Fracture of an endoluminal nitinol stent used in the treatment of tracheal collapse in a dog

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- In dogs, tracheal collapse is a common and often debilitating syndrome that is sometimes refractory to medical management, necessitating surgical intervention.
- Compared with other surgical treatment options, placement of an endoluminal tracheal stent is a relatively noninvasive intervention that can provide effective relief from signs associated with tracheal collapse in dogs.
- Implantation of certain endoluminal tracheal stents can be associated with complications and therefore may best be regarded as a salvage procedure for dogs with end-stage disease that are refractory to appropriate medical management, have extensive collapse of the intrathoracic portion of the trachea, or are poor candidates for surgery.

A 5-year-old 4.5-kg (9.9-lb) castrated male Pomeranian was evaluated at the Emergency Service of the **Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania (VHUP)** for dyspnea, intractable coughing, cyanosis, and collapse of a few hours' duration. Previous medical history included a chronic cough of 4 years' duration and increased anxiety during the previous 2 months. The dog was otherwise in good health and was current on vaccines.

The previous night, the dog was treated for similar clinical signs at an emergency veterinary clinic and was administered supplemental oxygen, dexamethasone sodium phosphate, aminophylline, and enrofloxacin. However, because of increasing respiratory difficulties and an episode of collapse following discharge from the emergency hospital, the patient was referred to the VHUP.

On initial triage at the VHUP, the dog had an intractable cough; it was cyanotic and had severe inspiratory and expiratory dyspnea. Initial treatment included administration of supplemental oxygen (**fraction of inspired oxygen** [F_{IO_2}], 0.5 to 0.6), butorphanol (0.2 mg/kg [0.09 mg/lb], IM), dexamethasone sodium phosphate (0.1 mg/kg [0.05 mg/lb], IV), and aminophylline (8 mg/kg [3.6 mg/lb], IM). Physical examination revealed a mildly high rectal temperature (39.4°C [103°F]), a heart rate of 108 beats/min, inspiratory and expiratory dyspnea, and a tense abdomen. The femoral pulse quality was fair and synchronous with the heartbeat, but loud upper and lower airway noises made adequate cardiac and pulmonary auscultation difficult.

Abnormalities detected via initial diagnostic testing included decreased **hemoglobin saturation as measured by pulse oximetry**[a] (**SpO_2**, 89% with F_{IO_2} of 0.21; reference range, 94% to 100%). A CBC and serum biochemical analyses were performed at the referring emergency clinic; no major serum biochemical abnormalities were identified and hematologic abnormalities included mild leukocytosis (20.2×10^9 WBCs/L; reference range, 6.0 to 16.9×10^9 WBCs/L) and mature neutrophilia (18.1×10^9 cells/L; reference range, 3.3 to 12.0 $\times$ 10^9 cells/L). Thoracic radiographs obtained by the referring veterinarian indicated severe tracheal narrowing at the thoracic inlet with no evidence of pulmonary parenchymal or heart disease. Results of serum ELISA tests for *Dirofilaria immitis* and *Borrelia burgdorferi* infections were negative.

The dog responded well to emergency stabilization and appeared to be comfortable while receiving supplemental oxygen; however, when handled outside of the oxygen cage, the dog would decompensate quickly. The next morning, the dog was transferred to the intensive care unit for further diagnostic testing and supportive care. While in the intensive care unit, medical management consisted of administration of supplemental oxygen (FIO_2, 0.4), butorphanol (0.2 mg/kg, IV, q 4 to 6 h), dexamethasone sodium phosphate (0.08 mg/kg [0.04 mg/lb], IV, q 12 h), terbutaline (0.01 mg/kg [0.005 mg/lb], IV, q 8 h), and hydrocodone (0.25 mg/kg [0.11 mg/lb], PO, q 6 h) and appropriate hemodynamic and respiratory monitoring.

