Poor people get cancer too

To the Editor: Three articles in your July 2007 issue need to be brought together to reveal the bigger picture.

Sa'ad Lahri[1] writes that the failure of a Third-World general practitioner to do a Pap smear is inexcusable and negligent. He appears not to appreciate the logistics of doing Pap smears in private general practice. The patient has to attend and pay a fee – perhaps R150 in the Third-World practice; the general practitioner sends the slide to a pathologist who charges about R100; the patient then has to return for the result of the smear, making the cost for that service about R400.

Sydney Rosen *et al*.[2] quantify the costs, including transport and loss of earnings, to patients of obtaining free antiretroviral drugs at state and charitable clinics. These costs, which are equally significant in Third-World private practice, must be added to the consultation and pathology fees. This is partly why, especially in the Third World, cancer patients present late in their disease process.

Ralph Kirsch[3] deplores the Western Cape Department of Health's budget reductions at the Groote Schuur and Tygerberg teaching hospitals, and the minimum 6 weeks' delay before indigent cancer patients are treated. At the time of writing this letter, patients with carcinoma of the cervix wait 3 months for an appointment at the gynaecology outpatient department at Groote Schuur Hospital. It takes the National Public Health Laboratory 2 months to report Pap smears – a service done in 2 days by private pathologists. Such patients therefore wait at least 5 months before being assessed, at which stage the cancer will probably be too far advanced for anything more than palliation.

Whether we practise medicine in the First or the Third World, in the state or the private sector, money available for disease management is limited and fixed. Using waiting lists is the state's method of rationing a service for which the demand cannot meet the supply. The National Health Service in the UK continues to use this technique after half a century.

Our political masters and health economist advisors appear to have decided that the limited funds are more cost-effectively spent on, for example, antiretroviral drugs for HIV-positive patients, thereby keeping them productive. In comparison the clinical and cost-efficacy prognosis for many neoplastic diseases is much worse. I am glad I do not have to make this difficult decision.

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1. Lahri S. Pap smears in the Third World (Letter). *S Afr Med J* 2007; 97: 474-475.
2. Rosen S, Ketlhapile M, Sanne I, DeSilva MB. Cost to patients of obtaining treatment for HIV/AIDS in South Africa. *S Afr Med J* 2007; 97: 524-529.
3. Kirsch R. Poor people get cancer too (From the Editor). *S Afr Med J* 2007; 97: 471.

Simultaneous filarial infection of the pleura and breast

To the Editor: Lymphatics are the most favoured site of filarial localisation, although *Wuchereria bancrofti* has been found in various atypical sites such as the thyroid, pericardial effusion, bone marrow, bronchial aspirate, etc.[1] An unusual simultaneous involvement of the pleura and breast by *W. bancrofti* is presented.

A 30-year-old woman presented with a swelling in the left breast of 4 months' duration, and left-sided chest pain, occasional dry cough and breathlessness on exertion for the past 3 weeks.

The breast lump was 3 x 2 cm in size, located in the upper inner quadrant of the left breast. It was slightly tender to the touch, firm in consistency and fixed, and the overlying skin was normal. Physical examination of the chest suggested a left-sided pleural effusion.

Peripheral blood examination showed eosinophilia (eosinophils 18%, and absolute eosinophil count 1 296/µl). Pleural aspirate showed an exudative pattern, and cytological examination revealed a predominance of lymphocytes, few eosinophils, mesothelial cells and microfilariae. The microfilariae were sheathed and their terminal ends (tips) were devoid of nuclei, characteristic of *W. bancrofti*. Needle aspiration of the breast lump showed a chronic inflammatory infiltrate, with a few interspersed eosinophils, occasional granuloma formation and parts of gravid adult worm. A few microfilariae of *W. bancrofti* were also seen. A thick peripheral blood smear examination (nocturnal) showed numerous live microfilariae.

The patient was treated with di-ethyl-carbamazine citrate, at a dose of 6 mg/kg/day for 4 weeks. Following treatment there was complete resolution of the pleural effusion, breast lump and peripheral blood eosinophilia.

Filariasis is most commonly caused by *W. bancrofti*, transmitted by mosquitoes of the *Cultex*, *Anopheles* and *Aedes* genera. The adult worm resides in lymphatic channels, while microfilariae circulate in peripheral blood. It has been suggested[2] that microfilariae appear in tissue fluids and exfoliated surface material due to lymphatic or vascular obstruction. The dead degenerating adult worms usually produce a severe inflammatory reaction (eosinophilic) and granuloma formation, whereas an intact healthy filarial worm may produce only a minimal reaction. Menon and Annamalai[3] proposed that living worm may also induce inflammatory changes in the surrounding tissue, explaining that 'parturition of the female in a blocked vessel may be followed by a discharge of embryos in surrounding tissue, and this may probably initiate inflammatory changes'. The above hypothesis can be used to explain the microfilarial seeding of the pleura and breast in our case.