significantly associated with poor OS (p=.05 and < .001) and RFS (p = .009 and <.001) (Figure 1). In addition, patients with grade III tumors were more likely to have lower OS and RFS rate than patients who had grade I or II tumors; although the differences were not statistically significant (p = 0.31 for OS and p = 0.79 for RFS) due to limited sample size. MYB-NFIB gene fusion was identified in 3 of 8 cases for which FISH study was performed.

Conclusions: Not all mammary ACCs have good prognosis. Old age, nodal metastasis at presentation and high histologic grade are poor prognostic indicators in this rare type of breast cancers.

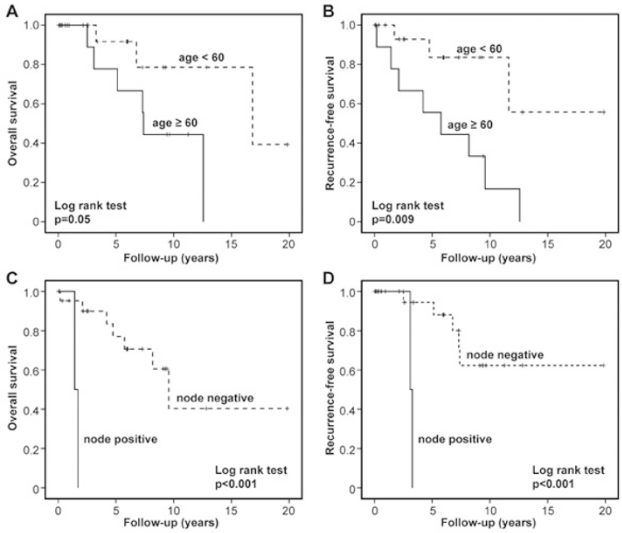
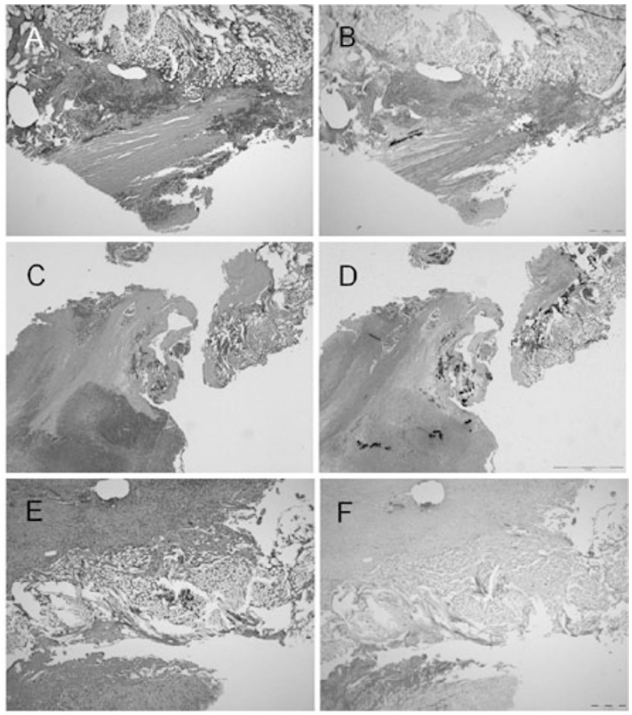


Figure 1. Older age (≥ 60 years) and the presence of regional node metastasis at presentation were significantly associated with poor OS and RFS in breast ACC.



Representative images of aortic specimens and adjacent graft material. **(A, B)** Images from a subject in Group A, Pig 2, showing small cluster of Gram (+) cocci near the graft implantation site. **(C, D)** Images from a subject in Group B, Pig 4, showing multiple clusters of Gram (+) cocci *(arrows)* and abscess formation *(asterisk)*. **(E, F)** Images from a subject in Group C, Pig 6, negative Gram staining.

Cardiovascular

310 A Novel Dual Antibiotic-Bonded Graft for Preventing Vascular Aortic Infection

I Aboshady, A Shah, D Vela, T Dvorak, I Raad, KG Khalil, LM Buja. The Texas Heart Institute, Houston, TX; The University of Texas HSC, Houston, TX; M.D. Anderson Cancer Center, Houston, TX; Baylor College of Medicine, Houston, TX.

Background: Perioperative infection of an aortic graft is associated with a mortality rate of 10%-30% and an amputation rate >25%. In vitro studies suggest that an antibiotic-impregnated graft could help prevent perioperative graft infection. In a pilot animal study, we bonded aortic grafts with 2 different antibiotics and evaluated their ability to prevent direct perioperative bacterial contamination.

Design: We surgically implanted a 6-mm Vascular Dacron graft in the infrarenal abdominal aorta of 6 Sinclair miniature pigs. Two pigs received grafts bonded with 60 mg/mL solutions of rifampin and minocycline; the other 4 pigs received unbonded grafts. Before implantation, both bonded grafts and 2 of the 4 unbonded grafts were immersed for 15 minutes in a 2-mL solution containing 1 to 2×10^7 colony-forming units (CFUs)/mL of *Staphylococcus aureus* (ATCC 29213). Two weeks after graft implantation, the pigs were euthanized, and the grafts were excised for clinical, microbiologic, and histopathologic study.

Results: The 2 *S. aureus*–treated bonded grafts showed no bacterial growth upon explantation, whereas the 2 *S. aureus*–treated unbonded grafts had high bacterial counts (6.25×10^6 and 1.38×10^7 CFU/graft). The 2 unbonded and untreated grafts had bacterial growth (1.8×10^3 and 7.27×10^3 CFU/graft) that presumably reflected accidental perioperative bacterial contamination; *Staphylococcus cohnii ssp urealyticus* and *Staphylococcus chromogenes,* but not *S. aureus,* were isolated. The histopathologic and clinical data confirmed the microbiologic findings. Only pigs that received unbonded grafts had histopathologic evidence of a perigraft abscess.

