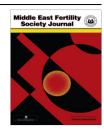


Middle East Fertility Society

Middle East Fertility Society Journal

www.mefsjournal.org www.sciencedirect.com



REVIEW

Management of hyperprolactinemic infertility

P.G. Crosignani *

Professor of Obstetrics and Gynecology, Scientific Direction, IRCCS Ca' Granda Foundation Maggiore Policlinico Hospital, Via M. Fanti, 6 – 20122 Milano, Italy

Received 10 April 2012; accepted 19 April 2012 Available online 31 May 2012

KEYWORDS

Hyperprolactinemia; Anovulation; Defective luteal phase; Pituitary adenoma; Prolactinoma **Abstract** Pathological hyperprolactinemia may cause defective ovulation and reduced fecundability. Abnormal prolactin (PRL) secretion is usually related to an idiopathic hypothalamic dysfunction or to the presence of a pituitary adenoma. The use of medication is the most common cause of functional hyperprolactinemia. Pituitary prolactin secreting adenoma is classified according to size: micro (the vast majority) being smaller than 10 mm in diameter or macroprolactinoma (very few) of larger size.

An excessive PRL secretion decreases the pulsatile release of GnRH impairing the pituitary production of FSH and LH. Furthermore it may directly impair the endocrine activity of ovarian follicles. As a consequence: defective luteal phase, inconstant ovulation and chronic anovulation are conditions frequently observed in young hyperprolactinemic patients. In addition 5% of unselected, asymptomatic infertile women show hyperprolactinemia. In such patients fertility may be promoted with long-term use of dopaminergic drugs. The normalized PRL level induced by the treatment allows the occurrence of spontaneous ovulatory cycles or the normalization of the defective luteal phase. Treatment should be continued for at least one year since half of the pregnancies occurring during dopaminergic therapy start after the first 6 months of drug assumption. An ovarian stimulation with gonadotropin and the pulsatile administration of GnRH may also induce ovulatory cycles and fertility in the infertile hyperprolactinemic patients.

Hyperprolactinemia either, due to hypothalamic dysfunction, as well as the presence of PRL secreting adenoma usually improves after delivery.

© 2012 Middle East Fertility Society. Production and hosting by Elsevier B.V. All rights reserved.

1110-5690 © 2012 Middle East Fertility Society. Production and hosting by Elsevier B.V. All rights reserved.

Peer review under responsibility of Middle East Fertility Society. http://dx.doi.org/10.1016/j.mefs.2012.04.003



Production and hosting by Elsevier

^{*} Tel.: +39 0255032256; fax: +39 0255032435. E-mail address: piergiorgio.crosignani@unimi.it

P.G. Crosignani

Contents

1.	Introduction	64
	1.1. Causes of hyperprolactinemia	64
	1.1.1. Functional hyperprolactinemia	65
	1.1.2. Pituitary tumors	65
	1.2. Ovarian function in young hyperprolactinemic women	65
	1.3. Prevalence of hyperprolactinemia among patients with infertility	65
2.	Clinical evaluation of prolactin related infertility	
	2.1. The impact of "big prolactin" on the diagnosis of hyperprolactinemia	
	2.2. Diagnosis of anovulation or defective luteal phase due to an excessive PRL secretion	
3.	Spontaneous and induced fertility	66
	3.1. Spontaneous pregnancies	66
	3.2. Dopaminergic treatments.	66
	3.2.1. Bromocriptine	66
	3.2.2. Cabergoline	67
	3.2.3. Quinagolide	
	3.3. Ovarian stimulation	
	3.4. Surgery	68
	Outcome and pregnancy complications	
	The role of pregnancy in the natural history of hyperprolactinemia	
6.	Conclusions	
	References	68

1. Introduction

1.1. Causes of hyperprolactinemia

In the woman hyperprolactinemia can be defined as the presence of abnormally high level of prolactin in the blood. Normal levels are typically 10-35 ng/ml and 1 ng is equivalent to 21.2 mU/ml (1).

Aside from high levels of prolactin observed during pregnancy and lactation, hyperprolactinemia can present as a pathological condition at any age and this excess of prolactin may result from a variety of causes, which are summarized in Table 1.

Prolactin secretion shows a circadian rhythm with higher concentration during the night and lower circulating level during the day. The regulating mechanism, independent of sleep, depends from an hypothalamic regulator and from the pituitary melatonin secretion (2).

The pronounced PRL elevation after the orgasm has been considered beneficial for decidualization and implantation (3).

A transient elevation in serum prolactin can be produced by the venepuncture stress while mildly elevated hyperprolactinemia is frequently seen in PCOS patients due to the raised circulating estrogen level (4). The raised hypothalamic TRH release observed in patients with primary hypothyroidism almost constantly stimulates prolactin secretion (5).

Dysfunction /disease	Mechanism	
Dysfunction/disease	Mechanism	
Idiopathic	Impaired hypothalamic dopamine secretion	
Pituitary tumors: micro- or macroprolactinoma,	Disruption of dopamine delivery and/or	
adenoma, hypothalamic stalk interruption	secretion of prolactin	
Acromegaly	Prolactin secretion from a GH adenoma	
Empty Sella syndrome	Damage of the pituitary	
Primary hypothyroidism	Increased hypothalamic TRH	
Polycystic ovary syndrome	Raised estrogen concentration	
Renal failure	Reduced PRL clearance	
Drugs	Mechanism	
Antidopaminergic drugs		
- Anti-psychotics (phenothiazines, haloperidol,	Inhibition of dopamine release	
butyrophenones, risperidone, monoamine,		
oxidase inhibitors, fluoxetine, sulpiride)		
- Anti-emetics (metoclopramide, domperidone)		
- Tricyclic antidepressants		
Opiates	Stimulation of opioid hypothalamic receptors	
Oestrogens	Stimulation of lactotrophs	
Verapamil	Unknown	

Download English Version:

https://daneshyari.com/en/article/3966212

Download Persian Version:

https://daneshyari.com/article/3966212

<u>Daneshyari.com</u>