Once the dog's condition was more stable, thoracic and cervical radiography and upper airway fluoroscopy were performed. Radiography revealed severe narrowing of the trachea from the caudal aspect of the fourth cervical to the second thoracic vertebra (**Figure 1**). The heart, pulmonary vessels, and pulmonary parenchyma

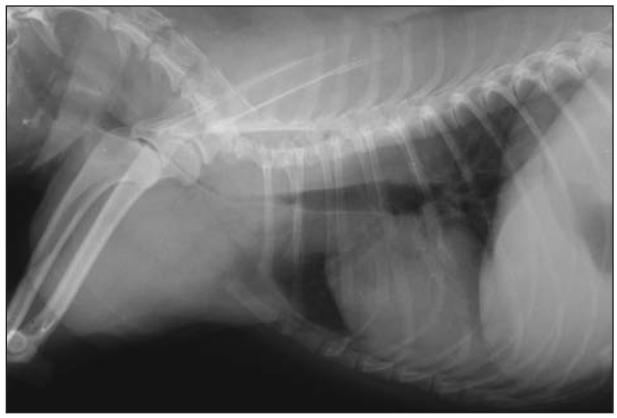

Figure 1—Lateral cervical and thoracic radiographic view of a dog with tracheal collapse. Notice the severe tracheal luminal narrowing from the level of the fourth cervical to the second thoracic vertebrae.

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appeared within normal limits. On fluoroscopic evaluation, the tracheal collapse was static (ie, the tracheal lumen remained collapsed during both phases of respiration). Because the dog would become severely dyspneic and cyanotic while experiencing paroxysmal coughing, elicitation of cough during fluoroscopic examination was avoided. The patient was anesthetized for an endotracheal lavage and bronchoscopy.

Bronchoscopic evaluation revealed a complete, static tracheal collapse in the area of the thoracic inlet that extended along 2 cm of the trachea (approx 15 to 16 cm caudal to the nares) with mild collapse cranial and caudal to the thoracic inlet. There was mild collapse of some of the major bronchi. Cytologic examination of an endotracheal lavage specimen revealed no abnormalities; however, bacteriologic culture of the specimen yielded growth of gram-positive cocci and gram-negative rods that were consistent with oropharyngeal contamination. On the basis of findings of the bronchoscopic and fluoroscopic evaluations, a complete tracheal collapse was diagnosed. After 48 hours of aggressive medical management, the dog remained dyspneic during handling or without administration of supplemental oxygen. The need to address the problem surgically was acknowledged, and the decision was made to place an endoluminal tracheal stent to help alleviate the signs associated with the tracheal collapse. Surgical consultation had been pursued and placement of either external ring prostheses or an endoluminal tracheal stent was considered. The owner chose to pursue tracheal stenting to avoid a potentially prolonged surgical procedure in this already severely compromised patient.

During anesthesia, a self-expanding, nitinol (nickel-titanium alloy) stent[b] was placed endotracheally with fluoroscopic guidance. The procedure involved 4 steps. First, the tracheal diameter was measured by use of a previously described technique to select an appropriately sized stent.[1] To obtain this measurement, a radiopaque marker of known size was placed within the radiographic field to account for magnification. The endotracheal tube was then retracted to just caudal to the larynx and positive pressure ventilation of 20 cm H_20 was momentarily administered while a lateral radiographic view was obtained. The tracheal diameter was measured at its widest point on the radiograph. Second, with the endotracheal tube in the same position and under fluoroscopic guidance, the delivery system was advanced to a position 1 to 2 cm caudal to the area of collapse and the stent (10 × 60 mm) was slowly deployed via retraction of the outer sheath of the delivery system over the stationary inner metal cannula. Third, the entire delivery system was carefully withdrawn. Fourth, a lateral radiographic view of the trachea was obtained as a record of the position of the stent at the time of placement (**Figure 2**).

After extubation, the dog received supplemental oxygen and recovered from anesthesia with minimal complications, which included mild, intermittent coughing. After complete recovery from anesthesia, the dog was able to breathe comfortably without supplemental oxygen and was moved to a regular cage in the intensive care unit. Postoperative treatment included sedation and administration of dexamethasone sodium (0.08 mg/kg, IV, q 12 h) and butorphanol (0.2 mg/kg, IV, q 4 h as needed for cough suppression). Two days later, the dog was discharged from the hospital; treatments included a tapering dosage of prednisone (0.22 mg/kg [0.1 mg/lb], PO, q 12 h for 14 days; then 0.22 mg/kg, PO, q 24 h for 14 days; and then 0.22 mg/kg, PO, q 48 h for 8 days) and hydrocodone (0.22 mg/kg, PO, q 6 h). The owners were instructed to return the dog for follow-up physical examinations and possible additional diagnostic tests in 2 and 6 weeks.

