Loeys-Dietz syndrome (LDS) is a rare autosomal dominant disease related to genetic mutations in receptors for the cytokine transforming growth factor-receptor type 1 (TGFβ-R1) or 2 gene (TGFβ-R2) on the cell surface. LDS results in abnormal protein synthesis and dysfunctional connective tissue, which can result in unique cardiovascular anesthesia challenges related to perioperative management. Patients with LDS may manifest hypertelorism, bifid uvula or cleft palate, and arterial tortuosity. Virtually all LDS patients show some type of abnormal skin findings and bleeding tendency. These patients may show a rapid progression of aortic dilation, regurgitation, and a propensity towards rupture and/or dissection at a much earlier age and smaller aneurysm size. LDS patients who require surgical intervention require meticulous vigilance from the anesthesiologist. We describe a 26 year old patient with documented LDS type 1 who presented for repair of an ascending/root aneurysm in this case report. Recognition of LDS and intra-operative management of the cardiovascular manifestations of this disease is paramount in ensuring successful surgical outcome and to limit morbidity and mortality.

Introduction

Loeys-Dietz syndrome (LDS) is an aggressive connective tissue disease leading to potential life-threatening aortic aneurysms. Genetic mutations leading to abnormal protein synthesis and poor development of the body’s connective tissue lends to some features similar to Marfan syndrome. However LDS has some unique physical characteristics that set it apart from other connective tissue disorders. Phenotypic abnormalities described as hypertelorism, bifid uvula or cleft palate, and arterial tortuosity can manifest in these patients. Almost all patients show some type of abnormal skin findings including translucent skin, soft or velvety skin, easy bleeding, easy bruising, recurrent hernias, and scarring problems. Mutations in receptors for the cytokine transforming growth factor-receptor type 1 or 2 gene have been identified as the etiology. It is important to distinguish between Marfan’s syndrome and Loeys-Dietz syndrome because there are a few management differences. LDS patients may show a rapid progression of aortic dilation, regurgitation, and a propensity towards rupture and/or dissection at a much earlier age and smaller aneurysm size. Surgical intervention necessitates teamwork between many different specialties often including the cardiologist, surgeons, nursing, and in particular, meticulous vigilance on the part of the anesthesiologist. Comprehensive care of patients with LDS involves a wide scope of knowledge, support, counseling, and education.
Case Report

We describe a 26 year old patient with documented LDS type 1, who presented for repair of an aortic arch aneurysm. One year previously, she underwent endovascular coil embolization of 3 of 4 gastric aneurysms. Access to the fourth aneurysm had proven to be technically challenging and not feasible. Upon review of her medical history, she complained of a history of migraines, hypertension and joint hypermobility. The patient stated that her abdominal pain had improved throughout the year following embolization. No family history of connective tissue disease was elicited. Hypertelorism and a repaired bifid uvula were noted in her past medical history. Craniosynostosis and cervical spine instability were not present. Diagnostic imaging previously revealed a right to left shunt per TTE bubble study, a tortuous left vertebral artery, numerous splanchnic artery aneurysms and a 37mm aortic root measured at the sinus of Valsalva. Selective angiogram a year prior demonstrated complete thrombosis of the common hepatic artery. Arterial inflow to the liver was noted at that time as via retrograde flow from the gastroduodenal artery via the pancreaticoduodenal arterial network. The patient had been placed on the angiotensin receptor antagonist Losartan at the time of her embolization. EKG, C-spine films and CXR were normal. Vital signs: 130/90, HR 82, RR 22, and Temp of 98.7 F. Airway examination was documented as a Mallampati I with good extension and flexion of the cervical spine.

Details of the anesthetic plan including all intravenous access and prolonged intubation were explained to her. PFT’s and baseline ABGs were noted to be normal. The patient was pre-mediated with Midazolam 2 mgs prior to entering the operating room. An additional 4 mg of Midazolam and 100mcg of Fentanyl was titrated during right radial arterial line placement along with a pulmonary artery catheter (48cm) via the right internal jugular vein. Ultrasound guidance was used during access of the internal jugular. All systemic and pulmonary artery pressures were within normal limits with SV02 readings around 80%. After thorough pre-oxygenation, induction was performed using Etomidate 10 mg, Fentanyl 250mcgs, 100mg of 2% Lidocaine and 100mg of Succinylcholine. Intubation was accomplished without difficulty using a conventional laryngoscope with a Miller 2 blade. Additional maintenance was provided with Sevoflurane 2% in oxygen.

