LETTERS

Trimethylaminuria (fish malodour syndrome): a "benign" genetic condition with major psychosocial sequelae

Helen Mountain, Joanna M Brisbane, Amanda J Hooper, John R Burnett and Jack Goldblatt

To the Editor: We report the case of a 41-year-old woman who sought medical opinion about an unpleasant body odour, first noticed when she was 7 years old. After experiencing ridicule, distress, shame, anxiety and low self-esteem during her school years, she first consulted a doctor about the problem at the age of 17 years, then again 2 years later, followed by a further four doctors over the next 20 years. All dismissed her concerns, and she was repeatedly told that she had a hygiene neurosis. Investigations and treatments during this time included being "sniffed", vaginal swabs and vaginal cauterisation. Finally, a general practitioner referred her to a dermatologist, who consulted a microbiologist, and the diagnosis of trimethylaminuria (TMAU), or fish malodour syndrome, was confirmed by urinalysis. Now having a name for her condition, she found an Internet-based support foundation and referred herself for genetic counselling.

TMAU is caused by an enzyme deficiency due to mutations in the flavin-containing mono-oxygenase 3 (*FMO3*) gene,[1] resulting in excess excretion of trimethylamine in urine, sweat and breath. It is diagnosed by clinical symptoms and urine analysis.[2] The characteristic body odour resembling rotting fish can be intermittent, variable and influenced by diet, hormones and medications. Restriction of choline- and carnitine-rich dietary precursors (eg, fish, eggs, soybeans, peas) is difficult to maintain and effective in only 25% of patients.[2] Acid soaps and body lotions can often reduce the odour.[3] The metabolic and clinical manifestations of TMAU are generally regarded as benign, as there is no associated organ dysfunction. This designation, and the fact that the condition is often unrecognised by doctors, can have important ramifications including missed or delayed diagnosis.[4]

Affected individuals experience shame and embarrassment, fail to maintain relationships, avoid contact with people who comment on their condition, and are obsessive about masking the odour with hygiene products and even smoking. The malodorous aspect can have serious and destructive effects on schooling, personal life, career and relationships, resulting in social isolation, low self-esteem, depression, paranoid behaviour, and suicide.[4,5] Psychosocial problems resulting from delayed diagnosis, body odour and the lack of cure are considerable, making this a far from "benign" disorder.

Recognition of TMAU as a significant clinical entity and increased understanding of the issues patients face are needed.[5] Awareness of the typical patient history would facilitate prompt metabolic diagnosis and pre-empt some of the associated psychosocial sequelae. Referral of patients for genetic counselling enables short-term psychosocial support and family cascade genetic testing. Consultation with a metabolic clinic for dietary management may also be beneficial.

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Unplanned admissions to two Sydney public hospitals after naltrexone implants

D Martyn Lloyd-Jones

To the Editor: In their recent case series, Lintzeris and colleagues state that the symptoms leading to hospital presentation were "associated" with naltrexone implants.[1]

In half of the 12 cases, the symptoms were related to the induction of withdrawal rather than the presence of naltrexone — an important distinction that may not be apparent to all, but of which the authors would be aware. Three of the remaining cases reflect the complexities of pain management in patients being treated with naltrexone, which are not restricted to those with implants. This dilemma is also encountered in patients taking buprenorphine. Patient 8 had an anxiety disorder, and his symptoms probably related to an absence of opiate; and Patient 9 had symptoms that were probably due to cocaine use.

Abstinence is a patient's choice, and naltrexone can be effective in minimising the risks associated with the consequent lowered tolerance to opioids.[2]

I am concerned that undue emphasis appears to have been placed on the association of adverse outcomes with implants, with such statements as: "This case series identifies severe adverse events associated with the use of naltrexone implants". In fact, the causal condition is usually the induction of opiate withdrawal rather than the presence of a naltrexone implant.[3]

The statement that "Most of these cases (8/12) can be attributed to the naltrexone implant or implantation procedure" is true, but it would be more appropriate to acknowledge that the procedure appeared to be the cause in most cases and that only in one case (Patient 7) could the implant be conclusively implicated.

It is not appropriate to refer to difficulties in pain management as "severe adverse events", as the blockade of the μ opioid receptor is the reason naltrexone is used. In addition, it is important to note that a trial of injectable naltrexone has shown promising results.[4]

While these facts are acknowledged by the authors, their emphasis on the *association* of adverse events with the presence of a naltrexone implant, rather than demonstrating causality, is concerning. Similarly, this approach appeared in Gibson and colleagues' earlier commentary on overdose deaths in patients with naltrexone implants.[5]

Nonetheless, I commend the authors for raising the issues relating to the need to examine the role of naltrexone implants in treating opioid dependence and exploring alternatives to the use of rapid detoxification. I also endorse the need for thorough assessments, treatment planning and evaluation in patients undergoing such therapies.

