# Galactorrhoea following thoracoscopic dissection of Schwannoma from left apical hemithorax

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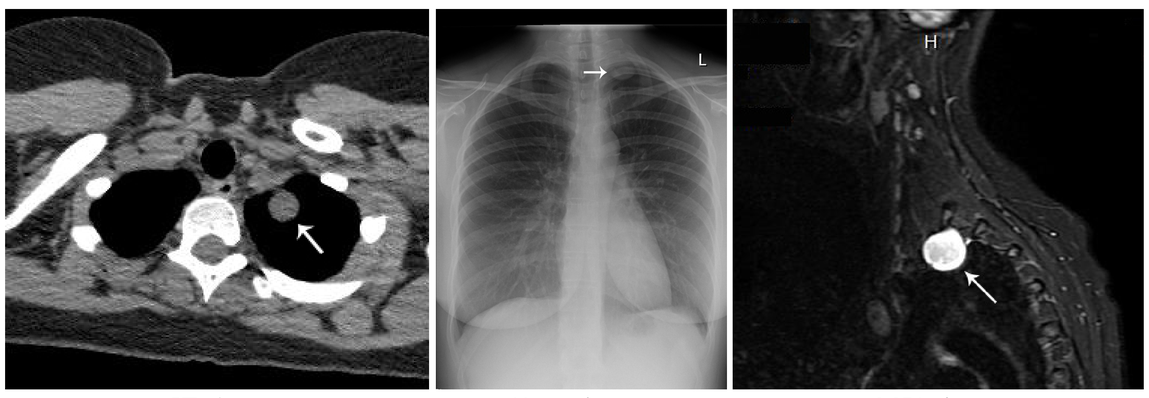
## Case History

In June 2016 a thirty four years old patient presented with symptoms of chest infection. X ray chest showed left basal consolidation and a small well circumscribed incidental shadow was noted on left apex, she had no symptoms relating to the apical shadow. She was treated for community acquired pneumonia. On discharge an outpatient CT scan of neck and chest was arranged to investigate apical shadow.

CT chest described a well circumscribed 2.4 x 2.7 cm left apical mass suggesting either a vascular lesion or a neurofibroma. Subsequent MRI showed the lesion abutting adjacent to nerve roots possibly C7 which lay anterosuperior to it, the posterior aspect extended toward the posterior aspect of the  of the first left rib, the lesion was anterior to sympathetic chain. MRI suggested high suspicion of schwannoma.

Three months post MRI she had a successful left thoracoscopic dissection for removal of schwannoma. Post-surgery she developed partial left Horner syndrome and some numbness of left arm.

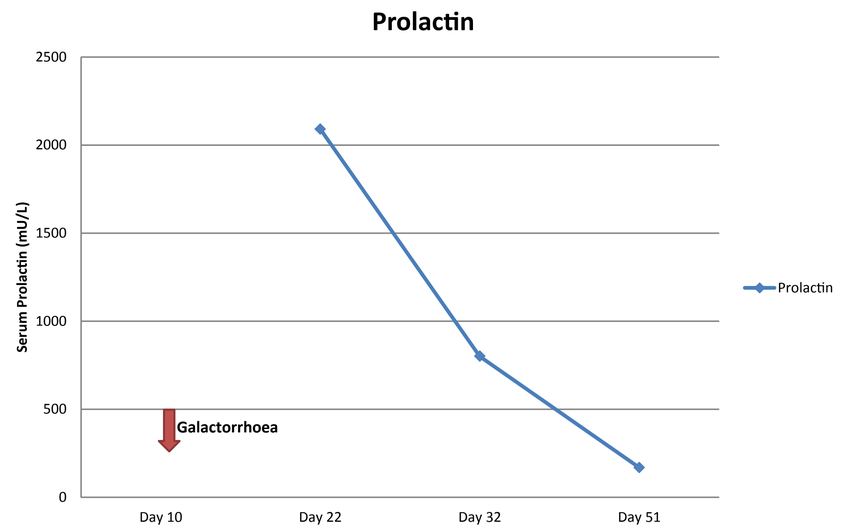


CT chest | Xray chest | MRI chest

Ten days following surgery she noted engorgement of left breast which was followed by  bilateral breast engorgement and spontaneous galactorrhoea. She had no headache or visual symptoms to suggest any pituitary lesion. She had normal mensuration following recent stoppage of oral contraceptive pill.

**On examination:** She was euthyroid, her vision was normal bilaterally, her visual fields were full to confrontation, she had mild bilateral expressive galactorrhoea. Systemic examination showed no abnormality.

**Investigation:** Reproductive hormonal profile showed raised prolactin of 2090 mU/L  (normal value 102-496 mU/L) and normal LH, FSH & oestradiol. TFT, LFT and renal functions were normal as well. Galactorrhoea and prolactin improved gradually without any treatment. Prolactin became normal after 7 weeks; mild expressive galactorrhoea persisted for further few weeks and then stopped.




## Discussion

Prolactin is a hormone secreted by anterior pituitary gland and plays an important role in lactation. Prolactin is under chronic inhibition by prolactin-inhibiting factor (PIF) which is mediated by dopamine. PIF is released by specialized neurons in the hypothalamus into the pituitary portal system and is transported to the anterior pituitary where it inhibits the synthesis and release of prolactin. Milk secretion occurs when suckling stimulates touch receptors, afferent impulses carry stimulus to paraventricular nuclei and supraoptic nucleus in the hypothalamus which signals to the posterior pituitary gland to produce oxytocin. Oxytocin stimulates contraction of myoepithelial cells to produce milk.

Various chest wall irritations have been reported to cause hyperprolactinemia and galactorrhoea, including chest wall surgery, mammoplasty, burns, herpes zoster, trauma and spinal cord injury. It is hypothesized that chest wall injury serves as peripheral triggers to autonomic control that impinge on the central neurogenic pathways that attenuate dopamine release into hypophyseal portal circulation. This stimulation of hypothalamic centre occurs through the same neural pathway involved in puerperal lactation. Cases has been described of galactorrhoea without sustained prolactin increase[1] suggested explanation: galactorrhoea occurs only in those individuals whose breast have been previously primed with oestrogen and progesterone. In these cases sudden peaks of prolactin induced by stress of surgery or chest wall injury initiate lactation which continue despite presence of normal prolactin levels.

This patient's prolactin was high 22 days post-surgery suggesting persistent rise due to neurogenic prolactin release via same neural pathway involved in puerperal lactation rather than acute rise post-surgery. She had discontinued oestrogen and progesterone containing oral contraceptive pills few weeks prior to surgery, her breasts were well primed with oestrogen and progesterone and this initiated lactation.

## References

1. I.A MacFarlan, Post Grad Medical Journal 1980

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