Top-up fees are not the solution

Sir,

The government has welcomed Professor Richard's (2008) report supporting the introduction of 'top-up' fees, allowing patients who have money to buy drugs that are unavailable through the NHS. This could bring a two-tier system into health care, where those who can afford treatment do well while patients who can't do not.

But why should there be two tiers of health provision? Why should it be that people who have funds can buy the right to life, while those who do not, may not? The rationale behind top-up fees is that new drugs are too expensive for the state to fund. New drugs are expensive, but pharmaceutical companies price them to recoup costs and provide profit to fund research that provides the next generation of drugs that provides profit and funds research.

This system has worked very well for the last 50 years and has provided treatments for conditions previously thought to be incurable. The 'market' and the profit motive have driven research and allowed us to be in the remarkable position of being able to double survival or cure many conditions. Why can't we follow the lead of other European countries, where drug costs are controlled by direct negotiation, allowing the introduction of new agents in a relatively short period of time.

Europe has the drugs that it needs, but in the UK we have poor access to modern treatment because the brake on costs is applied by the National Institute for Health and Clinical Excellence (NICE) and the primary care trusts (PCTs). They produce extraordinarily leisurely decisions, that are against the best interests of the country that they are meant to serve. It is not as if the UK's drug budget is enormous. We spend two thirds of the European average on cancer drugs, and the cost of these agents is considerably less than the amount spent on constipation cures.

So, what is the solution? Well, forget top-up fees. Let's tackle the root cause of the problems, and banish the idea of inequality in health care. Let's free up the health-care system from the bureaucracy that tramples freedoms. It is estimated that there are £5 billion of administrative costs entangled within the 150 PCTs that provide us with a postcode lottery for prescription. Let's shut down NICE, and liberate the enormous costs to our health-care system that this enterprise, and the costs of the legal challenges to its decision making, generates. We can have care for all if we reform (shut down) NICE and the PCTs.

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Richards M (2008) *Improving access to medicines for NHS patients.* Department of Health, London

Acute severe pancytopenia associated with clopidogrel

Sir,

Clopidogrel is widely used to treat acute coronary syndromes and ischaemic stroke. An analogue of ticlopidine, clopidogrel was adopted as an alternative as a result of severe adverse haematological reactions associated with ticlodipine. The mechanism of this reaction is unclear. Studies suggest that clopidogrel is as safe as aspirin with regard to haematological adverse reactions (Harker et al, 1999).

A 87-year-old woman with known arterial disease was admitted with chest pain via the accident and emergency department. On the basis of her risk factors, clinical history and electrocardiographic changes acute coronary syndrome was diagnosed and she was treated with clopidogrel, aspirin and dalteparin. Her baseline haematological parameters were normal. Her concurrent medications included omeprazole, digoxin, perindopril, frusemide, calcichew, folate, co-dydramol and salbutamol inhalers, none of which had been started in the weeks before her admission.

After 3 days she started bleeding from the mucous membranes in her mouth. A full blood count showed haemoglobin of 5.9 g/dl, white cell count 0.4×10^9/litre (neutrophils 0.1%, lymphocytes 0.3%) and platelets 3×10^9/litre. A blood film showed polychromasia and confirmed severe pancytopenia. Bone marrow aspirate showed myeloid hyperplasia with a good number of megakaryocytes and no excess blast or lymphoid cells. A direct Coombs test was negative. Clopidogrel was stopped within 24 hours of diagnosis of pancytopenia.

The patient was treated with 6 units of blood, 7 pools of platelets and granulocyte colony-stimulating factor (300 μg daily for 2 days). She developed neutropenic sepsis secondary to an *Escherichia coli* urinary tract infection, treated with broad-spectrum intravenous antibiotics. She made a complete recovery. Her haemoglobin level was 13.2 g/dl, white cell count 7.7×10^9/litre (neutrophils 6.4%) and platelets 379×10^9/litre, 9 days after pancytopenia was first recorded.

As her pancytopenia resolved on discontinuation of clopidogrel and continuation of all other medications, this is likely to have been an adverse drug reaction.

Clopidogrel has been associated with fatal aplastic anaemia (Trivier et al, 2001) and hypersensitivity reactions, including systemic inflammatory response syndrome (Wolf et al, 2003; Doogue et al, 2005). The haematological indices in this case are more severe than in any previously recorded case.

In the CAPRIE trial (Harker et al, 1999) the rate of severe thrombocytopenia was 0.19% for clopidogrel *vs* 0.10% for aspirin and thrombocytopenia, and neutropenia rarely occurred with clopidogrel. There were no data about rates of pancytopenia.

Clopidogrel is so widely prescribed that clinicians in both primary and secondary health care should be aware of the potentially fatal side effect of severe pancytopenia.

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