

Clinical Analysis of Breast Lactation after Laparoscopic Cholecystectomy in Nonlactation Period

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Breast lactation was very rare after laparoscopic cholecystectomy in nonlactation period and two patients with breast lactation after laparoscopic cholecystectomy in nonlactation period were analyzed who were in hospital in our department in February, 2012 to November, 2013.

Patient 1, female, 39-year-old, who was admitted because of iterative right upper abdominal pain for 9 days and aggravation for 3 h. Patient 2, female, 38-year-old, who was admitted because of right upper abdominal pain for 3 days. In two cases, physical examination showed right upper abdomen tenderness, mild rebound tenderness, liver percussion pain, and positive Murphy sign. B ultrasound showed gallbladder volume augmentation, acute cholecystitis, and stones in the gallbladder neck. Finally, we diagnosed it for acute supervening cholecystitis accompanying gallstones. Laparoscopic cholecystectomy was successfully executed in general anesthesia, and morphine was used by intravenous injection for analgesia after operation and patients rehabilitated well. But Patient 1 felt breast pain and lactated 5 ml milk from breast on the fourth day after operation, blood prolactin (PRL) 58.9 ng/ml (normal: 1.9–25.0 ng/ml), and Patient 2 felt breast pain and lactated 8 ml milk from breast on the third day after operation, blood prolactin (PRL) 48.6 ng/ml, and the milk was certified by smear observing under a microscope. Then, we did some checking and found that the pituitary was normal by head computed tomography check, and original gynecological disease was not found by gynecological examination. Hence, bromocriptine was administered, 2.5 mg, 2 times in a day for 2 weeks. Breast tenderness and lactation disappeared and PRL 8.3 ng/ml (Patient 1) and PRL 6.5 ng/ml (Patient 2) after 2 weeks. Finally, the patient was cured, and breast lactation did not occur in 1-year follow-up.

Generation and secretion of milk were regulated by neuroendocrine factors such as endocrine, physiological, environmental, and genetic factors, and serum prolactin was a determinant factor of milk secretion quantity. Reasons for prolactin rise consisted of the following: (a) Physiological factors, such as pregnancy, lactation, low blood sugar. (b) Pathological factors: (1) Pituitary disease, such as prolactinoma, sella tumors, and cysts, promoted prolactin secretion by declining secretion of pituitary gonadotropin. (2) Hypothalamus and pituitary stalk disease, such as sarcoidosis, tuberculosis, craniopharyngioma, hamartoma, cranial irradiation, and empty sella, promoted prolactin secretion by blocking inhibitory effects of prolactin inhibitor factors on prolactin. (3) Primary or secondary hypothyroidism increased prolactin by raising thyrotropin-releasing hormone and thyroid-stimulating hormone. (4) Liver dysfunction raised prolactin because of abnormal liver degradation of prolactin and kidney dysfunction due to renal metabolism slows down. (5) Polycystic ovary syndrome promoted secretion of prolactin because secondary estrogen induced synthesis of prolactin cells. (6) Ectopic prolactin secretion, such as bronchial tumor and hypernephroma. (7) Acromegaly: 50% of patients with growth hormone adenomas were associated with hyperprolactinemia.^[1] (c) Drug factors, some drugs increased secretion of prolactin by antagonizing prolactin release inhibitory factor and increasing prolactin-releasing

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factor leading to reducing the role of dopamine. Dopamine receptor antagonists antagonized effects of dopamine by combining with dopamine receptor, such as phenothiazines sedative and antiemetic (metoclopramide, domperidone). Catecholamine-depleting agents, such as reserpine, methyl dopa, consumed dopamine. Antidepressants, such as tricyclic antidepressants, selectively inhibited 5-hydroxytryptamine receptors. Estrogen and contraceptives promoted the synthesis and release of prolactin by effecting on pituitary prolactin cells. Opioids promoted the release of prolactin by inhibiting dopamine conversion. In addition, there were other drugs, such as domperidone, cimetidine, isoniazid, pyrazinamide, nitrendipine, urapidil, and dexamethasone. (d) Idiopathic factors, there were not belonging to the three above categories, and the cause possibly was hypothalamic pituitary dysfunction, leading to the rise of prolactin.^[2]

There were reports that pain rose terminal hormones of pituitary-adrenal system and catabolism hormones and increased catecholamine to suppress prolactin secretion, and analgesics made maternal increase prolactin secretion, thereby increasing maternal lactation.^[3,4] Some scholars have speculated that increase of prolactin was relating to anesthetic adjuvant drugs and extensive epidural blocking which reduced synthesis and release of stress hormone (catecholamine) by inhibiting generation of dopamine in arcuate nucleus of tuberoinfundibular system.^[5] Additionally, in CO₂ pneumoperitoneum, intra-abdominal pressure increased, airway pressure rose, and CO₂ was absorbed into the bloodstream. Then, the stress response of the body produced which appeared as neural and endocrine hormone changes, and the body stimulated secretion of PRL by alpha receptors and PRL in the plasma elevated.

It was rare that women in nonlactation period lactated after routine operation and was almost not reported in the clinical. Two patients stopped breastfeeding 14–16 years before, normal menstruation, no history of abnormal lactation, no relevant pathological factors, and they lactated on the postoperative 3–4 days. We considered that two following factors may be relevant: (1) Narcotic and analgesic increased prolactin by reducing the secretion of catecholamines, (2) Mental and physical stimulation from operation increased pituitary prolactin secretion and lead to a transient lactation, regardless of the gallbladder surgery itself, and we should differently treat them in clinical.

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Conflicts of interest

There are no conflicts of interest.

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