At the 2-week reevaluation, the owners reported marked improvement and that the dog had infrequent coughing episodes that occurred during periods of excitement. At that time, findings of physical examination were unremarkable. The owners were instructed to continue the gradual discontinuation of prednisone and administer hydrocodone as needed for cough suppression and sedation.

At the 6-week reevaluation, the owners reported continued marked improvement until the preceding week, during which mild progression of clinical signs that included increased coughing and gagging was detected. On physical examination, the dog appeared to be normal with the exception of mild inspiratory stridor and a cough that was easily induced by both tracheal palpation and excitement. Thoracic and cervical radiography and fluoroscopy revealed that the stent had fractured at the level of the thoracic inlet (**Figure 3**).

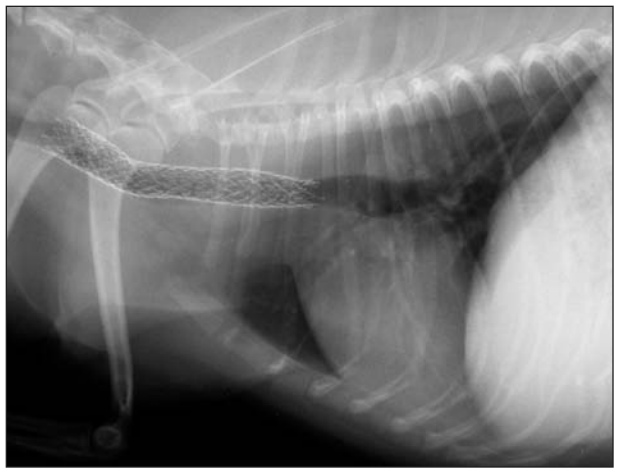

Figure 2—Lateral cervical and thoracic radiographic view of the dog in Figure 1 after placement of an endoluminal tracheal stent. Notice that the stent spans the area of tracheal collapse.

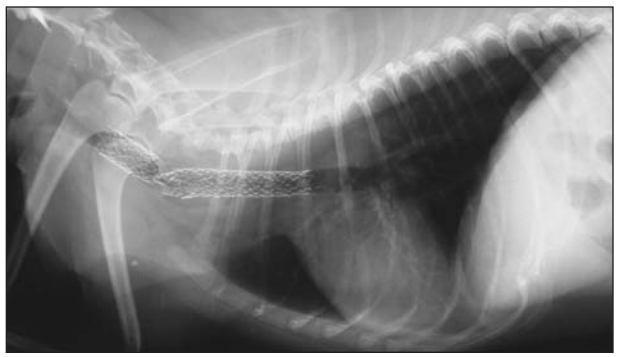

Figure 3—Lateral thoracic radiographic view of the dog in Figure 1 six weeks after placement of an endoluminal tracheal stent. Notice the complete fracture of the endoluminal tracheal stent at the level of the thoracic inlet with tracheal narrowing at the fracture site and cranial to the stent.

The tracheal lumen was narrowed at the fracture site, and there was mild tracheal luminal narrowing cranial to the stent. Bronchoscopic evaluation revealed a hyperemic tracheal mucosa with ulceration in the area of the fractured stent. At the fracture site, there were fragments of the stent protruding into the tracheal lumen that was narrowed by approximately 50% by what appeared to be a rim of granulation tissue. Despite this complication, the dog had only mild clinical signs and was still markedly improved, compared with its overall condition prior to surgery. The owners elected to pursue conservative treatment at this time; prednisone (tapering dosage) was again prescribed, and the dog was discharged.