There were minimal cardiovascular responses to intubation or sternal splitting and no abnormal bleeding was seen intraoperatively. Purse strings were placed in the arch of the aorta and right atrium. The patient was given heparin intravenously. Intra-op TEE revealed the ascending aorta at the sinuses to be 48mm in size, and described as “very thin walled”. Aortic and vena cava cannulas were placed. The patient was placed on cardiopulmonary bypass (CPB) and the aorta cross clamped. Antegrade and retrograde cardioplegia was given every 15 to 20 minutes. Buttons on the right and left main coronary arteries were made. A 10mm Hemaseel graft was sewn end to end to the left main and brought posteriorly. The aortic valve leaflets were excised and a 29 mm St. Jude valve conduit was seated, sewn and tied into place. An end graft to the ascending aorta just proximal to the innominate was performed. The Cabrol was brought around to the right side of the ascending aorta and sewed end-to-side to the valsalva portion of the graft. The button of the right coronary was sewn directly into the valsalva portion. The patient was weaned off CPB with the support of
Epinephrine at a dose of 5 mcg/min and Milrinone at a dose of 0.25 mcg/kg/min. Pulmonary artery pressures were 20-29/13-15 mmHg with a Cardiac Index of 3 to 3.5 L/min/m² post CPB. TEE monitoring showed an adequate valve repair along with adequate myocardial contractility. Biastral and Biventricular leads were placed and the chest was closed in routine fashion. She was weaned off the epinephrine continuous infusion and extubated approximately 4.5 hours later. The patient’s post-operative course was uneventful. Coumadin therapy was initiated. All lines were removed on post-op day 1. She was transferred to the floor post-op day 2 and discharged home on Post-op day 5.

Discussion

Molecular and pediatric geneticists, Dr. Harry Deitz, set out to understand the mechanisms behind aneurysm syndromes. Through genetically altered mouse models, he was able to reveal new and unanticipated processes that were driving connective tissue syndromes such as Marfan’s. Marfan’s syndrome is a deficiency in the protein fibrillin-1. This deficiency leads to enhanced activation of the TGF-B molecule, which then stimulates the cells by binding to a receptor (TGF-B-R) that sits on the cells surface. This enriched TGF-B stimulation of the cells leads to many features of Marfan’s such as dislocation of the lens of the eyes, overgrowth of long bones, low fat stores and weak tissues.

Throughout his research of aneurysmal syndromes, Dr. Dietz found that many genes for other connective tissue diseases are really all funneling down a single pathway, the TGF-B pathway. In 2005, Dr. Dietz and his colleague Dr. Bart Loeys described a new syndrome known as Loeys-Dietz. The natural history of this syndrome is characterized as being more aggressive than Marfan’s, having unique physical markers and higher risks of aortic tear at an earlier age.

Loeys-Dietz syndrome is an autosomal dominant genetic disorder, which has been characterized after discovery of heterozygous mutations in genes encoding for transforming growth factor beta receptors 1 (TGF-B-R1) and 2 (TGF-B-R2) on the cell surface. Gene mutations of TGF-B2, TGF-B-R1, TGF-B-R2 and SMAD3 have been implicated. The pathway known as the transforming growth factor beta signaling pathway is involved in an array of cellular processes. These genetic mutations cause increment or up regulation of downstream TGF-B signaling, which results in overproduction of collagen, disarrayed elastic fiber with loss of elastin content in the aortic media and vascular tree. Actual mouse models with introduced mutations in the type 1 receptor genes and type 2 receptor genes, LDS mice, showed rapid growth of the aorta, aortic wall abnormalities, tortuosity of blood vessels, winding coronary arteries, eventually leading to death by aortic tear. Many mice exhibited skeletal features of the LDS syndrome as well.

