**Clinical Algorithms**

**Loss of vision**

MICHAEL JAMIESON

A common and often major handicap, visual loss may be preventable and is often symptomatic of treatable intraocular and extraocular disease. Diagnosis of its underlying cause is often possible without specialist experience or equipment. Early diagnosis by a general practitioner or general physician may lead to early treatment and will be a useful guide to prompt and appropriate referral, avoiding time consuming and perhaps unnecessary referral to opticians and ophthalmologists. In particular, chronic glaucoma may be detected and treated early, refractive errors diagnosed and referred for retnoscopy, diabetic and hypertensive retinopathy found and appropriately managed, and disorders primarily of the central nervous system identified. This algorithm attempts to provide routes to diagnosis in cases where visual loss is the dominant symptom. Conditions in which pain predominates but where visual impairment may be present—for example, acute glaucoma, acute iritis—are not discussed. Likewise, conditions that are common world wide but rare in the United Kingdom—for example, vitamin A deficiency and infective diseases such as trachoma and onchocerciasis—are not considered further.

**History**

In all cases the history should take into account onset, progress, duration, and extent of visual loss. In particular, the clinician must distinguish between sudden and gradual loss, bearing in mind that gradual unilateral impairment may go unnoticed until vision becomes impaired (perhaps trivially) in the "good" eye. To distinguish between central and peripheral visual loss is also useful. Central visual loss presents as impairment of visual acuity and generally implies defective retinal image formation (through refractive error, lens, or vitreous opacity) or macular or optic nerve disease. Peripheral visual loss is more often silent but may present as an absolute or relative blind spot or as navigational difficulty (including night blindness and tendency to collide with unseen objects). Peripheral loss generally implies either diffuse non-macular retinal disease or a defect in the visual pathway (optic nerve, chiasm, radiation, and cortex). There are, however, areas of overlap as shown in the algorithm, and disorders may coexist.

The drug history may be relevant. Visual disorders that have been associated with drugs are listed in the table. The clinical importance and prevalence of these associations vary, and readers are referred to a more detailed discussion of the subject. ¹ Few of these associations are predictable.

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**Visual disorders associated with drugs**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Drug</th>
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<tbody>
<tr>
<td>Corneal opacities</td>
<td>Chlorpromazine, Chlorpromazine, Hydroxychloroquine, Indomethacin, Vitamin D, Amiodarone</td>
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<tr>
<td>Precipitation of acute narrow angle glaucoma</td>
<td>Antihistamines, Tricyclics, Mydriatics drops</td>
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<tr>
<td>Refractive changes</td>
<td>Thiocides</td>
</tr>
<tr>
<td>Lens opacities</td>
<td>Phenothiazines, Corticosteroids</td>
</tr>
<tr>
<td>Retinopathy</td>
<td>Chloroquine, hydroxychloroquine, Phenothiazines</td>
</tr>
<tr>
<td>Papilloedema (2° to benign intracranial hypertension)</td>
<td>Corticosteroids, Tetracyclines, Oral contraceptives</td>
</tr>
<tr>
<td>Optic neuropathy</td>
<td>Ethambutol, Dusodium, Ethanol (Tobacco)</td>
</tr>
</tbody>
</table>

**Examination**

Examination should include assessment of visual acuity and visual fields, fundoscopy, and, when appropriate, pinhole testing and measurement of intraocular pressures. Near visual acuity is estimated with newsprint or, more formally, with British Faculty of Ophthalmologists reading type; distant acuity with Snellen or E charts. Confrontation testing will detect major defects in the visual field; smaller, relative, and colour defects are more likely to be missed, and formal mapping will be necessary if no field defect has been found to explain a volunteered blind spot.

Intraocular pressures should be assessed in older patients, ideally in all cases but particularly when symptoms (characteristically, "halos" seen around lights) or fundal findings suggest chronic glaucoma. With practice, digital estimation will detect a considerable increase in pressure but is unreliable in assessing smaller rises. If a tonometer is available referral to an ophthalmologist should be considered. The pinhole test is useful if refractive error is suspected: a pinhole roughly 1 mm in diameter held in front of the eye will improve acuity in those with refractive errors (but also in some patients with early corneal or lens opacities) but will tend to worsen the acuity of those with retinal or optic nerve lesions. The pupillary light reflexes are usually preserved in chronic visual loss, even in the face of severe impairment. Acutely absent ipsilateral direct and contralateral consensual reflexes suggest retinal arterial occlusion or injury to the optic nerve.

