Lost for Words – Bilateral Vocal Cord Paresis Following Renal Dialysis of a Thyroidectomy Patient

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ABSTRACT

A patient underwent a total thyroidectomy for a small papillary thyroid cancer. Her vocal cord function was assessed as being normal post-operatively. A week later she underwent her first veno-venous renal dialysis for deteriorating renal function during the post-operative period. Within 48 hours she had developed bilateral vocal cord paresis, presumed to be on the basis of bilateral recurrent laryngeal nerve oedema precipitated by massive fluid shifts in the fresh surgical bed. Her voice recovered over the subsequent days as her fluid balance normalized.

Keywords: Recurrent laryngeal nerve, renal dialysis, vocal cord palsy.

I. CASE HISTORY

A 64-year-old lady was admitted to our hospital for a total thyroidectomy and ipsilateral central neck lymph node dissection for a small papillary cancer confined to her right lobe. A decision to perform a total thyroidectomy was based on her comorbidities, which included obesity, type 2 diabetes mellitus, asthma, stage IV chronic renal disease and a previous stroke, for which she had made a mostly complete recovery. She still used a walking stick to aid mobility. She was on numerous medications, mostly antihypertensives and vaso-active medications. She was taking Clopidogrel as single-agent antiplatelet therapy; the surgeon chose to withhold this anti-platelet medication prior to surgery.

Surgery was un-complicated; a total thyroidectomy with right central neck dissection was performed. Both recurrent laryngeal nerves were well-visualized and were preserved. The right inferior parathyroid gland was reflected inferiorly on a thinned vascular pedicle devoid of nodal tissue. The right superior parathyroid gland was auto-transplanted into the right sternomastoid muscle.

There was some slight wound oozing observed in Recovery; the surgeon appreciated that this was from a subdermal vessel and oversewed it with a continuous Prolene stitch, however by 10 hours post-operatively the neck had become more swollen, and the patient was taken back to theatre for a neck exploration. An awake intubation was performed. Both vocal cords were well-visualized and were functional. The neck was re-opened and, as expected, a sub-dermal bleeder, well in front of the strap muscles, was identified and controlled.

Clopidogrel was withheld for that next morning dose only. Following extubation the patient was found to be drowsy and had developed a left facial droop and weakness of her left extremities. A contrast CT scan of the brain did not show any cerebral changes. Aspirin commenced. A follow up contrast CT scan was done 48 hours later and showed no cerebral changes. A sensorimotor lacunar infarct was presumed. Over the course of the next few days the patient showed neurological and motor recovery. Unfortunately, the 2 doses of intravenous contrast used for the brain scanning tipped the patient’s renal function balance. Her creatinine increased from a pre-operative level of 95 micromole/L to 350 micromole/L (Fig. 1); the eGFR decreased from 21 to 11. On the 4th post-operative day continuous renal replacement therapy (CRRT), via a right femoral vein vascath, commenced. The patient became hypertensive and Prazosin was commenced with good effect.

On the 6th post-operative day neck swelling was again observed and the patient reported voice changes and difficulty swallowing. Clinical examination revealed peripheral oedema, slight swelling around the wound, but no bulge. The patient’s voice was soft and whispery. An awake intubation was again performed; this time a bilateral vocal cord paralysis was observed. The neck was re-explored; the strap muscles now being re-opened. No blood was seen within this compartment; however, the inferior parathyroid pedicle was extremely engorged, appearing as large as the surgeon’s thumb. The tissue around the laryngeal nerves was also noticeably swollen.

The following day the patient was again extubated with full voice and swallowing, recovering within 48 hours. She has continued to show neurological resolution.
II. DISCUSSION

Vocal cord paralysis following thyroid surgery is a recognised complication; in specialist units’ incidences of transient neuropraxia can be as low as 2.1–3.2% and for permanent palsy 0.1–0.3% [1]. Bilateral vocal cord paresis though is particularly rare, occurring at rates of 0.02% [2], with one reference quoting a single case in 30 000 operations [3]. Our patient did not wake up with bilateral vocal cord paresis following her total thyroidectomy but developed this over the course of the next few days. Compression from a delayed bleed was implicated, but this was ruled out following surgical exploration; a different cause for the cord paresis needed to be considered. Indeed, the possibility of a bleed causing compression of the recurrent nerves was considered most likely as renal patients are quite susceptible to hemorrhage secondary to uremic bleeding tendency and intermittent heparinization [4] and, in this particular case, a decision to continue antiplatelet therapy due to a past stroke history.

This patient did, however, undergo her first hemodialysis between her thyroidectomy and neck re-exploration. We conclude that the acute fluid shifts brought on by the hemodialysis, in the context of recent surgical trauma, let to reversible swelling of both recurrent laryngeal nerves, with early return to full vocal cord function on resolution of the local oedema.

A number of mechanisms have been reported for voice changes following hemodialysis; these relate either to injury to the laryngeal nerves, or vocal cord changes following fluid shifts precipitated by dialysis. We believe that this report is the first time where vocal cord paresis is from reversible laryngeal nerve, rather than vocal cord [5], oedema.

A number of papers have reported temporary vocal cord paralysis following hemodialysis catheter placement; however, this was secondary to laryngeal nerve anaesthesia following such vascular catheter placement within the neck, and was generally unilateral [6], [7]. In these cases, the RLN paralysis was likely due to local anaesthetic infiltration around the nerve.

Instances of vocal cord paresis have been described on the basis of possible neuropathic or microangiomatic changes to the recurrent nerve. Watanabe et al. describe a chronic renal patient with Alport syndrome [8]. This patient became hemodynamically unstable during major cardiac surgery. Confounders as to the cause of bilateral vocal cord paresis in that case include the major thoracic surgery, with possible traction on either of the RLN’s as they coursed around the major aortic branches, or the possibility of sub-acute injury when the patient previously underwent a parathyroidectomy. The longstanding chronic renal insufficiency may have been partially contributory to a complex multi-modality cause of this temporary paresis. Severe microscopic polyangiitis was described as a likely causative mechanism for a unilateral vocal cord paralysis, but in this case the nerve palsy preceded the renal insufficiency and need for dialysis [9].

The rate and direction of inter-compartmental fluid shifts in dialysis patients can be quite varied and unpredictable. Despite complex mathematical modelling [10] one of the most common problems associated with dialysis is that of hypotension induced by volume depletion [11]. To overcome this, crystalloids are often infused; this may lead to overexpansion of the extracellular fluid space leading to oedema. This sign was observed in our patient a few hours before she presented with vocal cord paralysis. Importantly, fluid overload at the commencement of CRRT may be most pronounced [12].

An occasional problem reported following dialysis is that of dialysis disequilibrium syndrome. While this syndrome typically refers to observed neurological signs secondary to cerebral oedema, one of the postulated mechanisms is that of reverse osmosis from a transient osmotic gradient [13]. Fluid may influx neurons following removal of cellular urea leading to relative isotonicity. Our patient was still fully alert and conscious just before her neck re-exploration so this mechanism may not have been responsible for central neuronal depression, however the possibility that similar local effects in post-surgically inflamed tissue exists cannot easily be evaluated.
III. CONCLUSION

We have reported a case in which a patient undergoing veno-venous renal dialysis shortly following a total thyroidectomy has developed a bilateral vocal cord paresis. We conclude that this was on the basis of reversible bilateral laryngeal nerve oedema due to pronounced fluid shifts occurring in an acute surgical field.

REFERENCES