
Internal Jugular Thrombosis Causing Increased Intracranial Pressure And Upper Airway Edema

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Citation

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Abstract

Obstruction of venous drainage from the head can result in increased intracranial pressure. We report a case of a patient with cerebral aneurysms who developed thrombosis of her right internal jugular vein. This resulted in significant edema of her head and neck, increased intracranial pressure and laryngeal edema that prevented extubation. In this case, resolution of the externally evident edema and a return to baseline neurologic function did not correspond to a complete absence of problems. In fact the true diagnosis was only ascertained when the evaluation of her respiratory difficulties included retrospective analyses of her operating room/radiology suite/ICU records and post-embolization CT scan.

Case Report: The patient was a 58 years old female, who presented with a two weeks duration of throbbing right frontoparietal headache accompanied by right retro-orbital pain, nausea and vomiting. The headache had progressively worsened, especially on the right side, and was associated with blurring of vision and ptosis of the ipsilateral eyelid.

Her past medical history included a right mastectomy for cancer and treatment for hypertension. Medications included atenolol and tamoxifen. She was allergic to sulfa drugs. The patient was a nonsmoker and without history of alcohol abuse. On physical examination she was awake, alert and responsive. Her vital signs included a blood pressure of 160/70, heart rate of 88/minute, respiratory rate of 16/minute and temperature of 98 degrees. Auscultation of the lungs revealed clear bilateral breath sounds. Her cardiac examination was normal with a regular rate and rhythm without murmurs. Positive findings included the following. She had slight nuchal rigidity and a right ophthalmoplegia. The right pupil was dilated and nonreactive with limited extraocular movement, and right sided ptosis. Muscle strength was judged to be 4/5 with hyperactive reflexes in both upper and lower extremities. She also has a left plantar extensor response. A MRI showed subacute infarcts in the

occipital lobes and ischemic changes in the periventricular white matter. Angiography showed a left carotid ophthalmic artery aneurysm with a broad neck; an aneurysm at the bifurcation of right middle cerebral artery, a right ophthalmic artery and a lobulated aneurysm involving a branch of right posterior inferior cerebellar artery.

The patient was admitted to the ICU. A pulmonary artery catheter was inserted in the right internal jugular vein. This procedure was accomplished without any complications, specifically no injury occurred to the right carotid artery during the procedure. A left radial arterial line was also placed. She remained in the ICU, neurologically and hemodynamically stable. Many discussions and conferences were held during the next ten days before she finally underwent a craniotomy for aneurysm clipping. The surgery was technically difficult and resulted in rupturing of the right middle cerebral artery aneurysm. However, the bleeding was promptly controlled and the aneurysm clipped. However there was significant concern regarding whether that clip was in optimal position. It was also found to be impossible to approach the other aneurysms surgically. Therefore the patient was left intubated and placed on mechanical ventilation postoperatively, pending a neuroradiology

attempt at embolization of the remaining aneurysms the next day.

On that morning prior to the radiology procedure, the right internal jugular vein central line site was noted to be erythematous and tender. It was decided to remove that catheter and it was replaced with another central line using the left internal jugular vein. This procedure was also uneventful.

The patient then underwent coiling and embolization of the right posterior inferior cerebellar artery aneurysm. Near the end of this seven hour procedure, the patient became severely hypertensive with systolic blood pressures above 200. Her heart rate however remained stable.

The hypertension proved to be resistant to vasodilator therapy. All of those medications were given through the freely flowing central line. The central venous pressure tracing did not change during this period. However it was felt necessary to move the radiology equipment to examine the patient and her central line. The patient's head and neck were noted to be significantly edematous. This was in spite of the patient being supine throughout the procedure with her head resting on a small pillow. The edema could not be attributed to the volume of intravenous fluids given during the procedure as she had received less than 2800ml with an urine output of 1840ml during the 7 hours procedure.

The hypertension remained very difficult to control, therefore she was again left intubated and returned to the ICU following the procedure. The next morning, the patient had a mild right-sided weakness. This had not been present the previous evening after the radiology procedure. On CT scan the diagnosis was felt to be either a left intracerebral hemorrhage and/or infarct. Also noted were a decrease in the size of her ventricles and other indications of mildly increased intracranial pressure. Her blood pressure remained labile and required sedating of the patient for optimal control. The patient was weaned from her vasodilator infusions and mechanical ventilation after 5 days. The overall edema of her head had seemingly resolved over the first three post procedure days. This edema was noted only over her head and neck, similar to what might be seen with a superior vena cava syndrome.

On the fifth post procedure day, the patient was alert and responsive and was extubated. However, she very quickly developed stridor and difficulty breathing. A racemic epinephrine nebulizer treatment was given without any

apparent relief. She became less responsive and her SpO₂ was difficult to maintain above 90% with a bag-mask system. The patient was therefore reintubated. During this laryngoscopy, her pharyngeal and laryngeal mucosa were noted to be edematous. Her vocal cords were also swollen but were moving equally and symmetrically. There was no difficulty maintaining her oxygen saturation after intubation and the patient was very comfortable. She required minimal mechanical ventilation of only 10 cm pressure support.