Ten weeks after placement of the stent, the dog was evaluated at the VHUP because of excessive coughing, acute respiratory distress, and cyanosis. Findings of physical examination were similar to those obtained at the initial evaluation; stabilization procedures (similar to those undertaken initially) resulted in improvement in clinical signs. Results of a CBC revealed leukocytosis (23.8 $\times$ 10^9 WBCs/L) and neutrophilia (19 $\times$ 10^9 cells/L; reference range, 3.1 to 14.4 $\times$ 10^9 cells/L) with 8.9% bands. Results of serum biochemical analyses indicated high activities of alanine aminotransferase (584 U/L; reference range, 16 to 91 U/L), alkaline phosphatase (1,969 U/L; reference range, 24 to 174 U/L), and γ-glutamyltransferase (115 U/L; reference range, 7 to 24 U/L). The high serum alkaline phosphatase and γ-glutamyltransferase activities were likely a result of steroid administration, and the high serum alanine aminotransferase activity may have been attributable to a hypoxic insult to the liver secondary to partial upper airway obstruction. Thoracic and cervical radiography revealed that the fractured tracheal stent was unchanged in position and there was narrowing of the tracheal lumen at the level of the fracture. There was no radiographic evidence of pulmonary parenchymal or heart disease at this time.

Treatment options included resection and anastomosis of the fractured stent and associated trachea or balloon dilation and subsequent stenting of the affected trachea; euthanasia was also considered. The owners elected to pursue surgical treatment, and surgery was performed the following day. Once the dog was intubated, an endotracheal lavage was performed. Cytologic examination of the sample revealed findings consistent with suppurative inflammation; no microorganisms were grown on bacteriologic culture. Through a ventral cervical approach, 7 tracheal rings and the associated damaged part of the stent were resected by use of scissors and the ends of the trachea were anastomosed by use of an annular ligament technique. Two prosthetic rings were placed individually 1 cm cranial and 1 cm caudal to the anastomotic site. Appropriate alignment of the fractured ends was confirmed radiographically after completion of surgery (**Figure 4**). Histologic examination of the resected tissue revealed focal mucosal ulceration and granulation tissue proliferation, whereas the cartilage rings were histologically normal. In the intensive care unit, the dog recovered from anesthesia without complications. Two days later, the dog was discharged; treatment at that time was directed at decreasing airway inflammation and cough-induced airway injury and included administration of prednisone (1 mg/kg [0.45 mg/lb], PO, q 12 h for 10 days; then 1 mg/kg, PO, q 24 h for 10 days; and then 1 mg/kg, PO, q 48 h for 30 days), hydrocodone (1 mg/kg, PO, q 6 h for 10 days), acepromazine (1 mg/kg, PO, q 12 h), and amoxicillin-clavulanic acid (22 mg/kg [10 mg/lb], PO, q 12 h for 14 days). One month later, the dog was returned to the VHUP for suture removal and the owners reported stable improvement at home. On physical examination, there were no abnormalities and the dog was eupneic with no tracheal noise and minimal laryngeal noise on auscultation. One year after surgery, the dog was doing well and required treatment with hydrocodone infrequently.

Tracheal collapse is a common syndrome that is observed most frequently in middle-aged and old toy- and miniature-breed dogs and is only sporadically encountered in large-breed dogs and other species. Clinical signs include paroxysmal coughing episodes and dyspnea, which may be exacerbated by excitement. The syndrome is characterized by a dorsoventral flattening of the tracheal rings with laxity of the dorsal tracheal membrane that can result in dynamic or static obstruction of the trachea anywhere along its length. The trachea at the thoracic inlet is commonly affected, and the mainstem bronchi and smaller airways can also be involved.[2] The underlying cause of tracheal collapse is controversial and is likely to be multifactorial. Congenital or acquired changes that result in weakness of the tracheal rings and laxity of the dorsal membrane have been implicated. Regardless of the primary abnormality, it is likely that chronic inflammation caused by repeated collapse adversely affects the biochemical, ultrastructural, and biomechanical properties of the trachea in these dogs.[3-5]

A diagnosis of tracheal collapse is typically made on the basis of clinical signs, history, and physical examination findings and confirmed via radiography, fluoroscopy, or tracheoscopy. Thoracic and cervical radiographic findings have been reported to yield a diagnosis of tracheal collapse in 59% to 84% of affect-