Conclusions: Bonding aortic grafts with 2 antibiotics appears to be a promising method of reducing direct perioperative bacterial contamination. Further studies are needed to explore this novel graft's ability to combat one of the most feared complications in vascular surgery.

311 Flat-Panel Computed Tomography for Longitudinal Assessment of Atherosclerotic Plaque Components: Quantitative Correlations with Pathologic Measurements

I Aboshady, DD Cody, EM Johnson, D Vela, KG Khalil, GW Gladish, LM Bula. The Texas Heart Institute, Houston, TX; M.D. Anderson Cancer Center, Houston, TX; The University of Texas HSC, Houston, TX; Baylor College of Medicine, Houston, TX.

Background: Flat-panel computed tomography (FpCT) provides better spatial resolution than 64-channel CT and better assesses atherosclerotic plaque components *in vivo* in animal aortas similar in size to human coronary arteries. We assessed the usefulness of FpCT in longitudinal studies of plaque development.

Design: We used a prototype FpCT scanner with a dual-panel rotating gantry and a commercial Performix CT x-ray source. 184 aortic histology sections from 6 Watanabe heritable hyperlipidemic rabbits were quantitatively compared with 64-CT (image thickness, 0.625 mm) and FpCT (image thickness, 0.150 mm) images. Images were reoriented perpendicular to the vessel centerline.

Results: Although FpCT was more sensitive in detecting eccentric lesions (42% vs 0%; P=0.000), the area under the curve (AUC) for FpCT (0.6) did not significantly differ from that for 64-CT (0.45; P=NS). In detecting plaques with ≤10% lipid (low-attenuation foci), FpCT was more sensitive than 64-CT (24% vs 0.7%; P<0.00) and had a greater AUC (0.6 vs 0.5; P<0.006). Additionally, FpCT was more sensitive (65% vs 0%; P<0.00) in detecting plaques with ≤5% calcium (high-attenuation foci) but not in detecting branch points. Both FpCT and histology could detect low-attenuation foci as small as 0.3 mm in diameter, whereas 64-CT could detect only low-attenuation foci ≥1.5 mm in diameter. In the current, long-term phase of the study, 30 New Zealand White hyperlipidemic rabbits receive a high-fat diet (0.5% cholesterol). Lesions are monitored and correlated through monthly serial scanning sessions over 6 months. Images are collected 30 seconds after Visipaque injection (560 mgI/kg; through an ear vein).

Conclusions: FpCT seems to have more potential in quantitative screening for low-risk small atherosclerotic lesions, whereas 64-CT is limited to imaging established, well-characterized lesions, particularly when measuring the vascular wall thickness in a rabbit model of atherosclerosis. FpCT seems to have potential for quantitatively monitoring the evolution of the calcific and lipid components of plaque.

312 Surgical Pathology of Native Valve Endocarditis in 310 Specimens from 287 Patients (1985-2004)

MC Castonguay, KD Burner, WD Edwards, LM Baddour, JJ Maleszewski. Mayo Clinic, Rochester, MN.

Background: Few large studies have separately documented the clinical and pathologic features of native valve endocarditis from those of prosthetic valve endocarditis. Furthermore, surgical management of valvular endocarditis has evolved considerably in the past 20 years.

Design: A retrospective study of medical records from all patients undergoing surgery for native valve endocarditis at our institution between 1985 and 2004. Medical records were reviewed from 287 patients for demographics, infected native valve(s), infecting organism, risk factors for endocarditis, and pathologic features. Because 22 patients

underwent removal of two or more affected valves, the study group included 310 valves. Histologically, slides were evaluated for the infecting organism(s) with tissue Gram and GMS stains.

Results: Patients ranged in age from 9-87 years (mean, 54). Of the 310 valves, 73% were from men and 84% were regurgitant. Risk factors for endocarditis included bicuspid aortic valve (23%), dental disorders (20%), mitral valve prolapse (17%), diabetes mellitus (16%), and others (< 5%, each); in 15%, no risk factor was identified. The four most commonly identified organisms were viridans streptococci (28%), *Staphylococcus aureus* (18%), coagulase-negative *Staphylococcus* (8%), and group D streptococci (10%). Native valve endocarditis was histologically active in 58% and healed in 42%, and affected the aortic position in 55%, mitral in 39%, and right-sided valves in 6%. It was associated with embolization in 29%, acute heart failure in 29%, and annular abscess in 20%. There were significantly more men in the aortic valve endocarditis cohort than in the mitral valve endocarditis cohort (81% vs. 63%, respectively; p=0.001). Among 182 valves with active endocarditis, 24.2% had no micro-organisms identified histologically, although purulent inflammation and focal cusp or leaflet destruction (along with positive blood cultures) were sufficient to establish a diagnosis of active endocarditis.

Conclusions: Native valve endocarditis affected men nearly three times as frequently as it did women. Male preponderance was 4:1 for the aortic valve but only 2:1 for the mitral valve. Diabetes mellitus emerged as a prevalent (and previously unrecognized) risk factor for infection of native valves. The most common infecting organisms were streptococci (45%) and staphylococci (26%). Micro-organisms were identified histologically in the majority of active endocarditis cases.

313 Surgical Pathology of Aortic Aneurysms Associated with Non-Infectious Inflammation (1994-2011): The Role of IgG4

MC Castonguay, JE Rocha, WD Edwards, JJ Maleszewski. Mayo Clinic, Rochester, MN.

Background: The increasingly recognized roleS that IgG4-related sclerosing diseases (IgG4-RSD) play in the development of aortic aneurysms has raised many questions regarding our understanding and classification of both thoracic and abdominal aortic aneurysms. Recently, investigators have suggested that a significant proportion of so-called "inflammatory aortic aneurysms" are associated with increased IgG4-reactive plasma cells, though definitive criteria have yet to be established.

