Digoxin is often used in the management of patients with heart failure (HF) with or without atrial fibrillation (AF). There is sound biological rationale for the use of digoxin, but the data on clinical outcomes with digoxin use in this patient population are conflicting. There is a single adequately powered randomised trial of digoxin in patients with HF in sinus rhythm. This trial showed a small but significant reduction in the rate of hospitalisation due to HF with the use of digoxin, when compared to placebo, among patients treated with ACE inhibitors and diuretics. There was no effect on mortality.

On the contrary, there are numerous secondary analyses of observational data from randomised trials which suggest that there may be an increased risk of death from using digoxin, both in patients with HF, those in AF, or both. However, observational data on digoxin use suffer from treatment bias (confounding by indication), as the sickest patients are the ones who are prescribed digoxin. Propensity matched analyses have been attempted to overcome the effect of this bias with conflicting results. However, it is likely that because the magnitude of this bias is large, no amount of statistical adjustment can yield reliable effect estimates. This highlights the need for large, randomised trials of digoxin.

REFERENCES


28 SHOULD WE BE USING DIGOXIN IN 2018?
Ganesan Karthikeyan, All India Institute of Medical Sciences, New Dehli, India
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Pulmonary arterial hypertension (PAH) is a complex and devastating disease. According to a longitudinal United States-based registry, connective tissue disease (CTD) accounted for more than 50% of all patients with PAH, in which systemic sclerosis comprised the largest CTD-related PAH. In contrast to Western countries, systemic lupus erythematosus (SLE) is a more common CTD than systemic sclerosis in the Asia-Pacific region. A cohort study from China has shown that SLE, instead of systemic sclerosis, comprised the largest proportion of all CTD-related PAH. The prevalence of PAH in SLE is estimated at 0.5%–17.5%. The pathogenesis of PAH involves multiple mechanisms including vasculitis, in situ thrombosis to intimal lung disease which may all increase pulmonary vascular resistance and lead to right heart failure. The leading risk factors for the development of PAH in SLE patients include Raynaud’s phenomenon, anti-U1 RNP antibody and anti-cardiolipin antibodies positivity. Since PAH is potentially life-threatening, early detection is crucial to improve the outcomes of this condition. Currently, the diagnostic algorithm for PAH in SLE patients follows that of international guidelines. Diagnosis is confirmed by right heart catheterisation. Treatments are similar to the therapeutic interventions for patients with idiopathic PAH. Since inflammatory and dysregulated immune components may play a major role in the pathogenesis of PAH in SLE, glucocorticoids and immunosuppressive therapies including cyclophosphamide are used, although the immunosuppressive therapy trials were small, uncontrolled studies only. Regular follow-up with prognostic evaluation and risk assessment should be performed and the treatment should be individualised accordingly.

REFERENCES


30 ADVANCES IN MEDICAL AND INTERVENTIONAL TREATMENTS FOR CTEPH
Yu Taniguchi. Department of Cardiology and Cardiovascular Intervention Unit, Kobe University Hospital, Kobe, Japan
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Pulmonary endarterectomy is the standard care for patients with chronic thromboembolic pulmonary hypertension (CTEPh), however, about 40% of them are inoperable.