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Mark Little and Lindsay M Murray

To the Editor: We read with interest the report of 12 hospital presentations related to the use of naltrexone implants.[1] The accompanying editorial highlights how the rigorous scrutiny required to evaluate the efficacy and safety of this procedure is lacking.[2] Regrettably, this study is likely to distort rather than inform the debate.

Lintzeris and colleagues[1] only identified patients with naltrexone implants who were referred to the Drug and Alcohol Consultation–Liaison services, not all patients presenting to the study hospitals. Additionally, the authors did not follow the methodology of chart reviews, as recommended by Gilbert and colleagues.[3] Four of the 12 patients clearly had problems unrelated to their naltrexone implants. There was no attempt to identify the number of naltrexone implantations performed (ie, the denominator), nor was there any attempt to compare the naltrexone group with others being treated with agents such as methadone or buprenorphine.

In 2003, we published our experience of naltrexone-accelerated detoxification in the emergency department (ED) of Sir Charles Gairdner Hospital.[4] The hospital's clinical toxicology service is based in the ED, and the hospital is located 2 km from the only private clinic in Perth that was using naltrexone during the study period in 2001. Working collaboratively with this clinic, patients developing complications from detoxification were referred to our service. In 6 months, 42 patients (7% of all those receiving naltrexone treatment) presented to the ED — 17 within 24 hours of treatment and 31 within 48 hours. Gastrointestinal symptoms of withdrawal were present in 18 patients, and central nervous system symptoms of withdrawal (predominantly agitation) in 14. Two patients required intubation for airway compromise secondary to a combination of agitation and chemical sedation. In 23 patients receiving naltrexone implants (rather than oral therapy), three developed infections and three complained of local pain. The mean length of stay for all patients was 18 hours (compared with 2.3 days in Lintzeris et al's study), with the longest stay being 92 hours for one of the patients admitted to the intensive care unit. During the study period and the subsequent 6 months, four deaths of individuals who had undergone naltrexone-accelerated detoxification were reported to the Coroner, all being classified as probable drug overdose, probably opioid. None of these patients had presented to the hospital's ED during the study period.

It is important that good data are made available to inform the debate on naltrexone implants in the management of opioid dependence. Better communication and collaboration between clinics using naltrexone, EDs, and alcohol and drug services will improve the care of these patients.

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Michael P W Kozminsky

To the Editor: The study by Lintzeris and colleagues[1] and the associated editorial[2] criticise naltrexone implants. However, the study is replete with errors regarding the 12 cases reported.

A key to successful rapid opioid detoxification (ROD) is octreotide. An agonist of non-μ gut receptors, it prevents gastrointestinal symptoms of withdrawal.[3,4] Earlier aggressive dosing is more effective. Only two of the six patients reported as having precipitate opiate withdrawal received octreotide, administered in both cases by their local doctor. Presumably this doctor performed the ROD. Why was the octreotide administration not repeated?

Patient 7 was reported as having a localised abscess at the implant site. These may arise a variable period after implant insertion. Magnesium-monostearate, used as a binder in naltrexone and many other implants, may liquefy, resembling pus. Results of repeated microbiological examination are always negative. An actual infection at the implant site is rare.

Patient 8 demonstrates that many patients use opiates and other drugs as self-medication for their psychiatric symptoms. It is regrettable that this patient's psychiatrist was not involved in the decision making. Was the doctor doing the ROD aware that there was a psychiatrist involved? Transient psychosis may occur following ROD; use of antipsychotics is appropriate.

Arrhythmias (as diagnosed in Patient 9) are a textbook in themselves. Pre-ROD preparation requires assessment for comorbidities including bacterial endocarditis and arrhythmia syndromes. Substance-misusing patients often use arrhythmogenic agents such as amphetamine. No mention was made of precipitate withdrawal symptoms in this patient, so an electrolyte abnormality is unlikely.

Patients 10–12 had problems that were unrelated to the naltrexone implants and poorly managed by the receiving hospital. Naltrexone has 2 log the affinity for opiate receptors to that of morphine and most other opiates.[5] This means that 1 mg of naltrexone will block 1 g of another opiate. Using opiates to treat patients in these circumstances therefore makes no sense. Ketamine, local anaesthetic blocks, paracetamol and non-steroidal anti-inflammatory drugs are alternative options for treatment.

To compare naltrexone implants with thalidomide[2] is emotive; all the components of the implants are approved agents. Similarly, to say they have not been fully tested is to condemn all products supplied by compounding pharmacists.

