Hyperprolactinaemia and Antipsychotic Drug Use:
The Benefits of Prolactin Screening
for Mental Health Patients

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Hyperprolactinaemia and Antipsychotic Drug Use: The Benefits of Prolactin Screening for Mental Health Patients

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Abstract:

Hyperprolactinaemia is associated with drugs used in the field of Mental Health to treat psychosis (antipsychotic).

This has been found to be affecting a significant number of service users taking this medication. Hyperprolactinaemia causes endocrine disturbances including sexual dysfunction and amenorrhea and infertility. However, in the longer term service users are at risk of some disabling conditions such as osteoporosis.

As part of our ongoing medication management work, early detection of hyperprolactinaemia enables safer use of antipsychotic drugs.

Keywords: Prolactin elevation, Psychosis, Antipsychotic Drugs, Medication Management.

Introduction

Antipsychotic drug use has for decades been associated with impaired sexual functioning. (Shader and Grinspoon, 1967). A recent survey showed that at least one symptom of sexual dysfunction was reported by 82% of men and 96% of women who had a diagnosis of schizophrenia (MacDonald, 2003).

It is my experience as a Community Mental Health Nurse working in a specialist Community Mental Health Team for Medication Management that sexual dysfunction is extensive for individuals prescribed antipsychotic drugs.

When I was appointed to the post of Team Leader (Community Treatment Team) in 1998 we began systematically reviewing side effect profiles of all individuals prescribed these drugs and although I was surprised by the number of service users who reported sexual dysfunction, I was even more surprised by the amount of people who, until this time, had not reported the problems they were having and the subsequent level of distress they were experiencing. For many, embarrassment was a key factor in "keeping quiet". Once this initial embarrassment was sensitively addressed, people often stated that they felt relieved that they now had an arena to talk about this problem and that the distress they were experiencing was taken seriously.

For individuals who already had a psychiatric diagnosis that erodes their self esteem and level of self confidence, the treatment they were receiving in many cases further contributed to the reduction of their self worth, which will inhibit recovery from psychosis and rehabilitation into the wider community. Having realised the extent of the problem within my locality, I
began to examine the research published within this area and realised that antipsychotic drugs induced sexual dysfunction is directly linked to elevated prolactin levels.

A definite cause and effect scenario was reported in the literature. I was also shocked to discover that hyperprolactinaemia has a causal link to osteoporosis (Meaney and O'Keene, 2003). Not only were individuals experiencing the shorter term misery of sexual dysfunction, but were at risk of a longer term disabling condition.

**The Role of Prolactin**

Prolactin is a glycoprotein, similar in structure to growth hormone. It is a milk producing hormone produced in the anterior lobe of the pituitary body. Its role is to stimulate the mammary gland preparing the mother’s body for lactation following childbirth.

During pregnancy, although prolactin levels rise, high levels of oestrogen and progesterone prevent lactation. After childbirth the levels of oestrogen and progesterone decrease triggering lactation. Through breast feeding raised prolactin levels causes annovulation and amenorrhoea. Prolactin influences gonadal function in both sexes, however normal prolactin levels are slightly higher in woman than men.

**Hyperprolactinaemia**

The inhibitory control of the neurotransmitter dopamine is the most important mechanism regarding prolactin secretion. Dopamine is secreted from the hypothalamus into the pituitary venous system where it binds to dopamine D2 receptors on prolactin secreting cells, thus inhibiting the secretion of prolactin.

Persistent hyperprolactinaemia is usually due to a micro-adenoma of the anterior pituitary. Dopamine receptor agonists including antipsychotic drugs can also be responsible (Walker and Renwick, 1994).

The therapeutic action of Dopamine 2 receptor blockade is to reduce the positive symptoms of psychosis such as perceptual and thought disorders including hallucinations and delusions. It has long been known that blockading this receptor with antipsychotic drugs, prescribed to treat these symptoms, will often cause extra pyramidal side effects (EPSE) such as parkinsonism, akathisia (motor restlessness), dystonias and tardive dyskinesia. Indeed the emphasis on pharmacological research and subsequent marketing in recent years with the introduction of atypical (second generation) antipsychotics has been to eradicate EPSE in mental health. However, blockade at this receptor is also responsible for elevated prolactin levels causing endocrine problems including sexual dysfunction.