The patient was examined to determine a cause for this problem. The only finding of note was a small mass at the previous right internal jugular venous puncture site. Ultrasound of her neck revealed a noncompressible right internal jugular vein with echogenic density within the lumen. The left internal jugular vein was normal and compressible. The patient was started on intravenous steroids and orders written to keep the head of the bed elevated to promote venous drainage.

After 3 days she was able to breathe easily around the endotracheal tube when it was occluded and the balloon deflated. A fiberoptic laryngoscopy examination showed both vocal cords to be moving well with the right cord to still be notably edematous. This time the patient tolerated extubation without problems. Several days later she was transferred out of the unit with no further complications.

Discussion: Venous thrombosis is a known complication of internal jugular vein catheterization. The incidence of internal jugular vein thrombosis (IJVT) based on radiographic or autopsy reports has been as high as 41% to 66%.^{1,2,3} Venous thrombosis results from altered blood flow, endothelial damage with activation of clotting mechanism.⁴ Clinical presentations are nonspecific and inconsistent. These include fever, tender neck swelling, facial edema or palpable knot at the punctured site. However, the majority of patients with IJVT remain asymptomatic.

Ancillary procedures that are helpful in establishing the diagnosis of IJVT include computerized tomography (CT scan), venogram studies and ultrasonography.^{5,6,7} The CT scan findings consistent with IJVT include a low density lumen, vein engorgement and enhancement of the venous wall.⁵ Common sonographic evidence of IJVT includes intraluminal low amplitude echoes, loss of venous pulsation and respiratory rhythmicity, and a distended and incompressible vein.^{6,7}

Catheter infection is closely correlated with IJVT.^{3,8,9,10}

Timsit et al³ reported the presence of catheter-related central vein thrombosis increased by 2.6 fold the risk of catheter-related sepsis. However it is not known whether catheter-related central vein thrombosis predisposes to catheter-related sepsis or if catheter infection induces thrombus formation.

Therefore, strict aseptic technique and proper catheter care should be emphasized.

Potential complication of IJVT include pulmonary embolism, sepsis and/or septic emboli, suppurative thrombophlebitis and propagation in the superior vena cava or intracranial sinuses or veins.^{3,11} Treatment of IJVT, secondary to infection, usually consists of intravenous antibiotics and removal of infected catheter. The use of anticoagulants may be limited and not applicable in situations wherein the risk of bleeding can be catastrophic. Surgical thrombectomy or fibrinolysis are reserved for IJVT's unresponsive to antibiotics and anticoagulants.^{4,11,12}

Our patient did not have any signs of infection possibly due to antibiotic therapy originally given for surgical prophylaxis. Surgical intervention was not undertaken since she was without symptoms and still had untreated aneurysms.

There have been numerous reports of associated elevations of intracranial pressure with IJVT.^{13,14,15} Clinical manifestations in an awake patient include headache, visual disturbances and alteration in level of sensorium. Brain imaging studies may show variable findings from normal to the presence of an intracranial venous sinus thrombosis. Our patient had uncontrollable hypertension accompanied by head and neck edema. This hypertension was probably in response to the increased intracranial pressure due to the internal jugular vein thrombosis resulting in a decrease in venous outflow. In most cases, collateral venous flow will allow adequate venous drainage and prevent increased intracranial pressure and the head and neck swelling. While the left jugular vein was found to be free of thrombus by ultrasound examination, the presence of the catheter in the left internal jugular vein and presumably a small change in the patient's position prevented adequate venous drainage. It is not known how long the venous drainage was obstructed, but it was long enough to develop significant edema and increased intracranial pressure. The head and neck edema combined with the right internal jugular vein thrombosis prevented a quick return to normal, as the edema itself probably further obstructed venous drainage. It was a total of

eight days before the swelling diminished to a point which would allow extubation. Meanwhile the patient's hypertension was under control, returning to her baseline blood pressure and medication requirements in only three days.

Besides the hypertension and generalized head and neck edema, the patient responded to extubation with stridor. Typical laryngeal damage after prolonged intubation consists of mucosal ulcerations with varying degrees of laryngeal edema which if significant can cause stridor.¹⁶ Our patient's stridor can be explained by significant obstruction to venous outflow due to a right internal jugular vein thrombosis. This resulted in more of a global pharyngeal and laryngeal edema rather than isolated traumatic laryngeal mucosal edema. Direct laryngoscopy, following reintubation and again at the time of final extubation, confirmed significant pharyngeal and laryngeal edema but no evidence of irritation or trauma to the vocal cords.

Few case reports were available of similar occurrences. Power et al reported a patient with superior vena cava syndrome that developed airway obstruction.¹⁷ And Kua described a patient with airway obstruction following internal jugular vein cannulation, however that patient had a large carotid artery pseudoaneurysm and not a venous thrombosis.¹⁸

Symptomatic thrombosis of a central vein is a rare occurrence. When a patient presents with the appearance consistent with obstruction to venous drainage from the head, physicians should be aware of all the possible consequences of this condition. Increased intracranial pressure will be evidenced by changes in vital signs and in the neurologic examination. Clinicians must be aware that the potential for airway edema exists and this should be evaluated prior to extubation.

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