Testing of serum naltrexone levels (to assess whether an implant is still working) is not rebatable under Medicare, so is expensive to perform. Research is a necessary component of naltrexone implants, and I would happily cooperate with any of the study authors in coordinating and performing such research.

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Colin L Brewer

To the Editor: A recent article by Lintzeris and colleagues,[1] ostensibly about naltrexone implants, actually has little to do with implant treatment. Only one of the 12 reported cases involved problems linked specifically to naltrexone (antagonist) treatment being administered in implanted rather than oral form. This case involved infection at the implant site — undesirable, certainly, but about as noteworthy as the occasional infections that occur after abdominal surgery or breast implantation, despite antibiotic prophylaxis and careful technique. The remaining 11 admissions reflected not implant use but either the procedure of rapid antagonist induction (RAI) or the desired pharmacological effects for which naltrexone was prescribed in the first place (and one admission for unrelated pneumonia).

RAI is needed because conventional antagonist induction requires complete opiate withdrawal before starting naltrexone. As true conventional withdrawal completion rates, even with inpatients, are typically below 30%,[2] conventional techniques will typically achieve only derisory naltrexone induction rates. Various forms of RAI or accelerated induction, with induction rates typically around 100%, are more effective and more cost-effective.[3,4] Similarly, complaining that pain management is difficult in naltrexone patients is like complaining that patients being treated with anticoagulants bleed or bruise more after surgery or injury. Fortunately, effective non-opiates (notably subanaesthetic ketamine) exist.

The acute post-RAI problems seen in this case series would have been identical had every patient undergone RAI to oral rather than implanted naltrexone. Indeed, while 150 mg orally could block opiate analgesia for up to 72 hours, implants produce low but consistently effective naltrexone levels, disappearing within hours of implant removal, should that be unavoidable. I agree, however, that RAI clinics should optimise immediate post-RAI management.

A recent conference featured the first presentations of the first four randomised controlled trials of implanted naltrexone. Three showed statistically and (more importantly) clinically significant advantages over "as usual" post-withdrawal treatment,[5] or over oral naltrexone and placebo implants.[6,7] The fourth,[8] comparing implants with methadone maintenance in pre-release prisoners, had only modest statistical power ($n = 21$), but implant patients had less dropout and used about 50% less heroin. We already know that long-acting implants can largely prevent the opioid overdoses that are otherwise an intrinsic and sometimes lethal hazard of all abstinence-based programs;[9] and that all implants prevent the otherwise high relapse rates typical of the first 4–6 weeks after detoxification.[10]

Surgeons, I am shocked to discover, have been using anaesthesia for 162 years without a comparable placebo-controlled evidence base.

Competing interests: I receive some income from a private clinic that provides, inter alia, both agonist and antagonist treatment for opiate addicts.

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Nicholas Lintzeris

In reply: Our case series[1] aimed to alert health practitioners to the types of hospital presentations associated with naltrexone implants and issues in managing such patients, particularly as these have not been well documented in the medical literature. These letters appear critical of any such communication.

We maintain that our article[1] identifies important clinical issues — such as the difficulties of providing analgesia in patients with implants of uncertain duration of effect (whereby the treating hospital staff have no way of knowing whether the implant is providing "active" plasma levels of naltrexone, given the lack of a licensed product). Ultimately, our study could not hope to provide the type of data that can only be accurately determined by independent clinical trials — such as the incidence rate of serious adverse events (as suggested by Little and Murray) or how these are most effectively managed (eg, the role of octreotide compared with ondansetron for managing protracted vomiting, as suggested by Kozminsky).

A key complaint from the correspondents is whether the serious adverse events of opiate withdrawal and dehydration were caused by the naltrexone implant or by the rapid opioid detoxification process that accompanied the implant's insertion. We acknowledged the difficulty of attributing causality in our article, but, unlike the correspondents, we cannot dismiss the possibility that the naltrexone implant may have contributed to the severity or duration of the opiate withdrawal syndrome. Clearly,

there is a pharmacological basis as to how the presence of naltrexone from an implant may contribute to opiate withdrawal in a recently detoxified individual. Research comparing rapid opioid detoxification plus a naltrexone implant with conventional detoxification plus a naltrexone implant is required, to allow an assessment of whether naltrexone implants themselves contribute to the incidence, severity or duration of any opiate withdrawal. Until such data exist, it is appropriate to associate opiate withdrawal with naltrexone from the implants.

We concur with several of the correspondents that further research is required to address many of the issues raised by our study, as well as better communication between service providers, better procedures for management of complications, and better assessment and patient selection. We maintain that naltrexone implants should not be routinely used until research has demonstrated their safety and efficacy, and, importantly, until there is a licensed naltrexone implant product with appropriate regulatory safeguards for patients and providers.

