Problem gambling.

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Imaging for cardiac disease: a practical guide for general practitioners

I read with interest Dr Storey’s article ‘Imaging for cardiac disease: a practical guide for general practitioners’ (AFP May 2014). In the second introductory paragraph, he states that a ‘... myocardial perfusion study (MPS...) is widely used and available for assessing whether reversible or fixed myocardial ischaemic changes are present’. Ischaemia is reversible, by definition, and there is no such diagnosis of ‘fixed’ ischaemia. On MPS, ischaemia is detected as a perfusion defect on a post-stress (exercise or pharmacological) image that reverses (fully or partially) on the rest image. A fixed perfusion defect, on the post-stress and rest images, is a transmural infarction. A partially reversible defect at rest is usually due to a non-transmural infarction with peri-infarctional ischaemia.

A non-invasive MPS is unique, compared with all other cardiac imaging modalities. The imaging agent used, 2-methoxyisobutyl isonitrile labelled with technetium-99m, (sestamibi or MIbI), for MPS is strongly cationic and lipophilic and is largely sequestered in myocyte mitochondria. MPS images the myocyte directly, hence its uniqueness. MIbI extraction by the myocardium is directly proportional to myocardial blood flow.

If there is significant coronary artery stenosis, the stress MPS depicts the lack of coronary flow reserve as a perfusion defect, decreased or absent (if severe), of myocyte uptake, which is not present at rest. Hence, MPS directly images ischaemia, the impairment of coronary artery flow reserve, due to haemodynamically significant stenotic coronary disease. No other cardiac imaging modality has this ability, making it still the best predictor of future cardiac events, compared with extensive other diagnostic studies.

Electrocardiogram-gated single photon emission computed tomography MPS provides multiple functional cardiac parameters: three-dimensional segmental perfusion (severity and extent of ischaemia and the presence or absence of myocardial infarction), left ventricular ejection fraction, ventricular volumes (end-systolic and end-diastolic), regional wall contraction and thickening (all of these at rest and post-stress) and post-stress transient ischaemic ventricular dilatation (a bad prognostic sign). No other cardiac imaging modality can provide all of these data from a single study.

Computed tomography coronary angiography (CTCA) is relatively non-invasive. However, it is anatomical and does not identify which stenoses are haemodynamically significant in causing ischaemia. MPS is the only functional study that directly images ischaemia. CTCA does not obviate the need for invasive cardiac catheterisation, which is essential for determining the suitability of lesions for revascularisation, stenting or bypass graft surgery.

The author’s diagnostic algorithm is misleading for GPs with only one pathway to MPS. There should be a direct one, in patients with risk factors or suspicious symptoms, for GPs to refer for an MPS. It must be borne in mind that GPs cannot refer for CTCA and only cardiologists do stress echography.

Andrew McLaughlin,
Burwood Nuclear Medicine, NSW

References


Problem gambling

Professor Thomas has produced an interesting article looking at problem gambling (AFP June 2014) but has missed a very important and easily treatable cause thereof, namely iatrogenically induced problem gambling.

It is now well accepted and irrefutable that dopamine agonists have the potential to evoke problem gambling, which is easily treated by suspension of the offending medication. Most recently, there has also been a suggestion that some antiepileptic medications may also provoke problem gambling, the mechanism being less clear but possibly also related to dopamine.

Professor Thomas has tactfully avoided any discussion of the possible pathophysiology of problem gambling but clearly, iatrogenically induced gambling has some connection to dopamine and further research in this area is required. Awareness of the potential for iatrogenically induced problem gambling is an imperative because it is by far the easiest mode of intervention if it is found to be present. It is

Reply

Thank you for the interest in this topic. I think there is no disagreement with the assertion that MPS and CTCA are complementary and I have been at pains to point this out.

CTCA is a relatively new imaging tool and its role is still evolving. It is unlikely that it will replace SPECT-MPS but there are newer applications of CTCA and also cardiac MRI, which assess wall perfusion, and these will be interesting technologies to follow. However, the real strength of the technology lies in its temporal advantage, in particular, the 12–20 second Triple Rule Out study, which has more of a role in the accident and emergency department.

The algorithm is of course a personal observation and discussion is always welcome.

