

Case Report: Fluorescein Angiogram During Vasovagal Syncope

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We report a patient with hypopigmented fundi who maintained her position at the camera during fluorescein angiography despite an episode of vasovagal syncope. This fortuity allowed a rare glimpse of choroidal and retinal perfusion during this event.

Case Report

An otherwise healthy 44-year-old woman had painless, non-progressive, acute onset of mild blurred vision in her left eye seven days before evaluation. Her visual acuity was 20/20 in each eye, and no relative afferent pupillary defect was observed. Humphrey visual fields (30-2 Program) were normal. Examination of the left fundus showed optic disc edema inferiorly with nerve fiber layer and deep retinal hemorrhages (Fig. 1A). Fluorescein angiography was performed for possible retinal vascular disorders.

Following intravenous injection of 5 cc 10% sodium fluorescein, the patient became light-headed and uncommunicative, but did not otherwise convey her distress to the photographer. She managed to maintain her position at the camera for several minutes before she lost consciousness. At that time, her blood pressure was 60/0 and her pulse rate was 60. Five minutes later, the patient recovered spontaneously and her blood pressure increased to 104/76.

The angiogram showed marked delay in choroidal and retinal arterial filling with elapsed times of 29 and 45 seconds after injection, respectively (Fig. 1B-E). Six days later, a second fluorescein angiogram was obtained during which the patient remained alert and normotensive. This study exhibited normal circulation times (Fig. 2). Her blurred vision, optic disc edema, and retinal hemorrhages resolved gradually over the next two months.

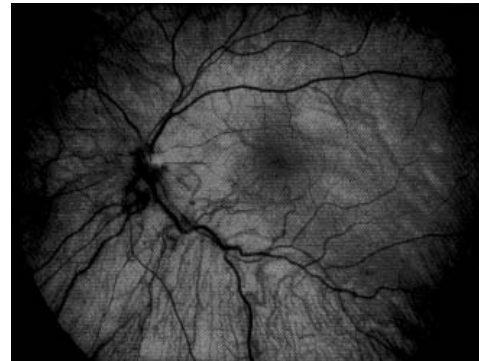


Figure 1a
Pre-injection

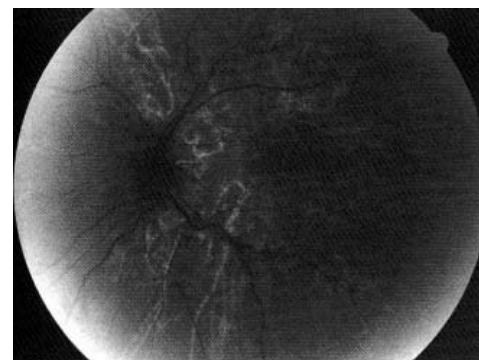


Figure 1 b
29.2 sec.

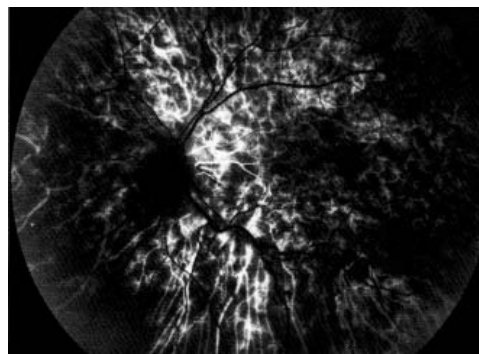


Figure 1 c
33.8 sec.

Comments

Light-headedness and syncope are occasional side effects of intravenous fluorescein angiography.¹ These reactions occur usually during the subject's first angiogram and, in most cases, are not associated with any specific pharmacologic effects of sodium fluorescein. Because of the self-limiting nature of these reactions, subsequent angiograms are usually not contraindicated.