Although this mechanism has been recognised for more than a quarter of a century (Beaumont, 1974) until recently this has attracted little clinical or scientific interest, despite the fact that it is common, distressing and has the potential for long term complications.
Service users, when asked, consider the neuro endocrine effects of antipsychotic drugs to be among the most undesirable of adverse effects, leading to reduced levels of compliance with the medication they are prescribed to treat their psychosis.

Table 1 below lists the physical effects associated with hyperprolactinaemia:

**TABLE 1: The clinical effects of hyperprolactaemia**

<table>
<thead>
<tr>
<th>EFFECT</th>
<th>MALES</th>
<th>FEMALES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annovulation</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Infertility</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Amenorrhoea</td>
<td></td>
<td>*</td>
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<tr>
<td>Decreased Libido</td>
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<tr>
<td>Gynaecomastia (breast enlargement)</td>
<td>*</td>
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<tr>
<td>Galactorrhoea (lactation)</td>
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<td>*</td>
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<tr>
<td>Erectile dysfunction</td>
<td></td>
<td>*</td>
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<tr>
<td>Ejaculatory dysfunction</td>
<td></td>
<td></td>
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<tr>
<td>Azoosperma</td>
<td></td>
<td></td>
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<tr>
<td>Weight gain</td>
<td></td>
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</tbody>
</table>

Research has consistently found that a great deal of service users with psychosis (including schizophrenia) were found to have sexual dysfunction. 54% of male schizophrenics taking antipsychotics were experiencing moderate to severe sexual dysfunction and 91% females reported menstrual changes (Ghadirian, Chouinard and Annable, 1982).

As mentioned, a great cause of concern is the link between hyperprolactinaemia and osteoporosis. Chronic low blood levels of sex hormones results in increased reabsorption of calcium from bones, reduced absorption of calcium from the gastrointestinal tract and increased loss of calcium from the kidneys. This results in an overall reduction of calcium content in bones resulting in oesteopinia or osteoporosis (Meaney and O'Keene, 2003). The Royal College of Physicians recommend women with hyperprolactinaemia and a 12 month history of amenorrhoea should have bone mineral density measurements. Further, hyperprolactinaemia may increase the risk of breast cancer. It has been suggested that women taking antipsychotic agents should undergo regular breast screening.

In generalised terms, individuals suffering from psychotic disorders such as schizophrenia, have several risk factors that can predispose to osteoporosis. They include reduced vitamin D, cigarette smoking, in some cases excess alcohol and decreased level of exercise (particularly weight bearing exercise). With the possibility of antipsychotic drug induced hyperprolactinaemia in consequence their bone health is at risk of impairment.
Recognising and Managing Antipsychotic Drug Induced Hyperprolactinaemia

The team I work with (Community Treatment Team) was commissioned in 1998 to work closely with service users in terms of medication management. Currently we are five Registered Mental Nurses (RMNs) with a specialist interest in psychotropic treatment strategies. We run several community clinics and provide a domiciliary service for treatment and monitoring. This includes depot, clozapine and mood stabiliser clinics (Shaw, 2004). A large part of our work is side effect monitoring and management. The aim of this is to reduce adverse effects and to enhance service user outcomes by improving levels of concordance.

Using validated systematic rating scales such as the Liverpool University Neuroceptic Side Effect Rating Scale (LUNSERS) (Day, 1995), we are able to work closely with the service user to identify general areas of concern. In order to gain more detailed information about the nature of unwanted effects experienced, the use of more specific tools is essential, such as the Abnormal Involuntary Movements Scale, (AIMS) the Barnes Akathisia Scale, the Antipsychotic Non-Neurological Side Effect Rating Scale (ANNERS), the Arizona Sexual Experiences Scale, and so on. However, I believe any rating scale (generalised or symptom specific) should be used in combination with physical monitoring. For example, a person may not be aware that he or she has developed drug induced neutropenia that can lead to aggranulocytosis and thus be potentially fatal. A person may report weight gain, but without an accurate, over time, record of weight and body mass index the full extent of the problem cannot be assessed.

Table 2 lists some of the side effect management strategies used by the Community Treatment Team:

**TABLE 2: Interventions in adverse effect monitoring**

<p>| | | | | | |</p>
<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Side Effect Rating Scale (Generalised) Lunsers, Sescam, Annsers, UKU</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Side Effect Rating Scale (Specific) AIMS, DISCUSS, Barnes Akathisia, Arizona sexual experiences etc.</td>
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</tr>
<tr>
<td>3.</td>
<td>Blood Samples: Electrolytes and urea, thyroid function, full blood count with differential, liver function, bone profile, random or fasting glucose, prolactin level, fasting lipid profile, drug plasma level (e.g. for Clozapine).</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5.</td>
<td>ECG (prescriber referral)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>EEG (prescriber referral)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Relative/carer’s concerns/observations (with service user’s permission)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>8.</td>
<td>General observations (e.g. pallor, movement, speech, co-ordination, etc.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Written/verbal communication to Consultant Psychiatrist, General Practitioner and Care Co-ordinator.</td>
<td></td>
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</tbody>
</table>
In terms of medication management, side effect monitoring is extremely useful, but needs to be used in conjunction with treatment education, current evidence for level of concordance and the service users’ beliefs and wishes relating to their treatment. All this is undertaken at regular intervals with the service users’ prescribed antipsychotics and is reported to the prescriber (usually the Consultant Psychiatrist), the G.P. and the Care Programme Approach (CPA) Care Co-ordinator.

The prescriber then has access to detailed and current information about the service users antipsychotic drug use and is able to make more evidence based prescribing decisions. The service user, having undergone this process, is more able to make an informed choice about treatment, validating their level of informed consent to treatment. The nurse who is often administering these treatments is now able to continue his or her work with the confidence that a thorough medication review has been undertaken, reported and hopefully addressed.

For the purpose of this article I would like to focus on the neuroendocrine adverse effects and how this problem can be addressed. Sexual dysfunction, which is commonly associated with drug induced hyperprolactinaemia, can be recognised at three levels:

The service user may report symptoms such as amenorrhea, erectile dysfunction or reduced libido. Health care professionals may undertake specific rating scales to identify problems, or physical tests (i.e. prolactin level) may be taken to identify hyperprolactinaemia. Only the latter intervention will diagnose hyperprolactinaemia with certainty. Prolactin levels are obtained by venapuncture, ideally fasting or at least one hour after food.

In Calderdale the range for males is 0 – 500 mIU/L and females 0 – 700 mIU/L (Milli international units per litre).

In Calderdale the Community Treatment Team has undertaken prolactin level measurement for 194 patients between 2003 and 2005 (89 females, 105 males). Levels were all taken in the morning. If antipsychotic treatment was altered, another baseline prolactin sample was obtained. Informed consent was obtained following explanation of reason for sample collection, all service users gave consent. 38% of service users were found to have abnormal prolactin levels, 21% had levels above 1000 mIU/L indicating significant hyperprolactinaemia.

Hyperprolactinaemia was significantly more prevalent in females than males (females 52%, males 26%).

Significant hyperprolactinaemia (> 1000 mIU/L) was more prevalent in females than males (females 38%, males 7%).

Of the 52% of females with abnormal values, 74% had values > 1000 mIU/L.

Although prolactin levels were above 1000 mIU/L in only 7% of males, current evidence suggests that any high value (> 500 mIU/L) might contribute to sexual dysfunction.
These findings show that generally females taking antipsychotic agents are more likely to develop hyperprolactinaemia than males, and further they are more likely to show hyperprolactinaemia at significant levels than males.