**Management**

Opportunities for management by the non-specialist are rather more limited, and most cases need appropriate referral. Chronic
Loss of vision

- Sudden
  - Transient? No
  - Unilateral? No
  - Bilateral

- Transient
  - Causes include MIGRAINE, TRANSIENT CEREBRAL ISCHAEMIA

- Unilateral
  - Is there a history of injury? No
    - Test pupillary light reflex
  - Is pain a prominent feature? Yes
    - Present
      - Causes include OCCIPITAL INFARCTION secondary to basilar artery embolism
    - Absent
      - Suggests CENTRAL RETINAL ARTERIAL OCCLUSION
      - Test visual fields. HOMONYMOUS HEMIANOPIA may present as apparent visual loss (see 2)

- Irregular or peripheral defect
  - Examine fundus for evidence of RETINITIS PIGMENTOSA, DISSEMINATED CHOROIDITIS
  - Consider FUNCTIONAL or HYSTERICAL VISUAL LOSS (tabes dorsalis?)

- Absent pupillary light reflex suggests OPTIC NERVE INJURY

- The assessment of the painful red eye is outside the scope of this algorithm. Causes include ACUTE CONGESTIVE GLAUCOMA, ANTERIOR UVEITIS

- Ask specifically about headache. Examine for evidence of TEMPORAL ARTERITIS Check ESR

- Examine fundus for retinal pallor, "cherry red" macula, embolus in central retinal artery

- Consider causes of unilateral loss occurring bilaterally. Patients may not be aware of pre-existing unilateral visual impairment until the good eye becomes affected
Lesson of the Week

Necrobacillosis: a forgotten disease

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Abstract

Over four years five previously healthy young adults developed necrobacillosis, a severe septicaemic illness caused by Fusobacterium necrophorum. The infections were characterised by sore throat followed by rigors and the formation of metastatic abscesses and all caused considerable diagnostic confusion.

Introduction

Necrobacillosis—infection with Fusobacterium necrophorum—is a severe suppurative illness that before the advent of antibiotics was often fatal. The presenting features are usually characteristic and were described in detail in 1936 by Lemierre, who suggested that the diagnosis may be strongly suspected on clinical grounds alone. Despite the intense interest in anaerobic infections over the past decade F necrophorum is seldom specifically mentioned in reports and necrobacillosis is now rarely, if ever, diagnosed. Over four years we saw five patients with this illness, all of whom showed the classic features of the disease.

Case reports

Case 1—An 18 year old man developed a severe sore throat and over three days became very ill with rigors, pain in the back and shoulders, pleuritic chest pain, haemoptysis, and dyspnoea. On admission he had cervical lymphadenopathy and jaundice. A chest x ray film showed bilateral abscesses and a pleural effusion. The white cell count was $21.4 \times 10^9/\text{l}$, blood urea concentration was raised at 13.9 mmol/l (83 mg/100 ml), and bilirubin concentration was 38 $\mu$mol/l (2.2 mg/100 ml). Initial treatment with intravenous erythromycin and cefuroxime produced no clinical improvement, and a week later shadowing was visible in the chest x ray film, which gallium scanning and computed tomography suggested was a paravertebral abscess. Metronidazole was added empirically to his treatment, and a few days later F necrophorum was isolated from two of the eight blood cultures taken on admission. Metronidazole was continued for two months, during which time he made a slow but complete recovery.

Case 2—A 20 year old woman developed a severe sore throat and over one week became very ill with fever, cough, abdominal pain, and dyspnoea. On admission she had bilateral cervical lymphadenopathy and jaundice. A chest x ray film showed extensive bilateral consolidation, which subsequently cavitated. The white cell count was raised at 17.0 $\times 10^9/\text{l}$, blood urea concentration raised at 38.3 mmol/l (230 mg/100 ml), and serum bilirubin concentration raised at 178 $\mu$mol/l (10.4 mg/100 ml). Treatment was initially with intravenous gentamicin, metronidazole, and ampicillin. Her abdominal pain worsened and the results of ultrasound scanning and computed tomography were highly suggestive of a perinephric abscess, but the result of laparotomy was entirely normal. Cultures of blood and pleural fluid taken on admission subsequently grew F necrophorum and a microaerophilic streptococcus. Metronidazole and benzylpenicillin, and then oral metronidazole and amoxycillin, were administered during her lengthy and severe illness, which was further complicated by joint effusions in hip and knee and an episode of obstruction of the small bowel. She eventually recovered completely.

Case 3—A 21 year old man developed a sore throat and over one week became very ill with high fever, pleuritic chest pain, haemoptysis, and severe dyspnoea. On admission cervical lymphadenopathy and haematuria were noted. A chest x ray film showed extensive bilateral consolidation and effusions. The white cell count was raised at 23.7 $\times 10^9/\text{l}$, blood urea concentration was normal, and serum bilirubin concentration was raised at 50 $\mu$mol/l (2.9 mg/100 ml). He improved after initial treatment with intravenous erythromycin but after a further five days again developed a fever. At this stage F necrophorum was