Design: A retrospective clinicopathologic review was undertaken of surgically resected aortic aneurysms associated with varying degrees inflammation. The study groups included 42 cases. Clinical records were reviewed to document the presence of traditional risk factors associated with aortic aneurysms and for the presence of other IgG4-RSD. Archived H&E- and Verhoeff-van Gieson-stained slides were reviewed to evaluate the type of aneurysm and the location and composition of the inflammatory infiltrate. Immunohistochemical studies evaluating the density and proportion of plasma cells expressing IgG4 (as a proportion of plasma cells expressing IgG) were undertaken.

Results: The forty-two patients ranged in age from 19 to 84 years. Thirty were men. Aneurysms were classified as "inflammatory aneurysms" (27 cases) and included inflammatory abdominal aortic aneurysms (AAA) and 1 inflammatory thoracic aortic aneurysm, and others, included atherosclerotic aneurysms with inflammation insufficient to qualify as inflammatory AAA (6 cases), those associated with aortitis (6 cases), and those associated with medial degeneration (3 cases). Age, sex, and traditional risk factors for atherosclerotic aneurysms did not differ between groups. Adventitial inflammation occurred more frequently in inflammatory aneurysms ($p<0.05$), but the proportion and density of plasma cells expressing IgG4 did not vary significantly. Additional features, such as reactive lymphoid follicles, phlebitis, and medial inflammation, did not differ between groups. 16 aneurysms had >100 IgG-positive plasma cells per high-power field; 11 of these were "inflammatory aneurysms." The average IgG4 proportion did not differ between these two groups (30.86% vs. 48.51%, respectively; p=0.16).

Conclusions: Our data does not support the contention that inflammatory aneurysms are more frequently associated with an increased proportion of IgG4-expressing plasma cells than other aneurysms with inflammation. In fact, the proportion of IgG4-positive plasma cells was less in inflammatory aneurysms, although this did not reach statistical significance.

314 Surgical Pathology of Atrial Appendages Removed during the Cox-Maze Procedure: A Review of 86 Cases (2002-2005)

MC Castonguay, Y Wang, JL Gerhart, WD Edwards, JJ Maleszewski. Mayo Clinic, Rochester, MN.

Background: Atrial fibrillation (AF) is a common cardiac arrhythmia that can complicate several forms of chronic heart disease. Persistent AF can be treated surgically with the Cox-maze procedure, which includes removal of one or both atrial appendages. To date, no large study has described the microscopic features of atrial appendages removed during the Cox-maze procedure.

Design: Atrial appendages, removed during the Cox-maze procedure from 86 consecutive patients with AF, were examined by microscopy and compared to atrial appendages procured at autopsy from 26 age-matched individuals without AF. Twenty additional control atria from individuals with heart disease but no AF were also obtained from our autopsy practice. Routine formalin-fixed, paraffin-embedded, H&E-stained sections were examined. Cases were also evaluated for amyloid with Congo red staining. The presence of moderate to severe myocyte hypertrophy, myocyte vacuolization, and interstitial fibrosis were noted. Additionally, any fatty infiltration, endocardial fibroelastosis, lymphocytic inflammation, thrombosis, and amyloid deposition was recorded. χ^2 analysis was utilized to assess differences between the cohorts.

Results: Moderate to severe myocyte hypertrophy was noted more frequently in individuals with AF than in either control cohort; however, the difference was statistically significant only when comparing the AF group to the normal control ($p<.02$). Myocyte vacuolization was noted more frequently in individuals with AF than those without ($p<.043$). Like myocyte hypertrophy, interstitial inflammation was more frequent in the AF cohort than either control, but only exhibited statistical significance when compared to the normal cohort ($p<.004$). Fatty infiltration was seen significantly more often in those with AF ($p<.007$). Interstitial fibrosis was noted more frequently in diseased hearts, while no statistically significant difference was observed between the groups with respect to amyloid deposition. Endocardial fibroelastosis and thrombosis were not seen with increased frequency in the AF cohort compared to either of the controls.

Conclusions: This is the largest study to date that quantifies the histopathologic changes seen in atrial appendages removed during the Cox-maze procedure. Myocyte hypertrophy, myocyte vacuolization, interstitial lymphocytic inflammation and fatty infiltration occur more frequently in atria with AF than those without. Interstitial fibrosis is frequently encountered in diseased hearts (including AF).

315 High Correlation between Molecular Sequencing and Histopathological Examination of Parallel Samples in Culture-Negative Endocarditis

AMB Collie, NK Shrestha, GS Hall, ER Rodriguez, CD Tan. Cleveland Clinic, Cleveland, OH.

Background: Microbiological cultures of blood and excised valves are often negative in patients undergoing surgical interventions. Thus, molecular sequencing of cardiac valve specimens is increasingly used to identify the specific organism(s) involved in culture-negative endocarditis. The present study examines molecular sequencing and histopathological results on parallel cardiac valve specimens and identifies histopathologic features common to positive specimens.

Design: All cardiac valve specimens with molecular PCR sequencing results were identified at our institution. For cases where a surgical specimen was available, the histologic features of acute inflammation, necrosis, chronic inflammation, granulation tissue, and fibrinous vegetations were analyzed. Organisms were identified with special stains including Gram, Gomori methenamine silver, and periodic acid-Schiff. PCR sequencing results, including bacterial 16S rRNA, acid fast bacterial heat shock protein 65, and fungal 28S rRNA, were obtained. Molecular results and histopathologic characteristics were compared.

Results: One hundred eleven cardiac valves from 100 patients had PCR results and histologic examination of a parallel surgical specimen. Seventy eight valves (70%) had agreement in the presence or absence of organisms in both studies (Table 1). Three valves (3%) had organisms identified histologically that were not identified by PCR. Thirty valves (27%) did not have an organism identified histologically but did have an organism identified by PCR. When organisms were not identified histologically, acute inflammation was more common in valves with positive PCR (50%) compared with negative PCR (25%) ($p<0.001$), and fibrinous vegetations were more common in valves with positive PCR (23%) compared with negative PCR (9%) ($p<0.01$). Necrosis was only observed in PCR positive cases.

