. In one case there was an abnormal vascular pattern of the retinal vasculature at the posterior pole (Fig. 5), which did not affect visual acuity prior to the hemorrhage.

Angiographically the hemorrhage blocked the normal fluorescence of retinal and choroidal vessels (Fig. 4b). Any abnormalities of the retinal and choroidal vasculature or retinal pigment epithelium such as fluorescein leakage, aneurysms, capillary dropout, branch occlusion, subretinal neovascular membranes or window defects could not be observed.

Nineteen patients were followed up. In 6 of these the observation period was too short to document complete resorption of the hemorrhage. Of the remaining 13, 7 revealed complete resorption within 7 weeks (median – 4 weeks). The other 6 patients returned for follow-up visits some 3–6 months after the event. In these patients no evidence of a hemorrhage was noted. All patients with complete resolution achieved the visual acuity they had prior to the hemorrhage, and no changes of the retinal pigment epithelium were documented. The two patients who had extension of their intraretinal hemorrhage into the subretinal space were unfortunately lost to follow-up.

A single recurrence was documented in one of our patients, 4 weeks following the first hemorrhage. No familial occurrence or retinal tortuosity were observed in this patient.

History and medical examination provided some pathogenetic clues for the development of a spontaneous macular hemorrhage in only a minority of our cases. A prolonged prothrombin time was found in one patient, who was treated with a coumarin derivative for cardiovascular disease. Four patients had a history of Valsalva maneuvers such as coughing or strenuous exercise. Only one of these patients, however, reported abrupt visual loss after Valsalva. Three patients were pregnant and four were taking oral contraceptives. However, none had evidence of thromboembolic disease.

Discussion

We have chosen the descriptive term solitary intraretinal macular hemorrhage (SIMH) for macular hemorrhages not associated with retinal or choroidal vascular disease, as clinical appearance and course rather than an obvious pathogenetic mechanism are the common denominators in these cases. The clinical and angiographic features are so characteristic, that, for practical purposes, SIMH can be regarded as an entity.

Results of ophthalmoscopic and angiographic examination in our cases were essentially identical to those reported previously as Valsalva retinopathy (Gass 1977), small unilateral foveal hemorrhages in young adults (Pitta et al. 1980) and microhemorrhagic maculopathy (Pruett et al. 1981). The hemorrhages were solitary in all cases, usually small, not exceeding $\frac{1}{2}$ disc diameter and located superficially in the retina. Perforation of the internal limiting membrane with a discrete preretinal hemorrhage was observed in one case. In two cases part of the hemorrhages extended into the subretinal space. Hemorrhages apparently arose from perifoveal capillaries and often extended into the central fovea, sometimes in a bilobed fashion.

During fluorescein angiography the hemorrhage blocked fluorescence of underlying retinal and choroidal vessels without dye leakage. There was no evidence of associated disease of the retinal or choroidal vasculature or retinal pigment epithelium.

In all cases where follow-up was possible there was complete recovery of visual acuity. A single recurrence was noted in one case.

Duane (1972) coined the term Valsalva hemorrhagic retinopathy, implying that venous pressure elevations during