Dr Peter Storey
Director of Imaging, Queensland X-Ray, St Vincent’s and St Andrew’s Hospitals
Toowoomba, QLD
very important that family physicians are aware of this potential so that it can be addressed if and when discovered.

Roy G Beran
Department of Medicine, University of NSW,

Reference

Reply
Thank you for the interesting comments from Dr Beran. The treatment of problem gambling using pharmacological agents is an intriguing possibility that reaches directly into the complex brain behaviour linkages.

The use of various pharmacological agents was systematically reviewed for the National Health and Medical Research Council gambling treatment guideline1 and it was concluded that, currently, there is insufficient research evidence to warrant positive recommendations for such treatments. Some research has been performed but it is very limited. Also, some case studies of the impact of using dopamine agonists for Parkinsonism (where the patients have developed gambling control problems) suggest that there are linkages.

However, at the present level of knowledge, pharmacological treatment of gambling would involve off-label use of agents for gambling treatment, drawing on agents with some demonstrated efficacy for substance and alcohol addictions. While pharmacological agents may manipulate and perhaps lower underlying enabling propensities such as impulsivity, my personal view is that problem gambling is a complex learned behaviour that has to be unlearned. So although some pharmacological agents may prove to facilitate this, their use would be as an adjunct to the primary psychological treatments.

Psychological treatments, including cognitive behaviour therapy and motivational interviewing, do have credible research evidence for their efficacy and thus should be the frontline treatments for problem gambling.

Shane Thomas
Monash University, Clayton, VIC

Reference

Flashes and floaters: a practical approach to assessment and management

We read with interest the recent article by Kahawita et al1 regarding the assessment and management of patients with flashes and floaters (AFP April 2014). This is a well-written piece providing practical guidelines for general practitioners regarding relatively common and important symptoms.

We do have some concerns, however, regarding the comments regarding the use of ultrasound. The authors claim that, especially for those in a rural setting, ultrasonography could be used ‘to determine the presence or absence of ocular pathology’. They seem to imply this can be used to substitute for an ophthalmic examination.

While ophthalmic ultrasound can be used to detect the presence of a retinal detachment, it is often difficult to detect peripheral retinal tears, even in experienced hands. It has been reported that the prevalence of retinal tear is up to 14% of patients with acute onset of flashes and floaters.2 Without prompt and adequate treatment, retinal tears can lead to rhegmatogenous retinal detachment and vision loss. The studies quoted did not assess the sensitivity of ultrasound for detecting retinal tears.

A small study from the United Kingdom showed that even an experienced retinal surgeon would miss more than one in 10 tears with 90D biomicroscopy alone if indented ophthalmoscopy were not done.3 A review of medico-legal litigation cases in the National Health Service showed that the majority of vitreoretinal negligence cases result from a delayed or missed diagnosis of retinal detachments and tears.4

We recommend all patients with acute onset of flashes and floaters, especially those with high-risk characteristics, be promptly referred for a dilated retinal examination to reduce the risk of vision loss.

Dr Neil Sharma
Dr Ju-Lee Ooi
Mr Jong Min Ong
Cambridge University Hospitals NHS Trust, UK

References

Reply
We would like to thank Dr Sharma and colleagues for bringing up the issue of ultrasonography of the eye for retinal detachment in a rural setting. We agree that ultrasound is not a substitute for ophthalmic examination. Country general practitioners who are not comfortable with examining the eye may have easier access to a local optometrist, who may be able to help diagnose retinal detachment and facilitate referral. However, in a rural setting, where the nearest ophthalmologist may be several hours away, ultrasonography may be helpful in distinguishing retinal detachment from the other differential diagnoses mentioned in our article.1 Ultrasonography seems to be readily accessible in rural areas and it may aid diagnosis for those practitioners who are experienced in ultrasonography.

Indeed, ophthalmologists themselves use ultrasonography when the retina cannot be viewed (eg vitreous haemorrhage) to aid in assessment of masses or retinal detachment; however, this is not a replacement for an ophthalmic exam.

Shyalle Kahawita
The Royal Adelaide Hospital, SA

Reference

Stroke
In the article ‘Stroke’ (AFP March 2014), Pollack et al1 provided some valuable data on encounters of cerebrovascular disease in general practice in Australia. Although the majority of strokes are managed acutely in hospital, general practitioners (GPs) need to be confident in assessing initial...