The acute vitreous haemorrhage

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SUMMARY One hundred cases of acute vitreous haemorrhage have been analysed prospectively. It was possible to identify the cause of the haemorrhage at presentation in 79% of the cases. 40% were due to retinal tears and only 6% were associated with diabetic retinopathy.

An acute vitreous haemorrhage presents as a shower of floaters with or without blurring of vision. On examination blood is found in the vitreous cavity, either within the vitreous gel in the retrohyaloid space or both. A wide variety of conditions may lead to bleeding of this kind. In 2 recent series diabetic retinopathy was found to be the commonest cause of nontraumatic vitreous haemorrhage.1 2

This paper presents an analysis of causes of vitreous haemorrhage in a prospective series of 100 consecutive cases as seen in a busy ophthalmic casualty department. We emphasise the possibility of definitive diagnosis at initial presentation in a high proportion of cases, the relatively low number of patients with diabetic retinopathy, and conversely the high proportion of cases with retinal tears.

Materials and methods

One hundred consecutive patients presenting with acute vitreous haemorrhage were entered prospectively into the study. The patients' medical and ophthalmic history, presenting symptoms, and clinical findings were recorded on a standard form. Each patient had a full ophthalmic examination including slit-lamp biomicroscopy using the Goldmann 3 mirror lens and indirect ophthalmoscopy with scleral depression. After the initial assessment in the casualty department the patients were generally admitted for further investigation and management. The case notes of all the patients were reviewed after 1 year.

Results

There were 55 male and 45 female patients. Ninety were Caucasian and there were 10 Negro.

Table 1 Causes of vitreous haemorrhage

<table>
<thead>
<tr>
<th>Cause</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal tears</td>
<td>40</td>
</tr>
<tr>
<td>Trauma</td>
<td>12</td>
</tr>
<tr>
<td>Retinal vein occlusion</td>
<td>10</td>
</tr>
<tr>
<td>Diabetes</td>
<td>6</td>
</tr>
<tr>
<td>Retinal detachment</td>
<td>4</td>
</tr>
<tr>
<td>Retinoschisis</td>
<td>4</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4</td>
</tr>
<tr>
<td>Posterior vitreous detachment</td>
<td>3</td>
</tr>
<tr>
<td>Malignant melanoma</td>
<td>3</td>
</tr>
<tr>
<td>'Vasculitis'</td>
<td>3</td>
</tr>
<tr>
<td>Sickle cell disease</td>
<td>1</td>
</tr>
<tr>
<td>Coats's disease</td>
<td>1</td>
</tr>
<tr>
<td>Disciform degeneration</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension with macroaneurysm</td>
<td>1</td>
</tr>
<tr>
<td>Undiagnosed</td>
<td>4</td>
</tr>
<tr>
<td>Lost to follow-up</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

HISTORY 66% of patients complained of floaters only. Photopsia was a relatively uncommon symptom and was seen combined with floaters in a further 15% of cases. 19% presented only with blurring of vision. 11% of patients had had a previous episode of vitreous haemorrhage. 12% gave a history of ocular trauma immediately preceding the onset of their symptoms. 5% were on treatment for hypertension and 6% were diabetic.

AETIOLOGY

The cause of the vitreous haemorrhage was diagnosed on presentation in 79% of patients. Of the remainder 14% were diagnosed on follow-up, 4% remained undiagnosed after 1 year and 3% were lost to follow-up (Table 1).

Rhegmatogenous causes. 40% of patients had a retinal tear. The majority of these (38%) were
diagnosed on presentation; the remaining 2 cases were diagnosed on follow-up and did not progress to a retinal detachment. Three patients had flat retinal tears which had been previously treated with cryotherapy. In all these cases a retinal tear was bridged by a patent retinal vessel and showed signs of vitreous traction on the operculum. In the absence of any other pathology this combination of signs was taken as evidence of the source of bleeding.

Four patients presented with acute vitreous haemorrhage as the first symptom of retinal detachment. All these patients were diagnosed at presentation.

**Trauma.** In 12 cases the vitreous haemorrhage was related to blunt trauma. Five patients had posterior vitreous detachment at the time of presentation. In these cases the cause of bleeding was attributed to an acute posterior vitreous detachment in association with the trauma. However, in 2 cases no posterior vitreous detachment was identified, and haemorrhage was present entirely in the vitreous gel. In the remaining 5 cases extensive haemorrhage made the assessment of the vitreous and the retina impossible.