Please see Table 3 for Hyperprolactinaemia and Gender results

**TABLE 3: Hyperprolactinaemia and Gender results**

<table>
<thead>
<tr>
<th>Prolactin (mIU/L)</th>
<th>FEMALES N = 89</th>
<th>MALES N = 105</th>
<th>TOTAL N = 194</th>
</tr>
</thead>
<tbody>
<tr>
<td>ULN &lt; 500 (Male)</td>
<td>43 %</td>
<td>78 %</td>
<td>121 %</td>
</tr>
<tr>
<td>&lt; 700 (Female)</td>
<td>48 %</td>
<td>74 %</td>
<td>62 %</td>
</tr>
<tr>
<td>500/700 – 1000</td>
<td>12 %</td>
<td>20 %</td>
<td>32 %</td>
</tr>
<tr>
<td>1001 – 2000</td>
<td>22 %</td>
<td>5 %</td>
<td>27 %</td>
</tr>
<tr>
<td>&gt; 2000</td>
<td>12 %</td>
<td>2 %</td>
<td>14 %</td>
</tr>
</tbody>
</table>

For males level 0 – 500 mIU/L or females 0 – 700 mIU/L were deemed to be within normal range. For the purpose of this naturalistic study it was decided that levels of 500 – 1000 mIU/L (males) / 700 – 1000 mIU/L (females) were deemed to be borderline high. Levels 1001 – 2000 mIU/L were deemed to be significantly high: that may need drug or dose alterations. Levels above 2000 mIU/L were deemed to be extremely high: may require referral to endocrinologist.

In terms of antipsychotic drugs and prolactin levels the following results were obtained:

1. **Risperidone (atypical antipsychotic)** – As monotherapy 69% of all service users were found to have elevated prolactin levels, 51% had levels above 1000 mIU/L. In terms of Risperidone Consta (injectable preparation of Risperidone) 53% were found to be hyperprolactinaemia, females had particularly high levels of prolactin and 100% of female service users had levels above 1000 mIU/L, 4 out of 6 female service users had levels above 2000 mIU/L.

2. **Amisulpride (atypical antipsychotic)** – 100% of service users of Amisulpride as monotherapy had elevated prolactin levels. However numbers were small (seven) for this cohort.

3. **Clozapine (atypical antipsychotic)** – Only one service user (out of 21) had elevated prolactin levels.

4. **Olanzapine (atypical antipsychotic)** – 6% of service users on monotherapy had elevated prolactin levels.

5. **Typical, or first generation atypicals**, 36% had elevated prolactin levels.

From this cohort of individuals prescribed antipsychotic drugs in Calderdale, many were experiencing hyperprolactinaemia. We found that two of the second generation drugs, Risperidone and Amisulphride, were associated with high prevalence of hyperprolactinaemia than with the
typical (or first generation drugs). Please refer to Table 4: Hyperprolactinaemia by Agent

In monotherapy one individual taking Clozaril and one taking Olanzapine were found to have had only borderline hyperprolactinaemia and for both individuals repeat prolactin levels (without any drug alterations) taken some weeks later, both showed normal levels, thus indicating that for these two individuals hyperprolactinaemia was transient and in both cases asymptomatic.

**TABLE 4: Results: Hyperprolactinaemia by Agent**

<table>
<thead>
<tr>
<th>HP</th>
<th>AMI</th>
<th>TYP</th>
<th>OLANZ</th>
<th>CONST</th>
<th>RISPoral</th>
<th>CLOZ</th>
<th>ARI/QU</th>
</tr>
</thead>
<tbody>
<tr>
<td>MONO</td>
<td>7 N</td>
<td>7 N</td>
<td>27 N</td>
<td>83 N</td>
<td>1 N</td>
<td>16 N</td>
<td>9 N</td>
</tr>
<tr>
<td>COMBO</td>
<td>1 N</td>
<td>2 N</td>
<td>13 N</td>
<td>29 N</td>
<td>6 N</td>
<td>19 N</td>
<td>0 N</td>
</tr>
<tr>
<td>% in HP</td>
<td>89</td>
<td>36</td>
<td>20</td>
<td>53</td>
<td>79</td>
<td>13</td>
<td>0</td>
</tr>
</tbody>
</table>

HP = Hyperprolactinaemia; N = Normal Level; AMI = Amisulpiride; TYP = Typical (first generation Antipsychotics; OLANZ = Olanzapine; CONSTA – Risperidone Consta (IMI Preparation); RISPoral – Risperidone Oral; CLOZ = Clozapine; ARI/QU = Aripiprazole and Quetiapine; MONO = Monotherapy; COMBO = Combination Therapy (polypharmacy).