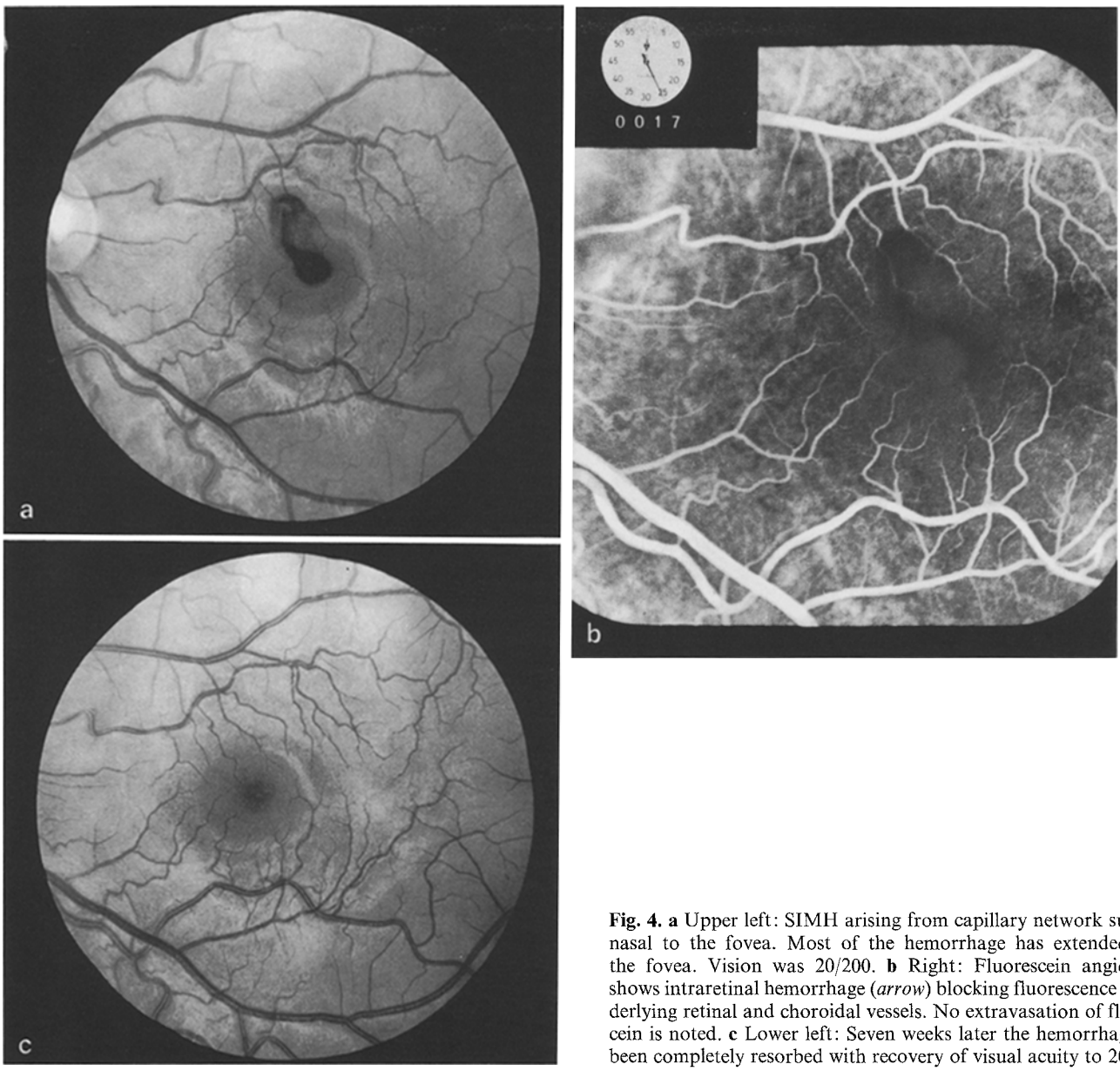

Fig. 4. a Upper left: SIMH arising from capillary network superonasal to the fovea. Most of the hemorrhage has extended into the fovea. Vision was 20/200. **b** Right: Fluorescein angiogram shows intraretinal hemorrhage (*arrow*) blocking fluorescence of underlying retinal and choroidal vessels. No extravasation of fluorescein is noted. **c** Lower left: Seven weeks later the hemorrhage has been completely resorbed with recovery of visual acuity to 20/15

a Valsalva maneuver would produce a particular retinopathy. However, two of the three patients he described had additional pathology (angioid streaks and severe anemia), which were probably the predominant causes of their hemorrhagic maculopathies. Gass (1977) adopted the term Valsalva retinopathy, referring to superficially located intraretinal macular hemorrhages with no associated retinal or choroidal vascular abnormality. Two of the three cases Gass reported in his macular atlas had a history of physical exertion such as tenesmus and vomiting. The term Valsalva retinopathy was later abandoned by other investigators (Pitta et al. 1980; Pruett et al. 1981) as they did not observe Valsalva maneuvers as the single common denominator among patients with hemorrhagic retinopathies identical to the ones described by Gass (1977).