**New vessel formation.** Nine patients presented with bleeding from neovascular complexes associated with a retinal vein occlusion. Six of these patients were diagnosed on presentation; 3 patients had extensive bleeding and diagnosis was only possible on follow-up. A further 6 patients had diabetes. In 3 of these there was evidence of neovascularisation, but in 1 new vessels could not be identified. In the remaining 2 patients assessment was impossible due to extensive bleeding. One patient had sickle cell retinopathy.

**Retinoschisis.** This was observed in 4 patients with vitreous haemorrhage. The amount of haemorrhage was minimal and tended to be in the retrohyaloid space. Abnormal paravascular vitreoretinal adhesions with punctate retinal haemorrhage were also observed in the area of retinoschisis.

**Acute posterior vitreous detachment.** Three patients presented with an acute posterior vitreous detachment which was recognised as the combination of posterior vitreous detachment, punctate retinal haemorrhages, and the presence of blood in the retrohyaloid space. No other ocular pathology was found in any of these 3 patients.

**Hypertension.** In 5 patients the vitreous haemorrhage was associated with systemic hypertension. In 1 of these patients a macroaneurysm had ruptured and in the remainder there was no apparent source of bleeding.

**Miscellaneous.** 'Retinal vasculitis' was associated with vitreous haemorrhage in 3 patients, though in none of these cases could definite neovascularisation be identified. In 3 patients a vitreous haemorrhage was the initial symptom of a choroidal malignant melanoma. Two of these were diagnosed on presentation and 1 was diagnosed on follow-up. There was one case of Coats's disease, and there was one case which appeared to have bled from disciform degeneration of the macula.

**Undiagnosed.** Four cases have remained undiagnosed. In 1 case a large vitreous haemorrhage has failed to clear, the eye has now no perception of light, and it is assumed that the patient has a total retinal detachment. In the other 3 cases a diffuse vitreous haemorrhage was found at the time of presentation, but this has cleared spontaneously with a good visual result. No pathology has been found except that in all 3 cases a posterior vitreous detachment is now present.

Three patients have been lost to follow-up.

**Discussion**

In this series the commonest single diagnosis (40%) was that of a retinal tear. Only 5% of these could not be diagnosed at the time of initial presentation. The majority were fresh tears associated with the recent onset of symptoms. However, 3 patients with treated horseshoe tears bled from a patent retinal vessel crossing the tear. An earlier report suggested that anterior extension of the retinal tear was responsible for recurrent bleeding. In our cases the retinal vessel seemed a more likely source of haemorrhage. Such crossing vessels are prone to cause recurrent haemorrhages until they either become occluded or their course interrupted. Acute posterior vitreous detachment in the absence of a frank retinal tear was identified in a further 3 patients.

In contrast to the studies of Linfoff et al. and Morse et al. we found diabetic retinopathy to be an uncommon cause of an acute vitreous haemorrhage. We feel this partly reflects a difference in the selection of our patients. In this hospital diabetics are managed in a specific diabetic clinic and therefore tend not to present with recurrent bleeding to the casualty department. In support of this, of our 6 patients with diabetic retinopathy 5 were experiencing their first vitreous haemorrhage at the time of presentation.

Acute vitreous haemorrhage in association with retinoschisis is well recognised, although the relatively high number of cases in this series is unusual. In these patients we were able to identify abnormal paravascular vitreoretinal adhesions which appeared to be the cause of bleeding. In all cases the haemorrhage was slight and cleared rapidly.
Choroidal malignant melanomas infrequently give rise to vitreous haemorrhage. Such bleeding probably implies necrosis of the tumour mass. In 1 patient the vitreous haemorrhage was sufficiently dense that the correct diagnosis was not initially suspected.

It is interesting that systemic hypertension, although frequently thought to be associated with vitreous haemorrhage, was relatively unusual as an aetiological factor. Ten of our patients had systemic hypertension, but 5 of these were associated with a branch vein occlusion and consequent neovascularisation, 1 with a macroaneurysm which had ruptured, and in only 4 cases did the hypertension itself appear to be directly related to the vitreous haemorrhage.

This study demonstrates that the cause of an acute vitreous haemorrhage can be accurately established at presentation in almost 80% of cases.

Retinal tears form the largest single group and can be almost always identified (95%).

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