Most patients who experience vasovagal syncope during angiography lose fixation and general muscle tonus, and thus, the angiogram is interrupted while the patient is attended. Our patient, however, was able to maintain her position at the camera until she lost consciousness. Marked delay in the perfusion of the eye was observed suggesting profound reduction in cardiac output. Fluorescein was not detected in the choroid until more than 29 seconds after initial injection, compared to the usual 8 to 15 seconds (Figure 1B and C.)² The interval between initial choroidal filling and retinal vascular

perfusion was 16 seconds, rather than the expected 1 to 2 seconds (Fig. 1D).²

Our patient had optic disc edema, hemorrhages, and two central venous trunks with an engorged inferior trunk, consistent with non-ischemic inferior hemi-central retinal vein occlusion.³ Although non-ischemic venous stasis may cause delayed retinal venous drainage, it has no effect on arm-to-choroidal and retinal arterial filling.

Most frequently, delayed arm-to-retina circulation times can be attributed to faulty injection technique, poor patient arm positioning, and vascular pathology. Perhaps the most common reason for apparent circulatory delay is inadvertent failure to remove the tourniquet prior to the bolus injection of dye. In the case of extremely large or obese patients, the fundus camera's vertical headrest support might cause compression of the venous system around the axilla. Subcutaneous injection can also cause a marked diminution of fluorescein intensity and filling patterns. Of the various pathologic causes, persistent arterial stenosis and chronic cardiac failure can compromise filling times. However, none of these other causes for delayed filling was seen in our patient.

The extreme delay in initial choroidal filling and subsequent retinal perfusion in our patient was caused by poor transient generalized perfusion and was not a sign of permanent vascular insufficiency.⁴ This was supported by the subsequent normal study. Although this finding has been suggested anecdotally, our report provides objective graphic evidence of this phenomenon. We believe the angiographic timing data demonstrated in our report to be consistent with known pathophysiologic principles.

These findings may be misinterpreted if the clinician is unaware of the instability of the patient's vital signs during angiography. Therefore, in cases where angiographic timing is crucial to the interpretation, vasovagal syncope during the procedure may influence the analysis of the results.

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Key Words: Vasovagal, Syncope, Fluorescein, Angiogram, Perfusion, Vascular.

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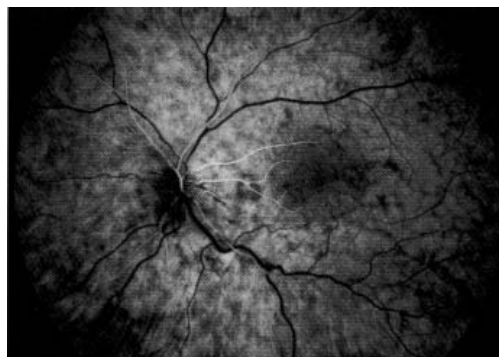


Figure 1d
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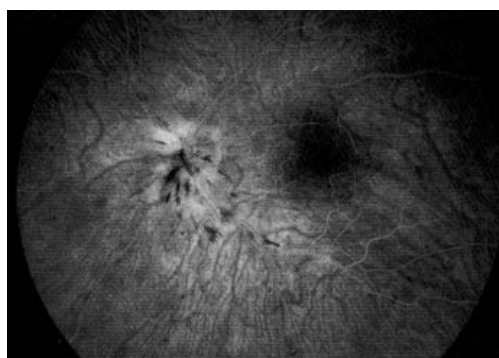


Figure 1e
271.8 sec.

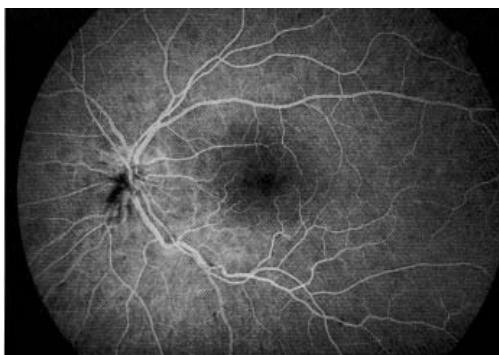


Figure 2
Six-day
follow-up/
30.6 sec.