

Failing older hearts? Of myths and leftovers

Sir,

Dr McIntyre's assertion (vol 67(4), 2006, p. 176) that the absence of robust echocardiographic criteria for left ventricular diastolic function favours the more pragmatic approach of characterizing such patients simply as having heart failure with preserved ejection fraction is to be applauded. This characterization would involve reinstating the combined clinical and radiographic criteria for the diagnosis of heart failure (Marantz et al, 1988), leading to more uniform, and probably more robust characterization of this syndrome regardless of aetiopathogenesis.

What we presently have instead is such a lack of uniformity that even the mere association of poor New York Heart Association functional class and sub-normal left ventricular ejection fraction (LVEF) is deemed, in some instances, sufficient to characterize chronic heart failure (Granger et al, 2003).

More decisively, a clinically oriented characterization would be an acknowledgement of the mismatch between sub-normal LVEF and clinical stigmata, exemplified by the fact that 20% of subjects with LVEF equal to or less than 40% met

none of the clinical criteria for congestive heart failure (Marantz et al, 1988).

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Granger CB, McMurray JJ, Yusuf S et al (2003) Effects of candesartan in patients with chronic heart failure and reduced left ventricular systolic function intolerant to angiotensin-converting-enzyme inhibitors: the CHARM-Alternative trial. *Lancet* **362**: 772–6

Marantz PR, Tobin JN, Wassertheil-Smoller S et al (1988) The relationship between left ventricular systolic function and congestive heart failure diagnosed by clinical criteria. *Circulation* **77**: 607–12

Sir,

I agree with Dr Jolobe. The issue of 'what is heart failure' will become increasingly important if the evidence for treatment is to be successfully applied to populations. We might envisage a more refined clinical definition with treatment with angiotensin-converting enzyme inhibitors (or angiotensin II receptor blockers) and beta blocker followed by definition of aetiology to allow prognostication and optimal further intervention.

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Cutaneous vasculitis may result from connective tissue diseases, cryoglobulin-aemia, bacterial infections and malignancy. A number of drugs including allopurinol, penicillin, tamoxifen, sulphonamides, thiazides, pyrazolone, hydantoin, propylthiouracil, streptomycin, phenothiazine, aminosalicic acids and oral contraceptive pills have been implicated. Multi-system organ involvement may occur.

In this case cutaneous vasculitis presented 1 week after starting co-amoxiclav, making it a likely cause. In all patients presenting with cutaneous vasculitis, with or without systemic complications, the possibility of a culprit drug must be borne in mind.

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Fake drugs: a problem for all

Sir,

Again fake drugs are putting patients' health at risk. This time it is counterfeit Lipitor that has been found in the legitimate supply chain.

Technology that reduces the chance of fake drugs reaching chemists is widely available. Putting unique identification on packs and pots ensures the genuine article is easily recognizable throughout the distribution network and would ensure that distributors and chemists are able to easily reject these dangerous fakes.

In such a highly regulated industry, it is surprising there is no legal requirement to have authentication equipment in every pharmacy in the UK. The industry needs to ensure best practice is in place to stamp out this illegal trade. Not only is it costing the drug companies millions of pounds but it is putting patients in potentially fatal situations.

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Cutaneous vasculitis: remember culprit drugs

Sir,

An 87-year-old woman was admitted with progressive purpuric rash affecting the distal aspects of both upper and lower limbs, including both palms (*Figure 1*), 1 week after she had been started on co-amoxiclav for an infected chronic venous ulcer affecting her right leg. Her regular medications

Figure 1. Cutaneous vasculitis rash.



included atenolol, bendroflumethazide, meloxicam and tamoxifen following radical resection of carcinoma of the right breast 5 years ago. Systemic examination was normal. Initial full blood count, renal and liver function tests were normal. Erythrocyte sedimentation rate (55 mm in 1 hour) and C-reactive protein (55 mg/litre) were raised. Blood cultures were negative and a chest X-ray was normal.

Vasculitis screen included normal results for rheumatoid factor, antinuclear antibody, anti-double-stranded DNA antibody, anti-smooth muscle antibody, antimitochondrial antibody and antineutrophilic cytoplasmic antibody assay. Cryoglobulin was not detected in the serum. A skin biopsy taken from the edge of a lesion revealed typical features of leukocytoclastic vasculitis and oral prednisolone 40 mg daily commenced. Unfortunately, during the second week following admission, she succumbed to rapidly progressive renal failure.