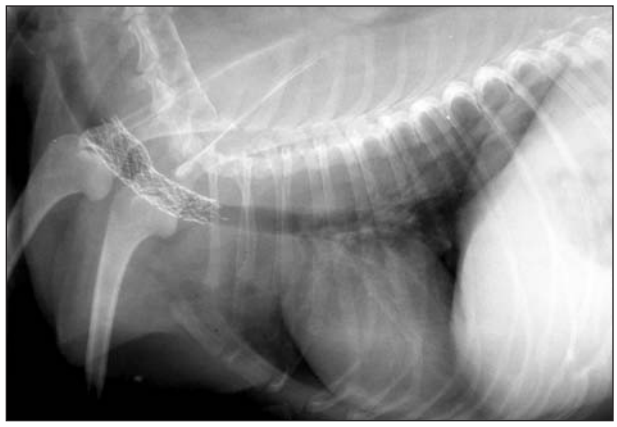

Figure 4—Lateral thoracic radiographic view of the dog in Figure 1 after resection and anastomosis of the fractured portion of the endoluminal tracheal stent and associated trachea. Notice the shortened stent. Radiographically, there were no signs of pneumothorax or pneumomediastinum. Evaluation of a ventrodorsal radiographic view revealed that the fracture ends were appropriately aligned.

ed dogs, but the tracheal dynamics during tidal breathing or coughing cannot be observed via radiography.[6,7] Fluoroscopic examination in awake animals enables evaluation of the trachea and mainstem bronchi during normal breathing and, importantly, during coughing. Whenever possible, it is preferable to elicit coughing during fluoroscopic examination because dynamic collapse of the intrathoracic portion of the trachea and mainstem bronchi may become more pronounced, thereby enabling assessment of the true severity and location of the collapse. As a means of direct evaluation of the airways, tracheobronchoscopy is a sensitive diagnostic technique for identification of the location and assessment of the severity of tracheal collapse.[7,8] Additionally, bronchoscopy may reveal signs of bronchitis, bronchiectasis, or collapse of smaller airways; during the procedure, specimens from the smaller airways can be collected for cytologic examination and culture.

Concurrent or concomitant diseases in dogs with tracheal collapse may result in acute exacerbation of a previously stable chronic disease state. Identification and correction of these disorders may decrease the frequency and severity of episodes of respiratory distress, thereby obviating the need for surgical management of the tracheal collapse. Radiographic evaluation of the thorax should be performed to detect small airway disease (eg, allergic or infectious bronchitis), pulmonary parenchymal disease (eg, pneumonia, edema secondary to congestive heart failure, neoplasia, or pulmonary hypertension), and heart disease (eg, left atrial enlargement causing compression of major bronchi). Laryngeal examination may reveal upper airway abnormalities such as laryngeal paralysis or collapse or brachycephalic airway syndrome in certain breeds. Routine hematologic and serum biochemical analyses and urinalysis should be performed, and on the basis of clinical suspicion, ancillary diagnostic tests may also be indicated (eg, to detect *D immitis* infection). Diseases such as diabetes mellitus, hyperadrenocorticism, and hypothyroidism may predispose to obesity, which can exacerbate tracheal collapse.

Medical management for stabilization of dogs with tracheal collapse is routinely recommended prior to surgery; treatments include administration of supplemental oxygen, steroids at anti-inflammatory doses (via the oral or inhaled routes), and antitussives and sedation.[9] Diagnosis and treatment of concomitant problems is essential and may include dietary management for obesity, administration of antimicrobials in dogs with pneumonia, and administration of bronchodilators for the treatment of small airway disease. In the acutely collapsed patient or in dogs for which appropriate medical management is unsuccessful, surgical correction may be recommended. Common surgical options for tracheal collapse include placement of extraluminal prosthetic devices and endoluminal tracheal stents.[2,6-14] The placement of extraluminal polypropylene C-shaped rings is an effective surgical treatment for dogs with collapse of the extrathoracic portion of the trachea, although the procedure has been associated with infection, disruption of tracheal blood supply, laryngeal paralysis, chronic coughing, and death.[6,8,11,15,16]