It is likely that venous pressure elevations may contribute to the development of retinal hemorrhages when other factors occur that compromise vascular endothelium, such as clotting disorders or hypoxia. It is less likely, however, that a physiologic Valsalva maneuver alone, such as sneezing, coughing, or weight lifting would commonly initiate hemorrhagic maculopathy because of their truly rare association. Most of our patients as well as those from previous series did not report significant physical exertion preceding the macular hemorrhage, and predisposing hematologic disorders were also rarely observed. We therefore agree with Pruett et al. (1981), who state that this type of maculopathy is most often idiopathic.

Although rarely restricted to the macula, intraretinal hemorrhages without retinal or choroidal vascular diseases can be observed in patients with blood dyscrasias, especially when associated with thrombocytopenia as in leukemias and anemias (Rubenstein et al. 1968; Kincaid and Green 1983). Impaired platelet function as seen in thrombasthenia (Vaiser et al. 1975), following prolonged use of salicylates (Mortada and Abboud 1973), or associated with paraproteinemias (Rubenstein et al. 1968) also predispose to spontaneous intraretinal hemorrhages. Vessel wall abnormalities and disorders of blood coagulation factors have to be excluded as well. Patients with intraretinal macular hemor-

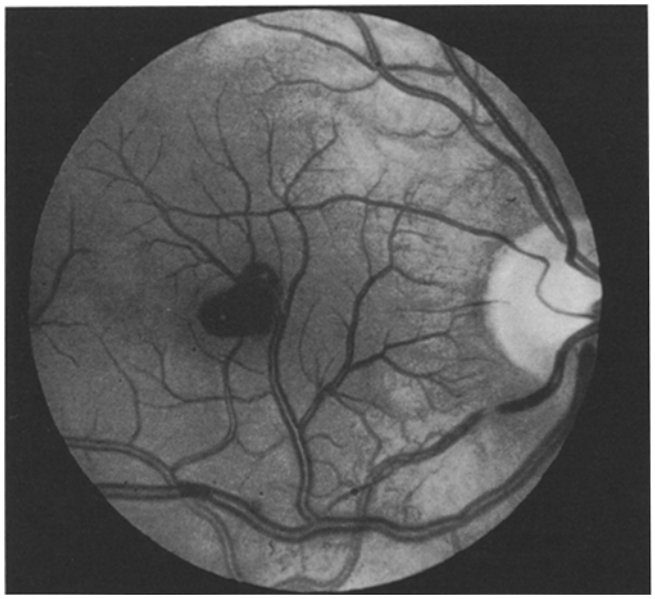

Fig. 5. SIMH associated with congenital abnormality of the retinal vascular pattern. Vision surprisingly was 20/30

rhages should be further examined for the presence of posterior vitreous detachment (Kanski 1975; Cibis et al. 1975) and the syndrome of familial retinal arteriolar tortuosity with retinal hemorrhage. The latter is characterized by familial occurrence of recurrent intraretinal hemorrhages and retinal arteriolar tortuosity with no other retinal vascular abnormality (Werner and Gafner 1960; Cagianut and Werner 1968; Kalina and Kaiser 1972; Goldberg et al. 1972).

Intraretinal spontaneous hemorrhages in the absence of retinal and choroidal vascular disease can also be observed in syndromes that are usually readily distinguished from SIMH, such as retinal hemorrhages in the newborn (Giles 1960; Besio et al. 1979) or high altitude retinopathy (Wiedman 1975; Shults and Swan 1975; Rennie and Morrissey 1975; Vogt et al. 1978). Superficial retinal hemorrhages have also been noted in association with arbovirus infections (Spitznas 1978) and oral contraceptives (Svarc 1977). The latter may be associated with a higher incidence of thromboembolic disease, which, however, was not found in any of our patients who took oral contraceptives.

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