Initially 2 types of LDS were distinguished; however, a continuum of the disease with variability in the gene mutation leading to differing physical attributes has been recognized. Four forms or types of LDS have been identified:

Type 1 (OMIM #609192) - Patients present with typical craniofacial features i.e., cleft palate/bifid uvula, craniosynostosis and/or hypertelorism. The triad of hypertelorism, palate/bifid uvula and arterial aneurysm/tortuosity is most specific for this type. These patients are more likely to have cardiovascular surgical interventions at younger ages. A shorter life span exists in type 1 LDS patients left untreated.

Type 2 (OMIM #608967 & #610380) - Patients have milder craniofacial features, but exhibit skin abnormalities such as velvety translucent skin, bruising and atrophic scarring, and joint laxity.

Type 3 (OMIM #61375) - Patients have a mutation in protein SMAD3 which was initially described as the aneurysm-osteoarthritis syndrome. Currently classified as LDS type 3, these patients exhibit craniofacial abnormalities similar to type 1. Radiographic evidence of osteoarthritis can be seen as early as age 12.

Type 4 (OMIM #614816) - TGF-B2 mutations are implicated in Marfan syndrome and LDS patients. The clinical phenotype shows features similar to Marfan patients; bifid uvula, mitral valve disease, club foot, aortic aneurysm, and skeletal signs. These individuals seem to have a high incidence of cerebral manifestations such as stroke, cerebral aneurysm, and
subarachnoid hemorrhage.

Most likely, in the past, many patients with LDS may have been diagnosed with Marfans. It is important to distinguish between Marfans syndrome and Loey-Dietz syndrome because there are subtle differences both with symptoms and treatment (Table 1).

Although, specific phenotypic features are noted and categorized into various types, much overlap is seen. One common theme throughout the continuum of LDS is the predisposition to aggressive vascular disease, leading to dilatation and to dissection of vessel walls. LDS is associated with a high risk of arterial rupture or dissection. Abdominal aortic aneurysms have been identified in 10% of patients, while head and neck aneurysms occur in 10% of patients. Additional clinical features of the two syndromes are presented in Table 2.

**Table 1**

*Comparison of Marfans versus LDS*

<table>
<thead>
<tr>
<th></th>
<th>Marfans</th>
<th>LDS type 1</th>
<th>LDS type 2</th>
<th>AOS/LDS type 3</th>
<th>LDS type 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotype defect</td>
<td>fibrillin-1</td>
<td>TGFβ-R1 or TGFβ-R2</td>
<td>TGFβ-R1 or TGFβ-R2</td>
<td>SMAD 3</td>
<td>TGF-b2</td>
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<td>Inheritance</td>
<td>AD</td>
<td>AD</td>
<td>AD</td>
<td>AD</td>
<td>AD</td>
</tr>
<tr>
<td>Age</td>
<td>Childhood through Adulthood</td>
<td>Childhood-20s</td>
<td>Teens-20s</td>
<td>Middle age adult but can be early teen</td>
<td>30s-40s</td>
</tr>
</tbody>
</table>
Table 2  
Specific clinical features of Marfans Syndrome and Loeys-Dietz Syndrome

Marfans
- Mutations in genes encoding fibrillin leading to deficiency
- Dislocation of lens of eyes
- Overgrowth of long bones
- Pneumothorax potentials
- Low fat stores
- Dural ectasia
- Aortic aneurysms
- Life expectancy mid to late 40’s
- Prophylactic surgery with sinus diameter > 5 cm or family history of rupture

LDs
- TGF – B receptors
- No dislocation of eye lens
- Occasionally pneumothoraces
- Dural ectasia
- Hyperteleroisma
- Craniosysostosis
- Bifid uvula
- More aggressive
- Life expectancy mid 20’s
- First surgery 18 yrs.
- Life threatening events in pregnancy
- Treatment options: prophylactic surgery earlier: Adults > 4 cm and Children > 3 cm
- Septal defects more common than in Marfans.

In order to understand connective tissue disorders, it is imperative to understand that cell behaviour is regulated by growth factors that activate transmembrane receptor kinases. Enzymatic cascades that regulate gene transcriptions are initiated by substrates of these kinases. Although growth factors have specific receptors, the pathways can be highly interconnected. The transforming growth factor beta (TGFβ) signaling pathway is one such pathway. It is involved in many cellular processes of developing embryos and also growth in adults. TGF-B regulates cell growth, cell differentiation, apoptosis and cellular homeostasis. Intracellular proteins known as SMADs phosphorylate this TGF-B ligand which is essential for extracellular signals to traverse the cell and gain entry to the nucleus. These proteins in the nucleus then up or down regulate gene expression.

In addition, intracellular effectors such as Erk 1 and Erk 2 phosphorylate an array of transcription factors, in order to regulate cells. (Davies et al, 2005) noted that Erk activation, and Smad signalling are both necessary for TGF-β-induced epithelial-mesenchymal transformation, a key event in neoplastic invasion and metastasis. Activation of Erk is necessary for TGF-β induced fibroblast replication.

During the past 2 decades, research has shown that genetic factors play a key role in the formation of thoracic aortic aneurysms. Dysregulation of the transforming growth factor (TGF)-B signalling pathway has been identified as the key culprit in the pathogenesis of thoracic aortic aneurysms. The majority of individuals with Loeys-Dietz syndrome are diagnosed with aneurysms. Aneurysms not only occur at the aortic root, but may be found throughout the arterial tree. Dissections have occurred at smaller aortic root diameters (4 cm), and at potentially younger ages as opposed to other connective tissue diseases such as Marfans (5cm). Clinically, type 1 patients seem to have a worse prognosis when compared to type 2 patients and usually present earlier at the time of their first cardiovascular surgery (mean age 16.9 years versus 26.9 years) and die younger (mean age 22.6 years versus 31.8). It is important in these patients to have both timely diagnosis and prompt imaging. Surgical intervention of aneurysms to prevent life-threatening vascular events is often warranted. Valve-sparing aortic root replacement surgery may be recommended to avoid the need for anticoagulation based on the degree of involvement.

These recommendations have been implemented by the vast majority of cardiovascular societies.

- Patients with Loeys-Dietz syndrome should undergo complete aortic imaging at initial diagnosis and 6 months thereafter to establish if enlargement is occurring.
- Patients with Loeys-Dietz syndrome should have yearly magnetic resonance imaging from the cerebrovascular circulation to the pelvis.
- It is reasonable to consider surgical repair of the aorta in all adult patients with Loeys-Dietz syn-
drome or a confirmed TGFBR1 or TGFBR2 mutation and an aortic diameter of 4.2 cm or greater by transesophageal echocardiogram (internal diameter) or 4.4 to 4.6 cm or greater by computed tomographic imaging and/or magnetic resonance imaging (external diameter).

This goes along with the premise of gene-tailored management leading to recommendations of prophylactic surgery based on underlying genetic makeup. LDS patients with a mutation in TGFBR1, TGFBR2 or SMAD3 should be a surgical candidate when the ascending aorta reaches 40 to 42 mm diameters by echocardiogram. However, the TGFBR2 patients seem to have milder aortic disease, and this group of patients is small with specific treatment guidelines under review.2,5

Despite recommendations that patients with LDS undergo extensive surveillance at a young age, knowledge and awareness of this newly described syndrome may not be established. In the case of our patient, she had been referred from a vascular surgeon, outside the institution, who failed to establish a clear diagnosis of LDS.

Cervical-spine instability has been observed in about 15% of individuals with LDS. Pre-operatively, C-spine imaging, both in the flexion and extension positions should be evaluated as it may impact anesthetic management especially during intubation. A small proportion of individuals with LDS require cervical fusion surgery to stabilize their spine.3,4