Table 1. PCR Results and Histological Examination in 111 Cardiac Valves

Histological examination (diagnosis)	PCR positive	PCR negative
Organisms identified	44	3
Organisms not identified	30	34

Conclusions: Molecular sequencing of cardiac valve samples in culture-negative endocarditis is valuable in diagnosis. The current study could be complicated by sampling error, in that parallel rather than exact specimens were analyzed. However, there is high correlation between PCR sequencing and histological examination. Both studies provide complementary and valuable results.

316 Cardiac Allograft Outcomes: A Retrospective Study Correlating DSA, Endomyocardial Biopsy and Immunofluorescence

R Frank, G Wertheim, M Kamoun, P Lal. Hospital of the University of Pennsylvania, Philadelphia; Children's Hospital of Philadelphia, Philadelphia.

Background: Donor specific antibodies (DSAs) are clinically significant and have been associated with cardiac graft loss and a predisposition to coronary vasculopathy. Antibody-mediated rejection is a recognized clinicopathologic entity by the International Society for Heart and Lung Transplantation (ISHLT). Detection relies, in part, on the immunofluorescence (IF) pattern of C4d, serologic findings, and clinical picture.

Design: We performed a retrospective analysis of the relationship between DSA detection, IF, and clinical outcomes in cardiac transplant patients at our institution (2005-2011). DSA were evaluated against HLA class I and class II specificities pre and post transplant using flow cytometry and/or Luminex bead assays. Acute antibody-mediated rejection (AMR) was based on the ISHLT 2006 report, including diffuse interstitial capillary C4d and DSA presence. In addition to DSA and IF, we examined graft failure (mortality or graft replacement) as well as graft dysfunction as assessed by precipitous decrease in ejection fraction (EF) of at least 20%.

Results: 330 RVC biopsies and concurrent IF studies from 110 cardiac grafts in 109 patients, who had pre and post transplant DSA measurements, were analyzed. Of these, 50 grafts had DSA against either HLA class I, class II, or both. In patients with significant graft dysfunction (44), 78 of 109 biopsies correlated with a positive DSA. Of those 78 biopsies, only 18 exhibited diffuse C4d staining. In patients with graft failure who demonstrated DSA against class I only, class II only, or both, significant diffuse C4d staining was seen in 100% (2/2), 17% (1/6), and 71% (5/7) respectively. Patients with DSA to only class I or to both class I and class II had high graft failure rates of 40% (2/5) and 43% (9/20) with an average time to graft failure of 33 and 36 months, respectively. In contrast, patients who developed DSA to only class II exhibited a 24% (6/25) failure rate with an average time to graft failure of 95.8 months. Patients who did not develop DSA had a failure rate of 20% (12/60).

Conclusions: 1. Episodes of cellular rejection, but not AMR, may be a more frequent cause of graft dysfunction.

2. C4d tended to be a better indicator of graft failure in patients with DSA to both class I and class II or DSA to class I only than in patients with DSA to class II only.
3. Patients demonstrating DSA to class I only or DSA to both class I and II, have a decreased graft survival compared with patients demonstrating DSA to class II only.

317 Ischemic Cardiac Myocytes Express Membranous CD56 in the Setting of Cardiac Allograft Vasculopathy (CAV): A Potential Predictor of CAV in Endomyocardial Biopsies

KJ Henriksen, A Chang, AN Husain. The University of Chicago, Chicago, IL.

Background: CAV is an accelerated, diffuse form of coronary artery disease, characterized by concentric intimal proliferation and progressive luminal stenosis, which is a major cause of morbidity and mortality after cardiac transplantation. Since the process primarily affects the large epicardial coronary vessels, a histologic diagnosis cannot be rendered until the time of explant or autopsy. Interestingly, it has been reported that CD56 is upregulated in ischemic cardiac myocytes, and might play a cardioprotective role in metabolic stress. Considering that the development of CAV results in progressive cardiac myocyte ischemia, we hypothesized that the presence and intensity of CD56 expression in post-transplant endomyocardial biopsies might predict the development of CAV.

Design: A retrospective review of autopsy cases was conducted to identify cardiac transplant patients with CAV at the time of death, who had undergone serial endomyocardial biopsies at our institution. Using the archived formalin-fixed, paraffin-embedded tissue blocks, immunohistochemical staining for CD56 was performed on 3-5 serial endomyocardial biopsies and autopsy heart from each patient. Control was autopsy heart without any pathology.

Results: Six patients were identified who had undergone cardiac transplantation (age range 6-57 years) and had serial endomyocardial biopsies over the course of 14 months to 18 years (average: 8 years) prior to death. In 5 of 6 patients, the initial biopsy examined at 3-6 months post-transplantation showed CD56 staining isolated to the intercalated discs, with minimal cytoplasmic or membranous staining. Over time, the cardiomyocytes developed membranous CD56 expression, and eventual membranous and cytoplasmic CD56 as seen in the autopsy specimens. In one control autopsy heart without cardiac pathology, CD56 expression was primarily seen in the intercalated discs.

Conclusions: The detection of CAV in cardiac transplant recipients is heavily reliant on invasive procedures, which are often non-diagnostic. By utilizing immunohistochemical staining for CD56, a marker which is expressed in ischemic cardiomyocytes, we were able to track the progression of ischemic changes in serial endomyocardial biopsies of multiple patients who developed CAV. We propose that upregulation of CD56 expression in post-transplant endomyocardial biopsies might help predict which patients will develop CAV, thus enabling earlier detection and guiding appropriate management.

318 A Silent Cause of Sudden Cardiac Death: Anomalous Coronary Arteries

SF Hill, MN Sheppard. Imperial College London, London, United Kingdom; Royal Brompton and Harefield NHS Trust Hospital, London, United Kingdom.

Background: Pathologists performing examinations on hearts of sudden cardiac death (SCD) victims are not actively excluding anomalous coronary arteries (ACA) as a cause of death. Our aim is to increase awareness of this cardiac anomaly, determine the incidence and educate the pathologist on a systematic approach to examining the coronary arteries.

Design: Retrospective non-case controlled analysis.