Attribution of hyperprolactinaemia to specific medications is complicated by use of antipsychotic polypharmacy in 35/194 (18%) of service users.

The wide spread use of second generation antipsychotics within psychiatry world wide was partly to reduce the prevalence of the movement disorder side effect known as Extra Pyramidal Side Effects (National Institute of Clinical Excellence, 2002). However in reality, for people taking these drugs, one set of distressing and disabling adverse events have been replaced with another: sexual dysfunction and endocrine disorder.

In our clinical practice, when hyperprolactinaemia is identified we have found in general terms that addressing this issue tends not to be too problematic for the service user, the prescriber or the mental health team. In most cases a simple dose adjustment or switch to another antipsychotic was found to effectively return prolactin levels to within normal parameters. The following two case studies illustrate these points. The two case studies have been anonymised in terms of any demographic information or information that may compromise confidentiality, such as personal details.

**Case Study (1)**

Helen has a diagnosis a diagnosis of Bipolar II Disorder.

She attends the Psychiatric Outpatient Clinic three monthly for Consultant Psychiatrist review and monitoring with the Community Treatment Team. She does not have Sector Community Mental Health Team input as she has been in remission for several years. At the time of her initial prolactin
screen she was prescribed Carbamazepine 200mg b.d. and Risperidone 4mg per day.

In May 2003 she was seen in the Outpatient Clinic and reported amenorrhea and reduced libido that had gradually worsened over several months. A prolactin level, thyroid function and fasting blood glucose was performed in addition to her routine Carbamazepine monitoring. Thyroid function and glucose levels were within normal range ruling out sexual dysfunction relating to thyroid changes or diabetes. Prolactin level was 1036 mIU/L. The range for females is 0 – 700 mIU/L. In response Risperidone was stopped and Olanzapine 10mg was introduced. She was seen again approximately 6 weeks later. Since the medication changes she presented with no deterioration in terms of mental health. She had started menstruating regularly quite quickly after the Risperidone was stopped and she was beginning to experience an increase in libido. A prolactin level taken at this time was 113 mIU/L (within normal range).

Gradually over time this lady’s libido returned to normal (for her) and by January 2005 she was discharged from the Outpatient Clinic back to Primary Care. Initially her level of sedation had increased when starting Olanzapine but this was fairly short lived. She did not experience any weight gain or glucose tolerance changes. Using a combination of early warning signs work and pharmacological treatments, she has remained well in terms of mental health.

This case study illustrates that for many individuals experiencing psychotropic drug induced hyperprolactinaemia, management can be fairly simple, in this case involving switching to an alternative antipsychotic drug, with no risk of relapse or an increase in adverse affects.

**Case Study (2)**

Peter has a diagnosis of Paranoid Schizophrenia.

He attends the Psychiatric Outpatient Clinic for three monthly Consultant Psychiatrist’s reviews and the Depot Clinic at the same venue.

Peter experiences ongoing psychotic symptoms, including persecutory delusional ideas and auditory hallucinations. However, these symptoms have decreased in frequency and intensity prior to transfer and he felt that most importantly he had learnt to cope with impact of symptoms through “creative distraction” and socialisation. Initially he had minimal activities, but was in regular contact with his elderly parents and the Community Mental Health Team involved in his care.