The use of an endoluminal stent for the treatment of tracheal collapse in dogs has been reported.[1,10,17,18] There are many types of stents available, each with their own characteristics and limitations. The endoluminal tracheal stents currently used in veterinary practice to treat tracheobronchial collapse include devices that were originally designed for use in the gastrointestinal tract, vascular or biliary systems, or airways in humans. The type of stent used in the dog of this report was a nonreconstrainable, nonforeshortening, self-expanding nitinol stent. Nitinol was originally developed in the 1960s by the Naval Ordinance Laboratory and has been described as a memory metal that can be constrained within a narrow delivery device and can resume its original conformation after release.[19] The delivery system consists of an outer sheath, which covers and constrains the stent, which, in turn, is compressed onto a metal cannula. The nonforeshortening property of the stent refers to its ability to maintain its length as it expands during deployment, compared with foreshortening stents that can shorten considerably depending on the degree to which they expand to their predetermined diameter. This nonforeshortening property allows for precise placement of the stent regardless of the diameter to which the stent opens. The term nonreconstrainability refers to the fact that the stent cannot be recaptured (reconstrained), even after partial deployment. This is a limitation of most currently available nitinol stents; however, the final position of a nonforeshortening stent is easily predicted with appropriate measurement, and the need to reconstrain is uncommon.

There are many advantages of the use of endoluminal tracheal stents for tracheal collapse. Stents can be placed within the intra- and extrathoracic portions of the trachea. Compared with other surgical treatment options, they are relatively noninvasive to place and do not require surgical dissection around the tenuous tracheal blood supply and recurrent laryngeal nerve. Duration of anesthesia is generally < 1 hour, and placement of the stent itself often requires < 10 minutes. Tracheal stents provide rapid and effective relief of clinical signs and are generally well tolerated by the recipient.

In addition to stent fracture, reported complications of placement of endoluminal tracheal stents in dogs include coughing, stent migration, growth of microorganisms on microbial culture of tracheal specimens, pneumonia, expectoration, granulation tissue formation, squamous metaplasia and ulceration of the tracheal epithelium, stent collapse or deformation, and acute pulmonary edema.[1,10,18,20] Other complications associated with placement of nitinol stents in humans include failure to expand, stent misplacement, perforation of tracheal tissue, and bleeding.[21,22]

There are no absolute contraindications to stent placement for treatment of tracheal collapse, but the procedure may not be appropriate in dogs with an active respiratory tract infection because of the impairment of the mucociliary apparatus by the stent. However, the tracheobronchial clearance mechanisms in dogs with tracheal collapse are likely markedly abnormal because of the tracheal epithelial changes

secondary to chronic inflammation and because of the collapse itself. Alleviating or reducing the chronic inflammation associated with tracheal collapse may provide the opportunity for growth of more functional tracheal mucosa through the stent. In 1 study,[18] the effects of placement of balloon-expandable intraluminal metallic stents in the trachea and mainstem bronchi of healthy dogs were evaluated. In those dogs, in the areas where the stent struts were integrated into the tracheal epithelium, growth of normal pseudostratified ciliated columnar epithelium and areas of inflammation were detected; not all sections of the trachea were examined histologically and problems with stent sizing, migration of the stent, and infection limited the ability of those investigators to make conclusions about the extent of integration or inflammation. The gross and cellular responses to self-expanding intraluminal tracheal stents require further investigation.

Nitinol stents have been used in dogs with tracheobronchial collapse at our institution and elsewhere.[1,10] To the authors' knowledge, this is the first report of a fractured nitinol stent in a dog. The fracture occurred at the thoracic inlet and may have been a result of cyclical fatigue of the metal at a high motion area of the trachea. It is interesting to note that the dog had minimal clinical signs associated with the stent fracture at the time it was detected. However, severe clinical signs did develop subsequently; the dog's progressive symptoms were likely a result of tracheitis, tracheal stenosis secondary to severe granulation tissue formation, and, potentially, progressive tracheal collapse elsewhere.

Our experience suggests that the placement of self-expandable endoluminal stents can provide relief from the signs associated with tracheal collapse in dogs; however, placement of stents may still be associated with various complications. At present, tracheal stenting at our institution is considered a salvage procedure in dogs with end-stage disease that are refractory to appropriate medical management, have extensive collapse of the intrathoracic portion of the trachea, or are poor surgical candidates. Because of the findings in the dog of this report, the authors have concerns about spanning high motion areas of the trachea such as the thoracic inlet with a stent. Vascular stenting across high motion areas (eg, joints) is routinely avoided in humans for this reason.[23] As the findings in the dog of this report indicate, there is a need for further investigation of the biomechanical properties and tolerance by host tissues of different stent materials.

[a]Ohmeda Biox 3700, Boulder, Colo.
[b]SMART nitinol stent, Cordis, Miami, Fla.

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