Setting: Cardiac pathology centre at the National Heart and Lung Institute and Royal Brompton Hospital.

Subjects: Between 1994 and 2011, the hearts of 2030 SCD victims were referred for pathological assessment to ascertain the precise aetiology of SCD.

Results: Twenty-nine (1.4%) of the 2030 cases of SCD have been associated with ACA (22 men (76%) and 7 women (24% age range [16 months-63 years]). The specific anomalies identified were 10 cases of anomalous right coronary artery arising from the left aortic sinus; 6 cases of anomalous left coronary artery (LCA) arising from the right coronary sinus; 7 cases of myocardial bridging; 3 cases of coronary artery ostial stenosis/atresia 2 cases of anomalous origin of the LCA from the pulmonary artery; and 1 case of high take off. The anomalous coronary artery had been identified by the referring pathologist in only 11 of the 29 cases (38%). Histological evidence of acute/chronic ischaemia in the regional myocardium supplied by the ACA was evident in 23 of the 29 cases (79%). In 15 cases (52%) SCD occurred during or immediately after physical exertion. Cardiac symptoms were documented to have occurred in only 7 patients (24%) prior to SCD.

Conclusions: Anomalous coronary arteries are an under-recognised cause of sudden cardiac death. Detailed examination of the coronary artery system identifying both coronary artery ostia and their course should be routinely performed on SCD victims.

319 Vascular Survey in IgG4-Related Systemic Disease

BJ Holmes, NG Delev, GR Pasternack, MK Halushka. The Johns Hopkins Hospital, Baltimore, MD.

Background: IgG4-related systemic disease (IgG4-SD) is a rare cause of non-necrotizing ascending aortitis. However, the extent of IgG4-related arteritis outside of the aorta has not been described. Although IgG4-SD is increasingly recognized as a cause of aortic aneurysms, coronary artery dissection and sudden cardiac death have not been reported.

Design: We describe the pattern of IgG4-related arteritis in a 61-year-old African-American female. This patient died unexpectedly at home one year following an episode of presumptive lupus cerebritis that resolved with immunosuppression. An extensive vascular survey was conducted at the time of post-mortem examination. Immunohistochemical staining using anti-IgG (dilution 1:20,000, Novacastra) and anti-IgG4 (dilution 1:2,000, Invitrogen) was performed on selected sections.

Results: Postmortem examination revealed a 3 cm dissection of the ascending aorta that had propagated in a retrograde fashion into the proximal left main coronary artery. This significant coronary artery dissection caused the patient's sudden cardiac death. Microscopic examination of the proximal aorta revealed a marked lymphoplasmacytic arteritis where the dissection occurred. The plasma cells demonstrated a cytoplasmic IgG4:IgG ratio of approximately 80%. Our expanded vascular survey identified the same plasma cell-rich infiltrate affecting the entire aorta (thoracic and abdominal) as well as the bilateral common iliac arteries, left renal artery, and bilateral internal carotid arteries. The coronary arteries, right renal artery and splenic artery were unaffected.

Conclusions: We report the first extensive vascular survey of IgG4-SD, demonstrating that IgG4-SD can cause large-vessel vasculitis beyond the aorta. While IgG4-SD is typically viewed as a chronic disease, this case is also notable as the first report of IgG4-SD-induced sudden death and illustrates the possibility of vascular catastrophe due to IgG4-related arteritis.

320 Investigations into eNOS and Phosphomimetic eNOS Gene Delivery to the Vasculature

SO Hynes, S Ganly, F Sharif, L Smith, K McCullagh, U Greiser, Z Katusic, T O'Brien. University Hospital Galway, Galway, Ireland; National University of Ireland, Galway, Galway, Ireland; Mayo Clinic, Rochester, MN.

Background: Endovascular procedures including stenting, denudes endothelium. Removal of this layer, decreases the levels of active endoethelial nitric oxide synthase(eNOS) and bioavailability of NO. Gene delivery of eNOS or an engineered phosphomimetic constitutively active eNOS may prove of benefit to the vasculature.

Design: Two principles are critical to improving outcomes from endovascular procedures. First, to accelerate re-ednothelialisation and second, to prevent neointimal proliferation. We have examined gene therapy approaches to produce beneficial effects in the vasculature. We compared eNOS and phosphomimetic eNOS for their effects on vasomotor activity. Thereafter, we examined the usefulness of eNOS delivery from a stent platform in preventing in-stent restenosis and promoting re-endothelialisation. Two rabbit models of vascular injury were used. The first was a physiological model where we overexpressed our two candidate transgenes. The second was an injury model where a stent carrying the lead gene was delivered following inflation/deflation injury of the external iliac artery.

Results: Overexpression of phosphomimetic and wild type eNOS both improved vasomotor activity in a rabbit carotid artery model. However, there was no difference between either enzyme. Therefore, wild type eNOS was examined for its beneficial effects following delivery from a stent platform. We then compared viral (adenovirus) versus non-viral (liposome) delivery of wild type eNOS, head-to-head. Both vectors resulted in improved re-endothelialisation, however, only the use of viral vectors decreased neointimal formation.

Conclusions: Our study found that overexpression of an engineered constitutively active form of eNOS is not superior to wild type delivery. This may be due to suboptimal mimicking of pophosphorylation. Interestingly, the vector is critical for delivery of eNOS. When compared head-to-head only the viral vector improved both neointimal and re-endothelialisation results. We speculate that this may be due to the expression of non-viral vectors by macrophages which also express inducible nitric oxide synthase. The direct competition for substrates may be the reason for less effective eNOS function. Whereas, eNOS delivered by virus is expressed in smooth muscle cells and these have no native NOS to compete with the transgene.

321 The Prevalence of Common RNA Enteroviral Pathogens from Formalin-Fixed Paraffin-Embedded Myocardium Tissues of Myocarditis

JY Jhuang, CK Hong, CT Shun. National Taiwan University Hospital, Taipei, Taiwan; National Taiwan University, Taipei, Taiwan.