From January to July 2003 his medication package was Olanzapine 10mg daily and Depixol 20mg fortnightly. Routine blood monitoring in July 2003 was as follows:

FBC (all normal); Thyroid Function (normal); LFT (normal) and a Prolactin level of 728 mIU/L (range for males 0 – 500). At this point he was reporting erectile dysfunction and enlarged “breasts” (however no galactorhoea). A LUNSERs (Liverpool University Neuroleptic Side Effect Rating Scale) was initiated with the following user report – dry mouth, constipation, reduced libido and tremor.
In an outpatient review the above was discussed and all involved, including Peter, agreed a medication change was indicated. Therefore, the Olanzapine dose was kept at 10mg per day with the Depixol frequency changed from two to four weekly. It was decided to postpone the addition of an antimuscarinic due to existing anticholinergic effects and the possibility of tremor reduction through dose reduction of the Depixol.

Some months later he was reviewed again in the outpatient clinic. The extent of psychotic symptoms described above were now practically gone and he described himself as currently having no mental health problems, despite the reduction of total antipsychotic dose.

A prolactin level was performed – 223 mIU/L – which was now within range for males. He was no longer experiencing sexual dysfunction or known endocrine disturbances. The Parkinsonism tremor had also stopped. Some time later the depot medication was stopped completely and he remains asymptomatic and functioning well on Olanzapine 10mg daily.

Again, this case illustrates in general terms the case of reducing prolactin levels to an acceptable level with sensitive medication management. In this case dose reduction of the drug probably responsible for the hyperprolactinaemia was the effective strategy.

These case studies show that for most service users experiencing hyperprolactinaemia, medication management strategies will be effective in returning prolactin levels to normal, increasing psychotic symptomology.

Not all of our service users who have reported sexual dysfunction, such as libido reduction, erectile problems or problems with orgasm, when tested for prolactin level have elevated prolactin levels, some other causal factor is involved. We screen for thyroid dysfunction and blood glucose abnormalities as both conditions can affect sexual functioning. Other physical or psychological factors are involved. In this case we refer to colleagues in the sexual health field, often with successful outcomes. This is only a small minority of individuals, and in very general terms for people experiencing sexual dysfunction who are taking antipsychotic drugs hyperprolactinaemia is the usual cause.

**Key Learning Outcomes**

1) Hyperprolactinaemia is a relatively common complication of prescribed antipsychotic drug use. It causes the extreme distress of endocrine disturbances, including sexual dysfunction.

2) Research shows that sustained hyperprolactinaemia is linked with osteoporosis, with long term health care issues to the individual, service providers and ultimately the health of the nation.

3) Many individuals with prolactin elevation may be asymptomatic in terms of sexual dysfunction. Therefore, they may go undiagnosed for hyperprolactinaemia. Routine prolactin monitoring by venapuncture will enable the prescribing clinician to make a diagnosis of hyperprolactinaemia and treat.
4) For the majority of individuals with drug induced hyperprolactinaemia the condition is easily managed. For many dose reduction, or drug change, will eradicate the problem with no detriment to mental Health.

**Conclusion**

National Guidelines, including the Maudsley Prescribing Guidelines (Taylor, Paton and Kerwin, 2003) recommend robust treatment pathways for the treatment of hyperprolactinaemia. However, not all locality trusts are advocating prolactin monitoring and treatment as standard base line clinical practice for their service users.

I believe that this should form part of the base line assessment procedure for any individual prescribed antipsychotic treatment, especially the drugs identified as strongly associated with hyperprolactinaemia. We have also found that prolactin monitoring also allows early identification of prolactin elevating conditions. One of our service users is currently being treated for a prolactin secreting tumour that was asymptomatic at the time of the base line prolactin test we undertook. It has become much clearer to me that effective medication management in mental health requires much more than a simple two page side effect assessment. Increasingly we are required to undertake physical assessments including blood tests to identify adverse effects that may not be symptomatic or recognised by the person taking the drug or the healthcare professionals responsible for monitoring the effects of medication.

Medication management interventions, including prolactin monitoring, will improve medication concordance, thus minimising the risk of psychotic relapse due to discontinuation, reduce the impact of sexual and endocrine problems on the individual and minimise the risk of further long term complications such as osteoporosis.
References


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