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Medication self-administration by patients: a way to prevent errors?

Frank T Formby

To the Editor: Two studies[1,2] and an editorial[3] on the vexed issue of medication errors in hospitals have recently appeared in the Journal. Yet none of them have mentioned patients, except as the passive victims of error, and all three have focused exclusively on public hospitals. This is unfortunate as, arguably, the public hospital system is evolving predominantly into a way station along the chronic illness journey within the wider health care system.

To quote an 86-year-old patient of mine: "I've been taking the same tablets for 30 years, but as soon as I come to hospital they take them away and give me other ones, as if I'm incapable." Indeed.

Could patients continue taking their own regular medications (patients' own drugs [PODs]) themselves? This would eliminate many errors of transcription and administration and eradicate in-hospital dispensing errors altogether. Although it might seem risky for patients to be given free rein with their medications, this is precisely what they are doing at home, and generally with no supervision whatsoever. Furthermore, if patients do make errors, they are more likely to omit some medications than to take toxic doses, and the consequences of such errors of omission are likely to be minor. If a self-administration system for PODs were to be implemented — including newly prescribed drugs that patients were expected to continue using on discharge, as well as medication strategies used in the home (eg, dosette boxes) — then any errors could be noted and corrected, just as a patient's mobility is assessed before discharge.

On the other hand, it is pertinent to note that many hospital admissions arise from medication errors, with one study demonstrating that up to 30% of admissions in patients aged 75 years and over are medication-related and up to 75% are preventable.[4] Clearly, certain groups of patients and certain drugs should be excluded from a POD system. A sample protocol is available from the author on request.

Would a POD self-administration system be cost-effective? Such a system has been used successfully in at least one Australian private hospital (in which I have worked) and in several hospitals in the United Kingdom.[5] A systematic review has also been undertaken to examine the risks and benefits of the use of PODs, but it did not discuss the issue of self-administration.[6] A MEDLINE search from 1996 onwards for patients' own drugs/medications and self-administration yielded no results.

As always, further research is required. What my patient's comment highlights is that patients do have autonomy and some competence, and that these factors should be taken into account in any strategies aimed at reducing medication errors.

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Physician on call: Sweden compared with Australia

Henrik Falhammar

To the Editor: In May 2007, I began working in an Australian public hospital as a permanent consultant endocrinologist and physician. There are many differences between the health systems of Australia and Sweden, where I previously worked. In particular, there are striking differences in on-call work for the rostered consultant.

In Sweden, the compensation (usually taken as leave) for every phone call, whether it was 1 minute or 30 minutes long, was half an hour during weekday hours of 4.30–9 pm and 7–8 am, and 1 hour during nights and weekends. Compensation also applied to ward rounds on weekends. I also received an on-call allowance of 6 minutes per hour during weekdays and 12 minutes per hour during weekends. During public holidays, rates were tripled. Every year, I earned 5–7 extra weeks on top of my normal 5 weeks of annual leave, some of which was used for well paid locum work, as private practice is almost non-existent in Sweden. Compensation could also be taken as payment; each department had its own agreement with staff on the preferred format, depending on staff levels and budget.

The on-call frequency was usually 1 in 10, and the physician on call was responsible for all general internal medicine patients. For most of my career I worked in a large tertiary referral centre,[1] but the rostering and compensation were similar in local general hospitals, with the only difference being fewer subspecialties with their own on-call roster. The surgical specialties had the same system.

As in Australia, I was rarely called to attend the hospital, although contact during an on-call session was more frequent in Sweden (2–10 v 0–2 phone calls). Junior doctors in Sweden were more inclined to

discuss patients and, in some wards, the nurses were instructed to call the physician on call first if the matter could be solved by phone. I was happy for every call, as I received extra compensation, but I never received any further phone calls once the on-call session finished.

In Sweden, I did ward rounds on weekends, either of all medical wards in the local general hospitals or only the general medical wards in the tertiary centre when I was physician on call. Before seeing patients, I did a paper round with the nurses, when we decided which patients I needed to see. Patients were not admitted directly under me, but under the specialists responsible for the wards during office hours. However, I was temporarily responsible for all medical patients in the hospital.

In Australia, at least in my current hospital, I have "my patients" in the wards (usually spread out over the entire hospital). If something suddenly happens to one of them, the intern, resident medical officer or medical registrar will phone me, even in the middle of the night or if I am away on leave. As the compensation is the same whether phone calls are received or not, and most medical patients are admitted under the physician on call, there is little incentive for the physician to volunteer for extra on-call sessions or to encourage junior doctors to phone.

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