Background: The etiological factor of sudden cardiac death due to acute myocarditis has long been an important issue in forensic medicine. Due to the progression of molecular technique, there are increasing researches about the etiology of myocarditis. Although the cause of myocarditis in any given pations, systemic diseases, drugs, and toxins have been associated with the development of this disease. Viruses are an important cause of myocarditis in North America and Europe. The prevalence of viral myocarditis is still unclear in Taiwan. The purpose of this study was to investigate the prevalence of myocarditis infected by RNA enterovirus in Taiwan.

Design: The formalin fixed paraffin embedded myocardial tissue blocks of myocarditis were obtained from endomyocardial biopsy (9 samples), heart transplantation (1sample) and forensic autopsy specimen die of myocarditis (1 sample). Tissue blocks were studied by using immunohistochemistry and reverse transcriptase-polymerase chain reaction (RT-PCR) for enterovirus. This study showed that histological sections from 7 of 11 myocarditis cases were positive for the viral capsid protein VP1 by immunohistochemical staining. In 4of 7 VP1 immunohistochemical positive staining specimens, viral genome of enterovirus was detected by RT-PCR using 5'NTR genomic fragment.

Results: These results showed most RNA fragment recovered by RT-PCR which fall between 152-470 nt. The product of RT-PCR probe should less than200 bp which is the best candidate in these recovery. Also the study provided data of rough enteroviral prevalence in myocarditis (more than 36.4%).

Conclusions: Further evidence of prevalence about enterovirus involvement in myocarditis needs more large scale of such cases using the method provided in the study to get in Taiwan.

322 Age-Related Histologic Features of the Sinus Node in Normal Human Hearts during the First 10 Decades of Life: A Study of 200 Cases

CM Keeney, MC Castonguay, WD Edwards, JJ Maleszewski. Mayo Clinic, Rochester, MN.

Background: Age-related histologic features of the normal human sinus node have not been reported in large studies. It is thought that nodal collagen progressively increases as humans age, based mainly on small case series.

Design: 200 formalin-fixed grossly normal human hearts, obtained at autopsy from subjects without heart disease, were retrieved from the tissue archives of Mayo Clinic in Rochester, MN. For each of the first 10 decades of life, 20 hearts (10 from males and 10 from females) were selected. The sinus nodes were dissected from the right atria and processed for histology. 4 um-thick tissue sections were stained with Verhoeff-van Gieson, and, photomicrographs taken of the section of node with that largest cross-sectional area. Analysis was performed on the chosen section using ImageJ 1.44 (NIH, USA), evaluating for the following parameters: area of the sinus node (in mm^2), percent collagen, percent fat, average diameter of nodal myocytes, and average diameter of contractile atrial myocytes. Relationships with age and gender were also investigated.

Results: A significant cubic association was identified between mean sinus nodal area and age ($p = 0.0008$). Linear associations were seen between nodal myocyte diameters and between contractile myocyte diameters and age after the first decade ($p = 0.006$ and 0.008, respectively), and were most pronounced for contractile myocyte diameters. There were no associations between nodal collagen content (median = 22.5%, IQR = 17.8 – 27.0%) and age, nor between fat content (median = 0%, IQR = 0 – 0%) and age. Significant inter-individual variation was noted in these parameters. Adjusting for body mass index and body surface area did not influence these results.

Conclusions: This is the largest and only series to date describing the appearance of the normal human sinus node evenly divided throughout the first 10 decades of life. It contradicts the long-standing belief that nodal collagen content increases with age, which is of special relevance to evaluation of the conduction system in cases of sudden death. Significant associations between nodal area, nodal myocyte diameter, contractile myocyte diameter, and age were identified, despite significant inter-individual variation in these parameters – this variation should be borne in mind on a case-by-case basis.

323 Mass Spectrometry-Based Proteomic Characterization of Aortic Aneurysm Medial Degeneration in Marfan Syndrome and Congenitally Bicuspid Aortic Valve

BT Larsen, S Bosio, JD Theis, JA Vrana, A Dogan, DV Miller. University of Arizona, Tucson, AZ; University Hospital - Parma, Parma, Italy; Mayo Clinic, Rochester, MN; Intermountain Medical Center/University of Utah, Salt Lake City, UT.

Background: Medial degeneration (MD) is common in ascending aortic aneurysms in Marfan syndrome (MS) and congenitally bicuspid aortic valve (BAV). MD is thought to result from a complex sequence of events, though the cellular processes and protein level alterations are not well understood. Whether there might be detectable changes preceding overt MD that either signify a precursor state or intrinsically weaken the aortic wall is also largely unexplored.

Design: Surgical aortic tissue samples were obtained from 11 patients with MS showing MD, 7 with BAV and MD, and 9 controls without MD. Areas of MD and adjacent areas of preserved aortic media were each collected from 10 um paraffin sections by laser-capture microdissection under fluorescence microscopy (elastic fiber autofluorescence). These were digested with trypsin and analyzed by liquid chromatography electrospray tandem mass spectrometry. Raw spectral data were queried by the use of Mascot, Sequest, and X!Tandem. Peptide and protein probability scores were assigned and for each sample a list of proteins based on peptides identified by MS was generated. These lists were compared between patient groups.

Results: Substantial differences in several proteins of interest (filamin A, transgelin, β-actin, myosin 10 HC, myosin 11 HC, myosin regulatory LC 2, α3-collagen type VI, α1-collagen type III, α2-collagen type IV, fibronectin 1, EGF-containing fibulin-like extracellular matrix protein 1, clusterin, heat shock protein 3, plakoglobin, and desmoglein 3 and to a lesser extent myosin LC 6, transgelin 2, annexin A2, fibulin 1, and fibulin 5) existed in MD areas and to a lesser extent the adjacent intact media from MS and BAV samples relative to normal histology samples. MS samples had increased α1-actin in MD areas relative to MD in BAV. BAV samples showed more abundant desmoplakin in the intact media relative to MS, but these were reduced in areas of MD.

Conclusions: The aortic media in MS and BAV demonstrate unique protein expression profiles compared to normal histology controls. Some of these differences are accentuated in MD relative to areas of preserved architecture, suggesting a role in MD development and possible progression. Some these proteins may prove useful in further development of biomarkers for aneurysmal disease and progression.

324 IgG4-Positive Plasma Cells in Ascending Aortitis: Are They Diagnostic for IgG4-Related Aortitis/Periaortitis?

K Notohara, DV Miller. Kurashiki Central Hospital, Kurashiki, Japan; Intermountain Medical Center & University of Utah, Murray, UT.

Background: Aortitis/periaortitis is a manifestation of IgG4-related disease (IgG4-RD), but, so far, reported cases are mostly of the abdominal aorta and aortic arch. Noninfectious ascending aortitis, especially of an isolated variant, may belong to IgG4-RD, but compared to abdominal aorta, the data are limited.

Design: Resected specimens from 98 patients were reviewed, and cases with dense plasmacytic infiltration and fibrosis but without granulomatous reaction were selected for immunostaining for IgG4 and IgG1. Cases with abundant giant cells or granulomatous reaction were presumed unlikely to be IgG4-related due to the rarity of this finding in IgG4-RD, but some cases (particularly those with concomitant dense plasmacytic infiltration) were also included for comparison. The number of $IgG4^+$ plasma cells (PCs) in a high power field were graded as: 1 (10 or fewer), 2 (11-20), 3 (21-30), 4 (31-50), and 5 (51 or more). The ratio of $IgG4^+$:$IgG1^+$ staining PCs was also determined for each case.

Results: Among 14 cases that met the criteria, 11 cases had available paraffin blocks and were selected. 8 cases with plasmacytic plus granulomatous/giant cell reaction were also selected for comparison. Among 11 cases, only one was consistent with IgG4-RD. This case had a marked number (grade 5) of $IgG4^+$ PCs, high $IgG4^+/IgG1^+$ PC ratio (80%) and a histological finding of storiform fibrosis mainly in the adventitia. Increased (grade 2-5) $IgG4^+$ PCs were present in 8 other cases, however, all of these cases had lower $IgG4^+/IgG1^+$ PC ratios; not consistent with IgG4-RD. 3 cases with a mild (grade 2) increase of $IgG4^+$ PCs and lower ratio of $IgG4^+/IgG1^+$ PC showed the typical storiform fibrosis, but the lesion was admixed with mild infiltration of neutrophils, which is unusual for IgG4-RD. $IgG4^+$ PCs were also increased (grade 2-4) in the 8 cases with granulomatous reaction, but all of these cases showed a low $IgG4^+/IgG1^+$ PC ratio.

Conclusions: Although $IgG4^+$ PCs were commonly identified in ascending aortitis with or without granulomatous reaction, only one patient (out of 19) was considered to have true IgG4-RD after taking the $IgG4^+$:$IgG1^+$ PC ratio into account. The diagnosis of IgG4-related aortitis/periaortitis should be evaluated with $IgG4^+/IgG1^+$ PC ratio. Storiform fibrosis, which is believed to be specific to IgG4-RD, may not be entirely specific to IgG4-related lesion in the aorta when neutrophils are present. The incidence of IgG4-RD affecting the ascending aorta appears to be significantly less than in the abdominal aorta.

325 Carotid Plaque Inflammation and Morphology Is Associated with Early Stroke Recurrence

S Prendeville, M Marnane, A Merwick, O Sheehan, N Hannon, T Grant, P Kelly, N Mulligan. Mater Misericordiae University Hospital, Dublin, Ireland; University College Dublin, Dublin, Ireland.

Background: Patients with symptomatic carotid stenosis (CS) have a high risk of stroke recurrence (SR), even within the recommended 14-day time window for endarterectomy. Inflammation has been implicated as a major driver of atherosclerosis progression and plaque destabilisation, however a direct relationship with SR has not been established. The aim of this study was to investigate histological plaque features associated with early SR pre-endarterectomy, in a cohort of patients with recently-symptomatic CS.

Design: Endarterectomy tissue from 44 consecutive patients with recent transient ischaemic attack, non-disabling ischaemic stroke, or retinal embolism was analysed using a validated system for grading plaque inflammation and morphology. All patients were followed prospectively for SR occurring pre-endarterectomy and not related to surgery.

Results: SR occurred in 27.3% of patients and was significantly associated with: carotid plaque macrophage and lymphocyte infiltration ($p=0.002$, $p=0.009$ respectively), extensive (>25%) fibrous cap disruption ($p=0.004$), neovascularisation ($p=0.04$) and low plaque fibrous tissue content ($p=0.003$). There was no association found for intraplaque haemorrhage, lipid-rich necrotic core size, plaque calcification, foam cell content or the American Heart Association morphologic plaque classification.

On life-table analysis, actuarial pre-endarterectomy SR rates were 82.3% (CI 49.2-98.8%, 11/23 patients) in patients with extensive plaque inflammation (Oxford Plaque Study [OPS] macrophage content grade ≥3) compared to 22.2% (CI 3.5-83.4%, 1/21 patients) in those with lesser degrees of inflammation (OPS macrophage content grade <3) (log-rank $p=0.009$). In a multivariable Cox regression model including plaque inflammation, age, and degree of CS, plaque inflammation was the only variable independently associated with pre-endarterectomy SR (adjusted HR 9, CI 1.1-70.6, $p=0.04$).

Conclusions: This study demonstrates for the first time that histologically proven plaque inflammation is an important predictor of early SR in recently symptomatic CS. Histological features of plaque instability such as intraplaque haemorrhage and lipid necrotic core size were not associated with early SR, which suggests a different plaque biology. Imaging technology, which can assess plaque inflammation in vivo, as well as biomarkers, may identify the subgroup of patients at high risk for early SR, independent of degree of stenosis, who can be targeted for early intervention.

