Oligo-amenorrhoea – a triple whammy?
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INTRODUCTION
➢ Oligo-amenorrhoea is defined as a menstrual cycle length of > 35 days.
➢ It has various causes including thyrotoxicosis, hyperprolactinaemia, hypothalamic oligo-amenorrhoea (excessive exercise, anorexia nervosa), and Polycystic Ovary Syndrome (PCOS).
➢ PCOS is recognised as the most common endocrine disorder of reproductive-aged women around the world [1].
➢ An association of PCOS with eating disorders and autoimmune thyroiditis has been described [2,3,4].
➢ We describe an unusual case of a young patient with three different causes of oligo-amenorrhoea.

CASE HISTORY
A 20-year-old female first presented to our endocrine clinic in 2013 with a six-month history of feeling faint, palpitations, weight loss of 6kg and oligo-amenorrhoea.

➢ She was found to have autoimmune thyrotoxicosis with a fT3 of 15.2, fT4 43.3 and TSH <0.05.
➢ Her TPO antibodies were strongly positive.
➢ She was subsequently commenced on Carbimazole 20mg once a day and was biochemically euthyroid within 6 months.
➢ Interestingly, however, she continued to lose weight and remained oligo-amenorrhoeic.
➢ Her BMI was now 18 (weight 46kg).

INVESTIGATIONS

<table>
<thead>
<tr>
<th>PITUITARY PROFILE</th>
<th>2013</th>
<th>2015</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>4.7 mmol/L</td>
<td>4.2 mmol/L</td>
</tr>
<tr>
<td>IGF-1</td>
<td>9.8 mmol/L</td>
<td>13.2 mmol/L</td>
</tr>
<tr>
<td>Prolactin</td>
<td>124 µU/L</td>
<td>132 µU/L</td>
</tr>
<tr>
<td>TSH</td>
<td>&lt;0.05 mIU/L</td>
<td>1.78 mIU/L</td>
</tr>
<tr>
<td>fT4</td>
<td>43.3 pmol/L</td>
<td>13.2 pmol/L</td>
</tr>
<tr>
<td>fT3</td>
<td>15.2 pmol/L</td>
<td>4.1 pmol/L</td>
</tr>
<tr>
<td>LH</td>
<td>0.9 u/L</td>
<td>9.3 u/L</td>
</tr>
<tr>
<td>FSH</td>
<td>3.7 u/L</td>
<td>7.1 u/L</td>
</tr>
<tr>
<td>Oestradiol</td>
<td>161 pmol/L</td>
<td>255 pmol/L</td>
</tr>
<tr>
<td>Testosterone</td>
<td>1.2 nmol/L</td>
<td></td>
</tr>
<tr>
<td>SHBG</td>
<td>&gt;180 nmol/L</td>
<td></td>
</tr>
<tr>
<td>Cortisol</td>
<td>603 nmol/L</td>
<td></td>
</tr>
</tbody>
</table>

Pelvic Ultrasound: Endometrial thickness 1.5mm, no polycystic ovaries.
DEXA scan 2014: T-score -0.2 at lumbar spine, -0.1 at left hip.
MRI pituitary 2014: unremarkable

FOLLOW UP
➢ She was diagnosed with anorexia nervosa by the specialist eating disorders team (oestradiol 65 pmol/L).
➢ With their treatment and support, she gradually gained weight from 46kg in January 2014 to 54.3kg in April 2015, 65kg in September 2015 and now 73kg in 2016 (BMI 30).
➢ However despite normalisation of her body weight, spontaneous periods did not resume.
➢ Repeat pelvic ultrasound (October 2015): endometrial thickness 3mm, bulky ovaries with several small peripheral follicles, suggestive of polycystic ovaries.
➢ Reverse FSH: LH ratio (FSH 7.1U/L, LH 9.3U/L, oestradiol 255pmol/L, Prolactin 132mU/L, TFTs normal) suggestive of polycystic ovary syndrome.
➢ She has no immediate plans to start a family and is taking an oral contraceptive pill at present, which is giving her regular withdrawal bleeds.
➢ She has been given lifestyle advice to help her regain a normal weight.

CONCLUSION
➢ This case highlights multiple diagnostic and treatment challenges in a young patient with oligo-amenorrhoea.
➢ It is proposed that:
  • Her oligo-amenorrhoea may originally have been due to Graves’ disease;
  • It was subsequently due to her persistent low body weight (hypothalamic amenorrhoea secondary to anorexia nervosa);
  • and ultimately, after gaining excess weight, due to exacerbation of underlying polycystic ovary syndrome.
➢ A clinical conundrum or a triple whammy?

References: