Housecalls

Winter 2019

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INSIDE THIS ISSUE

GENETIC TESTING

The Nuances Matter

It is that time of year again. You know what I mean, time to look back and give your opinion on the most significant innovations that have the potential to affect our industry. For underwriting in general you would have to say accelerated underwriting or automated underwriting in some form is huge as it is now impacting millions of policies. And the potential exists for even more impact as more sources of data become available.

But, my focus is more on medical underwriting: the cases that are too large to automate or the proposed insured's age is outside the typical age range or, more commonly, there is a medical impairment that is kicked out by the algorithms.

My choice for the medical innovation that may have the most impact on risk assessment is the liquid biopsy. This is a commonly used term for the isolation and analysis of cell free DNA (cfDNA) or RNA that is derived from malignant tumors. Due to the cell turnover associated with tumors, partial or intact cells are released into the blood stream or other bodily fluids. Technology has improved to the point that very small amounts of this material can be detected and analyzed for mutations that are unique to the tumor.

Clinical applications include early detection of tumors before they become clinically evident, early detection of recurrent tumor after treatment and detection of clonal tumors different from the one that may have been biopsied. There will also be the ability to monitor tumors that are difficult to access or monitor. And, finally, it will be possible to monitor and adjust chronic cancer therapy as new mutations and resistance to treatment emerges.

This is good news for all of us as we can expect improved survival in cases of cancer. But this is not necessarily good news for risk classification. In the effort to safeguard their citizens from "genetic discrimination," some countries have prohibited the use of genetic information or information about mutations to DNA or RNA. They have not necessarily made a distinction for tumor-derived RNA or DNA. This leaves the door open for an imbalance of material information that could potentially damage an insurers' ability to meet its obligations.

Continued, back cover

SCOR
The Art & Science of Risk

CASE #1

An Unbiopsied Pigmented Skin Lesion

By Bill Rooney, MD
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A 39-year-old female is applying for $1 million of life insurance. Records reveal this otherwise healthy woman was recently evaluated for a pigmented lesion on her face. This lesion had been present for years, but she had scratched it recently and it bled slightly. While it seemed to heal normally and there was no appreciable difference in the appearance once healed, she consulted with her dermatologist for further evaluation. She was concerned about the possibility of a malignant melanoma. Her best friend who had been recently diagnosed with a melanoma had encouraged her to seek an opinion.

There was no family history of skin cancer. The dermatologist documented the lesion to be 4-mm in size with uniform color, border and symmetry. A dermascope evaluation was normal. The dermatologist recommended a biopsy to ensure it was benign. The patient refused as she was concerned about the potential scar on her face. Therefore, the dermatologist ordered another test called a Pigmented Lesion Assay (PLA), which was negative. Follow-up was recommended in three months. Records reveal the three-month follow-up was normal and follow-up was now planned in one year.

What are the mortality considerations for someone with a pigmented lesion such as this who has had a biopsy recommended but declined? Should this request be postponed pending favorable review of a biopsy pathology report?

There are several forms of common skin cancer including basal cell carcinoma, squamous cell carcinoma and malignant melanoma. Malignant Melanoma (MM) has the greatest mortality risk.

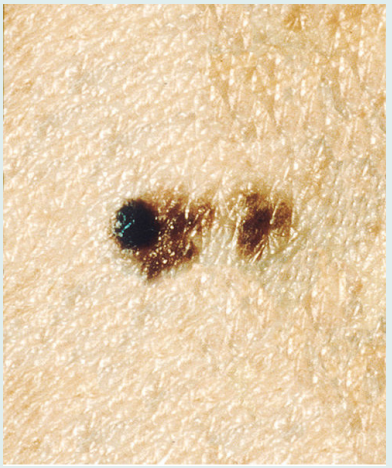
MM will be diagnosed in approximately 96,000 people annually and will result in more than 7,000 deaths. It is the fifth-most common cancer in men and women. The incidence has been trending upwards during the past few decades. It is important to detect MM early as survival is significantly improved.

The American Joint Committee on Cancer (AJCC) 8th edition describes five stages of melanoma classification:

- Stage 0 is in situ.
- Stages I and II involve invasive but localized disease.
- Stage III involves the regional nodes.
- Stage IV involves distant metastasis.

The five-year relative survival rate for localized disease is 98%, for regional involvement it is 64%, and for distant disease it drops to 23%.

Figure 1 - A malignant melanoma (not the lesion described in this article)



Source: https://commons.wikimedia.org/wiki/File:Melanoma.jpg
Image obtained using Google's "labeled for reuse with modification" search engine

Risk factors

Risk factors for developing MM include sun exposure, indoor tanning, the presence of dysplastic nevi or atypical moles, many moles, congenital melanocytic nevi, fair skin, family history, previous skin cancer, prior radiation treatment and/or an immunosuppressed system. Approximately 10% of all people with melanoma have a family history of melanomas.

Malignant melanomas can occur anywhere on the skin and can also occur in the eye. The malignant melanomas of the skin are most commonly detected by the patient themselves. Unfortunately, MM can be difficult to diagnose based on visual inspection.

Diagnostic techniques

There are several different clues to the presence of MM, and there are several different analysis techniques.

The first method to evaluate the skin for malignant melanomas is visual inspection. Features examined include the color, size, regularity of the borders and symmetry of the lesion.

Finally, the lesion is evaluated for change in any of these characteristics, either by good history or by serial examination. Worrisome features could include any of the following:

- Asymmetry
- Borders that are irregular
- Color that is variegated (multiple shades of red, black, gray or other colors within the same mole)
- Diameter >6mm
- Evolution of the lesion over time (a new mole or a change in the appearance or size)

Notice these five characteristics establish the rule of melanoma known as "the ABCDE rule".

Next the lesion is compared to other similar pigmented lesions. On most individuals, moles tend to look like each other. When a pigmented lesion looks different from the other lesions on that individual, this has been called "the ugly duckling sign." Even if the ABCDE rule criteria has not been met, the presence of an unusual appearing mole raises the concern that this lesion could be a malignant melanoma.

After visual inspection many suspicious pigmented lesions are evaluated by use of a dermoscope. This device requires special training to be used properly, but use of the device improves not only the sensitivity but also the specificity of the diagnosis of melanoma.

Visual inspection alone has been documented to be 71% sensitive and 80-90% specific. Adding the dermoscopic component to the evaluation increases this to 90% sensitive with a similar degree of specificity.

Another diagnostic aid used for further evaluation of suspicious skin lesions is the reflectance confocal microscopy. This device uses a laser to emit near-infrared light that evaluates the epidermis and is felt to increase both the sensitivity and specificity of the diagnosis of skin cancers. Unfortunately, this technique requires additional training, is more time consuming, more expensive and is not always readily available.

Continued

CASE #1

Unbiopsied Pigmented Skin Lesion

Continued, page 3

Figure 2 - A dermascope

Source: https://fr.wikipedia.org/wiki/Fichier:Dermatoscope.jpg Image obtained using Google's "labeled for reuse with modification" search engine.

Typically, when a lesion is suspected of being a malignant melanoma after one or more of the preceding tests, the next step is to biopsy the lesion for histological evaluation. Even though the biopsy is considered the "gold standard," the sensitivity and specificity of this procedure is not perfect.

Several studies have documented a significant degree of variability in the diagnosis among even experienced and well-trained pathologists. One study evaluated this variability and found that only 82.8% of cases were confirmed when compared to a follow-up evaluation by three dermatopathologists.

FIGURE 3 - TESTING OPTIONS FOR PIGMENTED LESIONS

	Sensitivity	Specificity
Visual inspection	71%	80% - 90%
Dermascope	90%	80% - 90%
Reflectance confocal microscopy	86% - 100%	72% - 91%
Pigmented lesion assay	91%	69%
Biopsy	The Gold Standard but the results are not always agreed upon when secondarily reviewed	

New tools

Recently, several other diagnostic aids for the evaluation of pigmented skin lesions have been introduced. These include use of analysis of autoantibody biomarkers, comparative genomic hybridization and tumor gene expression evaluation.

For pigmented lesions found in areas with significant cosmetic concerns or in patients who are anti-coagulated, making biopsy less desirable, a recent development has been the use of an adhesive skin collection kit. This consists of adhesive strips that are applied to the suspicious lesion, removed and then submitted to the lab for further analysis.

This noninvasive test uses gene expression profiling to analyze two genes (LINC and PRAME) involved in melanoma. This pigmented lesion assay (PLA) test is reported to be 91% sensitive and 69% specific. It has been reported to have a negative predictive value of 99%.

Another test named Nevome has recently been introduced to further evaluate the genetic material removed by the adhesive patches. DNA mutation analysis is performed analyzing the BRAF, NRAS and TERT promoter genes. This test, when combined with the PLA test, is reported to have a sensitivity of 97% and negative predictive value of >99%.

Returning to the case

In this case the skin lesion is not described as having any changes using the ABCDE rule and there is no mention of it being "an ugly duckling." In addition, a dermascope evaluation test was reassuring.

It isn't completely clear why a biopsy was recommended, but when this was refused for cosmetic reasons, a PLA test was performed and was negative. While none of these tests are 100% sensitive or specific, the tests that have been done are very reassuring.

Finally, the individual is compliant with follow-up evaluations. The risk of this lesion being a melanoma is extremely low. No increased mortality risk is expected. No postponing is needed.

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CASE #2

Coarctation of the Aorta

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A 38-year-old female applied for life insurance. At age 28 she underwent successful stenting for an aortic coarctation. A regular follow-up by transthoracic echocardiography four and six years after stenting revealed a small intrastent gradient of 14 mm Hg. But very recently this gradient increased to 26 mm Hg, then two months later to 30 mm Hg. There was no associated cardiac defect and no left ventricular hypertrophy. The left ventricular ejection fraction was normal at 60% with a pulmonary artery systolic pressure in normal range as well. She remains asymptomatic, her BP is normal at 110/78 mm Hg without treatment. The BMI is 22.3, and labs are within normal limits.

What is coarctation of the aorta and what are the mortality implications?

Coarctation of the aorta (CoA) is a congenital cardiac defect leading to a narrowing of the descending aorta, which is typically located at the insertion of the ductus arteriosus just distal to the left subclavian artery at the level of the aortic isthmus. However, it is more likely to represent a spectrum of aortic narrowing from a discrete entity to tubular hypoplasia, with many variations seen between these two extremes.

Etiology

The etiology of CoA remains unclear. The primary mechanism is thought to be medial thickening and intimal hyperplasia that leads to the formation of a posterolateral ridge that encircles the aortic lumen, resulting in the CoA segment.

Another theory is that of aberrant ductal tissue residing in the wall of the fetal thoracic aorta and abnormal intrauterine blood flow through the aortic arch. A genetic predisposition is also suggested by reports of CoA occurring in family members and by its association with Turner syndrome.

Pathophysiology

CoA does not cause a hemodynamic problem in utero, as two-thirds of the combined cardiac output flows through the patent ductus arteriosus (PDA) into the descending thoracic aorta, bypassing the site of constriction at the isthmus.

During the neonatal period, when the PDA and foramen ovale (between the right and left atria) begin to close, the bloodflow that must cross the narrowed aortic segment to reach the lower extremities steadily increases. As this occurs the hemodynamic changes may range from mild systolic hypertension to severe heart failure depending upon the severity of the coarctation and the presence of other associated lesions.

At birth, the left ventricular afterload increases because of outflow tract obstruction resulting in increased systolic pressure in the left ventricle and proximal aorta.

Several compensatory mechanisms arise to surmount the left ventricular outflow tract obstruction. These include left ventricular myocardial hypertrophy, which maintains normal systolic function and ejection fraction, and the development of collateral blood flow involving the intercostal, internal mammary and scapular vessels, which circumvent the stenotic lesion (Figure 1).

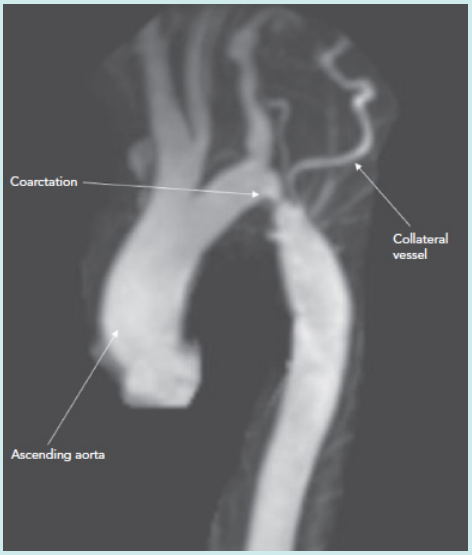

Figure 1 - Cardiac MRI of a 53-year-old patient with severe coarctation and evidence of collateral flow.

Source: Marc G Cribbs Coarctation: a review www.USCjournal.com

Although CoA can be an isolated congenital heart disease, it is commonly found in other congenital syndromes and cardiovascular anomalies. The most common cardiovascular malformation associated with CoA is bicuspid aortic valve (BAV), occurring in more than 50% of cases. Other defects like atrial septal defect, ventricular septal defect, atrioventricular canal defect, aortic stenosis, transposition of the great arteries, patent ductus arteriosus and hypoplastic left heart syndrome occur less frequently.

The most important non-cardiac lesion is cerebral aneurysm, which is present in up to 10% of patients.

Epidemiology

CoA is the fifth-most common congenital heart defect, accounting for 6% to 8% of live births with congenital heart disease. The estimated incidence is 1 in 2500 births, occurring more commonly in males than females (59% versus 41%).

Clinical presentation

The clinical presentation and exam findings are variable based on patient's age. Typically, earlier presentation corresponds to severe disease.

Younger children: Newborns and neonates are usually asymptomatic right after birth as the PDA helps perfuse the lower body irrespective of severity of CoA. Neonates with severe/critical CoA develop signs and symptoms of cardiogenic shock as the ductus arteriosus closes after birth. Clinically, babies may show absent/feeble femoral pulse, delayed capillary refill, feeding problems, decreased responsiveness, metabolic acidosis, mesenteric ischemia, myocardial depression, etc.

Older pediatric patients are usually diagnosed due to weak femoral pulse, upper extremity hypertension, a systolic murmur over upper sternal border with radiation to the back and upper-lower extremity systolic blood pressure gradient. Newborn pulse oximetry screening is a great tool in detecting cases of critical congenital heart disease in newborns, although its utility is limited in patients with pure CoA and a closed PDA due to lack of blood mixing/shunting.

Adolescents and adults: Almost always these patients are diagnosed with CoA during workup of systemic hypertension or heart murmur. Clinical signs may include upper extremity hypertension, weak femoral pulse, arm-leg systolic blood pressure gradient (>20 mmHg is significant), a systolic murmur on the back from flow through the coarctation segment or a continuous murmur from the collateral flow around the coarctation site. Intercostal pulses from collateral vessels are a pathognomonic sign.

Patients may also complain of frequent headaches from systemic hypertension and lower limb claudication from chronic hypoperfusion. If the collateral circulation around the coarctation site is significant then distal pulses may be adequate and arm-leg blood pressure gradient may not be significant.

Continued

CASE #2

Coarctation of the Aorta

Continued, page 7

Diagnostic evaluation

EKG may be normal or demonstrate evidence of left ventricular hypertrophy from chronic left ventricular pressure overload.

Chest X-ray in adults may show a normal cardiac contour. Double contouring of the descending aorta known as the "3 sign" beneath the aortic notch is characteristic and represents narrowing of the aorta at the level of CoA and dilatation of the aorta pre- and post-CoA. Rib notching of the posterior fourth to eighth ribs caused by the dilated intercostal collateral arteries can be visible in patients with longstanding severe CoA.

Fetal echocardiography has advanced significantly during the past couple of decades to allow us to make prenatal diagnosis of CoA and avoid a cardiovascular catastrophe after birth.

Transthoracic echocardiography (TTE) is the primary imaging modality for suspected CoA. It has the capacity to provide us with diagnosis, to assess severity and to detect associated cardiac defects.

Cardiac magnetic resonance imaging (cMRI) or *computed tomography angiography* (CTA) clearly defines the location and severity of CoA as well as collateral vessels.

cMRI is a preferred non-invasive, advanced imaging modality for patients with CoA since it does not include any exposure to ionizing radiation but provides excellent image resolution. This makes it ideal for initial imaging and serial follow-ups.

Furthermore, cMRI angiography with gadolinium-enhanced contrast provides excellent visualization of extracardiac vasculature and allows for optimal three-dimensional reconstruction as needed. Utilization of phase contrast flow analysis helps to assess peak gradient across the coarctation site. If cMRI is contraindicated, CTA imaging may be used to diagnose CoA.

Catheter angiography is currently limited to coronary angiography before intervention/surgery and when catheter-based intervention such as balloon dilatation and/or stenting is considered.

Natural history

We know from postmortem reports prior to the availability of operative repair that average survival age of individuals with unoperated CoA was approximately 35 years of age, with 75 percent mortality by 46 years of age.

Approximately one quarter will die from aortic dissection or rupture, one quarter will die from heart failure, one quarter will die from intracranial hemorrhage, and the remainder will die from other complications.

Treatment

Indications for intervention: According to the most recent recommendations for the management of CoA issued by the American Heart Association/American College of Cardiology and the European Society of Cardiology, corrective intervention should be performed in patients with CoA with any of the following:

- Critical CoA
- CoA gradient >20 mm Hg
- Radiologic evidence of clinically significant collateral flow
- Systemic hypertension attributable to CoA
- Heart failure attributable to CoA

These guidelines stress that correction of coarctation should be performed as early as possible (optimally early in childhood) to reduce long-term morbidity, to prevent the development of chronic systemic hypertension and to improve survival.

Treatment options: Surgical repair, transcatheter balloon angioplasty (BA) and transcatheter stent implantation are treatment modalities available for management of CoA. The preferred treatment option depends on the anatomy of the coarctation, the age of the patient, the size of the patient and other comorbidities.

Surgical options, depending on the lesion, include subclavian artery patch aortoplasty, patch aortoplasty, bypass of the coarctation, tube graft replacement, aneurysm replacement, two-stage combined bicuspid valve surgery and arch and descending aorta replacement or ascending aorta–to–descending aorta bypass. Endovascular balloon dilatation and stent placement has been used successfully and is becoming a less invasive alternative to conventional open surgical procedures except for neonates and infants less than four months old.

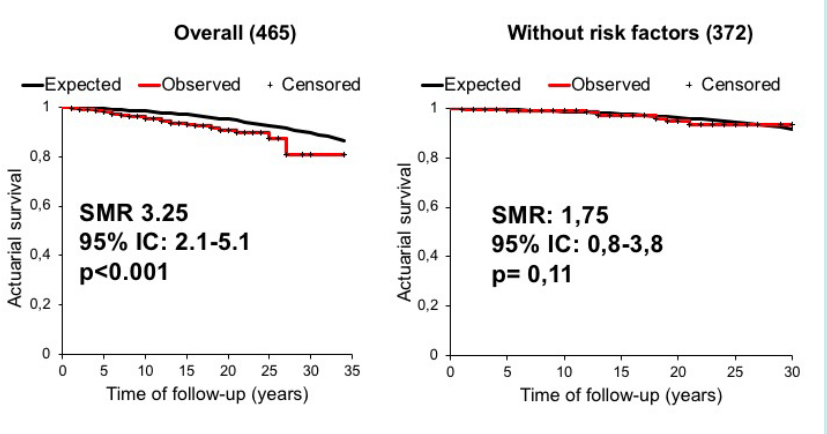

Figure 2 - Age at diagnosis-, sex-, and time of follow-up-adjusted Standardized Mortality Ratio (SMR) in adults with aortic coarctation

Source: Sanchez Recalde A et al Risk factors for excess mortality in adults with coarctation of the aorta; https://academic.oup.com › eurheartj › article › suppl_1 › ehy565.2113

All three treatment modalities show significant improvement in systolic blood pressure and peak pressure gradient. The rate of acute complications is lower after stent implantation compared to BA or surgery. However, planned re-intervention is more likely in stented patients. Stent implantation and surgery achieve superior hemodynamic results over BA.

Long-term cardiovascular complications

Major long-term complications following CoA repair include recoarctation (restenosis after an initially successful dilatation or operative repair), aortic aneurysm, systemic hypertension and intracranial aneurysms.

Recoarctation rate is approximately:

- 5% to 15% after surgery
- Up to 50% in neonates and young infants, 20% to 30% in older children and 8% in adolescents and adults after balloon angioplasty
- After stenting intermediate outcomes are promising.

Catheter-based intervention is preferred over surgical repair of recoarctation because mortality for reoperation is higher than for primary repair.

Aortic aneurysm may develop at the site of prior coarctation following surgery, BA or stent implantation. Its incidence after surgical repair or BA ranges from less than one percent to nine percent and is generally treated surgically.

Systemic hypertension is more common in patients whose repair was performed after 20 years of age compared with those who were corrected in early childhood. The risk of hypertension increases over time in all patients and, associated with left ventricular hypertrophy, is among the factors that contribute to premature death from coronary and cerebrovascular disease in patients with repaired CoA. Thus, it is paramount to control hypertension with medical treatment.

Intracranial aneurysms are associated with CoA in up to 10% of patients. This fact may justify routine screening for cerebrovascular aneurysms.

Whether the patient has undergone a surgical or transcatheter treatment, lifelong follow-up care is required to detect long-term complications.

Prognosis

The estimated 10-year survival following CoA repair is greater than 90%.

In a single-center series of 819 patients (mean age at repair 17.2±13.6 years) who underwent surgical repair of isolated CoA at the Mayo Clinic between 1946 and 2005, the survival rates at 10, 20 and 30 years after primary repair were 93%, 86% and 74%, respectively.

In an earlier report from the same center, the most common cause of late death was coronary artery disease, followed by sudden death, heart failure, cerebrovascular accident and ruptured aortic aneurysm.

Data on long-term prognosis after transcatheter intervention are more limited. In a report of intermediate outcomes in patients enrolled in the Coarctation of the Aorta Stent Trial (COAST)-I trial, five-year freedom from reintervention was 75 percent.

Factors associated with decreased survival include older age at initial repair (i.e., older than 20 years) and preoperative hypertension.

Continued

CASE #2

Coarctation of the Aorta

Continued, page 9

After reintervention for restenosis, aneurysm or pseudoaneurysm, ML Brown et. al. showed that survival was 97% at five and 10 years respectively, and there was no difference in survival between operative and endovascular patients at 10 years (99% versus 91%, respectively).

Recently, A Sanchez Recalde et. al., in a retrospective study in Spain, found that the standardized mortality ratio (SMR) in the cohort of patients with aortic coarctation was 3.25 (Figure 2). The main independent predictors of death in this multivariate analysis were: left ventricular dysfunction, severe pulmonary arterial hypertension, and aortic wall aneurysms. SMR was 1.75 in patients with no risk factors, similar to the reference population.

Returning to the case

First, this young woman underwent the repair of her CoA at 28 years old, which is usually not a favorable predictor of survival, but fortunately she is asymptomatic. And more importantly, she has no hypertension, no heart failure, no aortic aneurysm and no arterial pulmonary hypertension.

However, the peak pressure gradient through the stent has steadily increased over the years, reaching 30 mm Hg on the latest transthoracic echocardiograpy. This is suspicious for a recoarctation which may require a reintervention. It appears prudent to delay any offer pending resolution of the increasing gradient.

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SOLVE

EKG Puzzler

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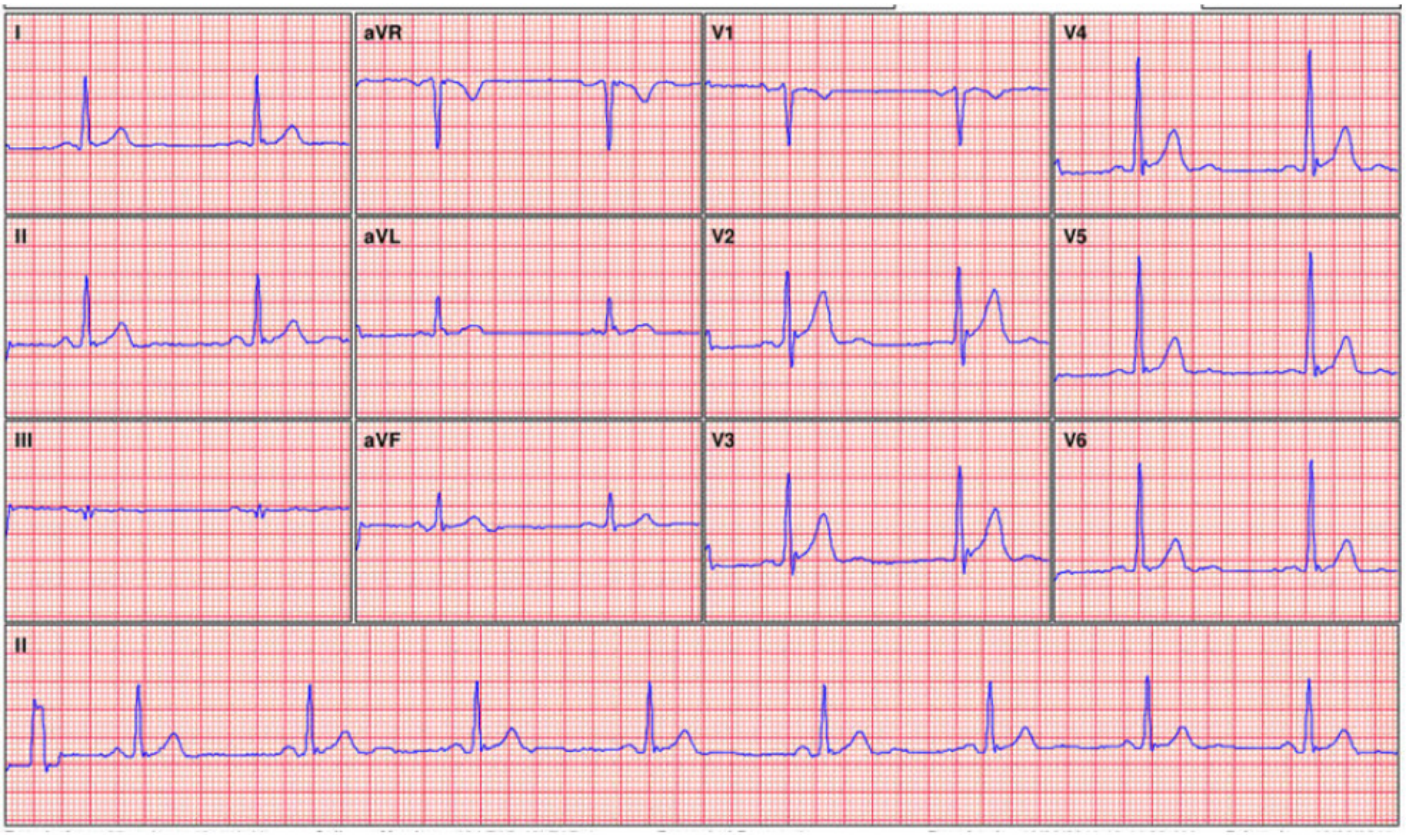
Here is the latest ECG Puzzler to solve.

A 48-year-old manager applies for $3 million of life insurance. His BMI is 27. Blood pressure, lipids and LFTs are normal. There is no significant family history. This EKG was provided. What do you see?

Visit the *Housecalls* page on our website (www.scorgloballifeamericas.com) to find the answer. Click on Winter 2019 Puzzler to confirm your findings.



GENETIC TESTING

The Nuances Matter

Continued, page 1

What to do? Perhaps it is time to revisit those blanket prohibitions on access to genetic information. Certainly every effort should be made to make regulators and legislators understand the nuanced difference between tumor-derived DNA or RNA and somatic genetic material.

In this issue Dr. Rooney discusses a case with an unbiopsied skin lesion. Dr. Kadouch examines the implications of coarctation of the aorta, and Dr. Rosace has an interesting EKG for our puzzler.

Here's hoping that you and yours have a very happy holiday and a prosperous new year.

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