THE RETROVIRUS OF A THOUSAND FACES

After two decades in the neurological trenches of a busy, developing world, general hospital, one hopes to have reached a state in which, by and large, one's adversary is usually clinically recognizable. Rising to the challenge of making a diagnosis, and, often equally important, knowing what to suggest as the wisest course of action for each individual patient, is part of the job satisfaction. So it comes as something of a shock to find yourself robbed of certain clinical impressions that you may have comfortably harboured. For this is what happens when you come face-to-face with an epidemic in which the nervous system is involved in over 90% of the patients, and in which the neurological picture has a thousand faces. I refer to HIV/AIDS. In our hospital, the daily general medical ward intake of about 30 patients now contains about 40% with HIV infection. Only a few years ago, the percentage would have been negligible. And the projections are that the number of new cases will continue to rise for at least several years.

For neurologists, the first illness that earned the description ‘The Great Imitator’ was, of course, syphilis. The range of clinical presentations was so great, that a standing rule here was that all admissions must, at the very least, have a serological test for syphilis. That remains true on our neurology ward. Perhaps the next illness that, with somewhat less justification, gained the title, was systemic lupus erythematosus. But the third horseman of this apocalypse, HIV, is the latest and most deadly title-holder. We now have to have a very good reason not to test for HIV in our practice. A cursory glance at a modern textbook will reveal a depressingly long list of neurological syndromes in HIV/AIDS. It soon becomes clear that the lists are so inclusive as to be of limited practical use, and there are also clinical pictures that have not yet made the lists and that we have never seen before in other illnesses.

Experienced clinicians appear to work, so it has been reported, by quickly forming a syndromic diagnosis, and then sticking out their necks to pick a winner from a short list of likely causes that simply presents itself without conscious effort. If that is so, and I would agree with the premise, then a typical day on our service goes something like this. A thin, young woman with a typical Bell’s palsy – possibly early HIV with a still-preserved CD4 lymphocyte count but altered immunity. A man with a typical Guillain–Barré syndrome but a few too many lymphocytes in the CSF – almost certainly HIV. A severe right thoracic chest wall pain – probably herpes zoster but without the rash as there is a muted inflammatory response in HIV. A subacute, painful cauda equina syndrome – probably late-stage HIV with a markedly depressed CD4 lymphocyte count, a high viral load, and cytomegalovirus radiculitis. A severe burning pain on the soles of the feet – often an HIV-associated sensory axonal polyneuropathy. A subacute, progressive thoracic myelopathy – exclude tuberculosis, syphilis and lymphoma before considering the vacuolar myelopathy of HIV infection. A subacute, areflexic paralysis of both legs but with retained sensory and sphincter function and normal arms – almost
certainly a lumbo-sacral anterior radiculitis associated with HIV. Myalgia and a proximal myopathy with ragged red fibres - likely to be due to mitochondrial change due to antiretroviral therapy such as Zidovudine. Transient headache and a brief rash - may be sero-conversion shortly after acquiring HIV infection. Seizures and a CT head scan showing a single enhancing lesion in the right frontal lobe - assume the most likely cause, toxoplasmosis, and treat accordingly. Failure to respond, and tuberculoma and lymphoma are likely. A CSF containing a mildly raised protein and a mild pleocytosis - always the question of whether or not this is related to the present symptoms, or due to an underlying HIV infection in the long asymptomatic phase while the immune system is being slowly eroded. Three weeks of headache with mild neck stiffness and a raised CSF protein but no cells and a depressed glucose - probably cryptococcal meningitis despite the negative antigen test, so request enriched medium culture of the fungus.

Once the clinical suspicion of HIV is raised, there is, of course, the need for HIV testing. Our ethical rules state that, in general, a person may only be tested for HIV at their own request and after adequate pretest counselling. Counselling is not merely reading a leaflet, but a structured interview with the doctor, which fully covers the advantages and disadvantages of taking the test. This may be challenging in a multicultural country with 11 official languages. Post-test counselling should also be provided. There is still a great deal of denial of the problem, both by individuals and, less understandably, by some health authorities. Stigma is also very much in evidence (see this issue's editorial on p.2).

And then there is the matter of treatment. In most circumstances, the combination of three synergistic antiretroviral agents remains the standard of care. Unfortunately, the present high price of these drugs means that the public health services cannot provide them. For the most part, we can but treat opportunistic infections like tuberculosis, syphilis, toxoplasmosis and cryptococcosis and provide general support and care.

As jobbing neurologists we have found ourselves in a new arena. There is a great press of patients and we are no longer strangers to the rural primary care clinics as we try to assist in devising treatment algorithms that suit local conditions and do not start with 'First, obtain an MRI head scan'. Ethical dilemmas of course abound where there is the mix of ill patients and limited resources, and these have made us very much more alert to the strength of the evidence that our often costly treatments are indeed reasonably useful.

As Charcot might well have sighed and said, 'Out of Africa there is always something new!'