Female LDS patients are at increased risk of obstetric complications and genetic counseling is necessary. There is a high risk of aortic dissection or uterine rupture during pregnancy and the immediate postpartum period. Many women with LDS have had successful pregnancies. It is unfortunate that there are no predictors to better identify which women may potentially experience complications. Beta blocker usage is often maintained throughout pregnancy for hemodynamic control. Early delivery and elective Cesarean section to reduce high intra-abdominal pressure, and reduce the risk of complications are often employed. Though intuitive, there is an absence of studies comparing the efficacy of cesarean and vaginal deliveries.5 However, Dr. Dietz points out that the vast majority of aortic tears occur a few weeks after delivery, and that performing Cesarean sections did not seem to prevent aortic ruptures from occurring. The question of what starts at the end of pregnancy, and is maintained after delivery is the hormone oxytocin. Oxytocin stimulates uterine contraction, and also milk letdown in the postpartum period. Oxytocin is sustained during breastfeeding, and mediates its effects on peripheral tissues through Erk activation. Through mouse models of MFS, Habashi et al. found a response to oxytocin receptor expression upregulation in the aorta during pregnancy and estrogen release. 96% of the mice died in pregnancy due to aortic rupture. Mice pups separated from the mothers, resulted in a 70% female survival rate. Therefore the natural hormone, oxytocin may not be good in LDS individuals. The FDA approved drug Atosiban, which inhibits preterm labor, also blocks oxytocin and may prove to be helpful in preventing aortic growth.20

Dural ectasia has been identified in connective tissue diseases such as Marfan’s and Ehler’s Danlos and LDS. Regional anesthesia has been performed on these patients for labour and Cesarean section. However, Lacassie et. al describe two cases of Cesarean section where subarachnoid anesthesia failed to provide adequate surgical analgesia. The authors attribute this patchy block to an increased lumbosacral CSF volume. The erratic spread of spinal anesthesia in both of these cases was most likely the result of dural ectasia and increased CSF volume.

Our G4P2 patient had previously undergone extensive genetic testing, leading to a diagnosis of LDS, after giving birth to a premature dysmorphic appearing child. An IUD had been placed in our patient, but subsequent symptoms of increased abdominal pain prompted removal. She had previously been counseled extensively regarding risk of pregnancy.

Medical therapy had been aimed at decreasing wall stress and tension within the aorta by using anti-hypertensive agents such as Beta blockers and ACE inhibitors. Losartan, an angiotensin receptor antagonist, has been shown to improve aortic wall architecture along with slowing the rate of aortic dilation in Marfan mice. The angiotensin pathway is a regulator of the TGF-B pathway. Thus, the strategy of utilizing Losartan as a means to arrest aneurysmal growth has been studied.5,5 Losartan is not just lowering blood
pressure, but blocking the TGF-B activity. Lacro et al are conducting an ongoing multi-center clinical trial in humans to compare outcomes in individuals with MFS randomized to either atenolol or losartan\textsuperscript{21}. Losartan has relatively low side effects; however, the drug has the potential for fetal toxicity.

The assumption that all blood pressure medications are good for these syndromes may not be accurate. Calcium channel blockers in mouse models have shown aggressive increases in aortic wall thickness by activation of Smad and Erk. This activation of the TGR-B pathway may propagate aggressive growth of an aneurysm\textsuperscript{22}.

As with the case of our patient, hemodynamic stability during the intra-operative period in order to minimize shear stress and aortic wall tension is of utmost importance. It has been shown previously that general anesthesia can be successful in these patients often using a “balanced” approach of narcotic therapy along with inhalational agents. Our general anesthetic technique was effective in preventing tachycardia and hypertension in this patient.

In conclusion, Marfan and LDS mouse models have helped with the development of treatments to prevent aneurysms from forming for the lifetime of the mouse. Through this advanced research, geneticists continue to unravel the mysteries on TGF-beta signaling, and the potential treatment role of TGF-beta antagonists.

Physician awareness of this disease is critical due to the aggressive nature of the disease, and high risk of mortality and morbidity. Anesthesiologist will be faced with challenges related to this new syndrome. Exposure will occur not only in the operating room but also in the labor and delivery suites. Recognition of various treatment modalities for blood pressure management, obstetric pain management, and perioperative risks should be foremost on the anesthesiologist mind. Recognition of LDS and intra-operative management of the cardiovascular manifestations of this disease are of utmost importance. Parents, families and individuals affected with Loeys-Dietz syndrome may seek support and guidance from multiple specialists including their anesthesiologist.
References

1. LOEYS BL, CHEN J, NEPTUNE ER, et al: “A Syndrome of Altered Cardiovascular, Craniofacial, Neurocognitive and Skeletal Development Caused by Mutations in TGFBR1 or TGFBR2”.


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