326 How To Use Serial C4d and C3d in the Diagnosis of Antibody Mediated Rejection vs. Accommodation: 51 Month Experience with 550 Heart Transplant Patients

ER Rodriguez, CD Tan. Cleveland Clinic, Cleveland, OH.

Background: We have shown that serial assessment of C4d and C3d allows accurate diagnosis of antibody mediated rejection (AMR) in heart transplants, with high positive predictive value and high correlation to donor the presence of specific antibody (DSA) and clinical symptoms. In addition there is high mortality in the C4d+ C3d+ patients. However, the meaning C4d deposition alone is difficult to interpret by pathologists.

Design: All endomyocardial biopsies from October 2006 to December 2010 (51 months) were stained for C4d and C3d by immunofluorescence. A total of 4876 biopsies from 520 adult patients (age 20-73) were reviewed. There were 405 males and 115 females. An average of 13 biopsies per patient were examined. Electronic medical records were queried for: 1. Allograft dysfunction; 2. Serologic evidence of anti HLA antibodies; 3. Evidence of cardiac allograft vasculopathy (CAV).

Results: Thirty six (7%) of 520 patients were positive for C4d and C3d, diffuse in capillaries and 42 (8%) of 520 were positive for diffuse C4d only. Thirty three were focally positive for C4d only. There were no C3d positive only patients in this cohort. Histologic features of AMR were seen in 36% of biopsies. The median time for conversion of negative biopsy to positive for complement deposition was 30 months for C4d+ / C3d+ patients. The median for C4d+ diffuse was 10 months and for C4d+ focal was 15 months. Diffuse C4d positivity when examined in serial biopsies is mostly fortuitous and not associated with allograft dysfunction. Focal positive C4d is not correlated with DSA or dysfunction over time. There was no significant correlation

found between the number of positive biopsies and mortality. Only 6 of 42 diffuse C4d+ cases evolved to show DSA and dysfunction. Thirty six of 42 diffuse C4d+ cases showed no dysfunction, despite the presence of DSA in 6 of them. Thus interpreted as accommodation.
Conclusions: Serial staining of biopsies shows that: 1. Concomitant C4d and C3d positivity correlates highly with allograft dysfunction and DSA; 2. Diffuse C4d capillary staining alone should not be equated with AMR; 3. Most C4d+ episodes are single occurrences and asymptomatic; 4. The presence of C4d staining and DSA without allograft dysfunction may indicate accommodation; 5. Only a small fraction of patients with C4d staining alone may develop AMR on follow-up.

327 Carbonic Anhydrase IX – Hypoxia Marker in the Aortic Wall

Y Sheykin, S Rosen. Beth Israel Deaconess Medical Center, Boston, MA; Harvard Medical School, Boston, MA.
Background: Carbonic anhydrases (CA) catalyze conversion of CO_2 and H_2O to HCO_3^- and H^+. CA IX is particularly interesting because of its over expression in human cancers, likely to protect the tumor from acidotic environment engendered by anaerobic metabolism. Its expression is triggered by hypoxia-inducible factor-1 alpha, the master regulator of cellular response to hypoxia. Anti-CA IX antibody is clinically available for diagnostic imaging and potential cancer immunoradiotherapy.
Hypoxia in the vascular wall is one of the factors contributing to development of atherosclerosis and aortic aneurysms. Large arteries are particularly susceptible to hypoxia by nature of their blood supply. Luminal diffusion and adventitial vasa vasorum provide adequate vascular wall oxygenation in a healthy vessel, but fail to do so in various pathologic states. Indeed, frank aortic infarction is commonly seen in dissecting aortic aneurysms, following rather than preceding the latter (Circulation 1978;58:876-81).
Design: Using immunohistochemistry, we investigated expression of CA IX in aortic specimens obtained from patients with non-dissecting aortic aneurysms (n=13), dissecting aortic aneurysms (n=6) and granulomatous vasculitis (n=4). These were non-selected vascular surgical specimens received by our department within an 8-month period.
Results: In non-dissecting aneurysms, staining was frequently found in smooth muscle cells in the central media. In dissecting aneurysms, smooth muscle cells surrounding the areas of dissection were positive for CA IX. Vasculitis specimens exhibited strong CA IX staining within inflammatory foci and the surrounding smooth muscle cells. Staining was also seen in smooth muscle cells underlying atherosclerotic plaques and within the infarcted zones (CA IX is relatively resistant to proteolytic degradation). A further confirmation that CA IX staining truly indicates hypoxia was that prostate, testis and kidney infarcts (n=5) showed intense staining in zones bordering infarcted areas in which hypoxia would be anticipated.
Conclusions: These findings show that CA IX is consistently expressed in the diseased aortic wall and likely reflects hypoxic injury. CA IX up-regulation in the vascular wall is likely an adaptive mechanism aimed at preservation of tissue viability during hypoxic stress. A potential exists for in vivo assessment of CA IX expression in aortic wall for identification of past or on-going hypoxia.

328 The Changing Face of Infective Endocarditis: Ten Years Experience

EA Swanson, S Shahbazi, C Lai, MC Fishbein. University of California, Los Angeles, Los Angeles, CA.
Background: Infective endocarditis (IE) has high morbidity and mortality. Over the years, there has been a shift in predisposing conditions, causative organisms, guidelines for prophylaxis, and treatment. For example, routine prophylaxis for dental procedures is no longer recommended in cardiac conditions which have lower risk of adverse outcome from endocarditis, such as mitral valve prolapse and aortic stenosis.
Design: Eighty-four cases of IE requiring surgical intervention were identified from the surgical pathology database over the past ten years. Clinical and pathologic features were obtained from the medical record and pathology reports, including demographics, source of infection and predisposing factors, disease course, and surgical treatment.
Results: Seventy-one cases of native valve, along with 13 cases of prosthetic valve IE were examined. In the native valve cases, the median age was 53 years and 64.8% were men. Of these cases, 24% were temporally associated with dental procedures, or occurred in patients with poor dentition; 18.6% of the cases occurred in patients with diabetes mellitus (DM), and 17.1% in patients with end stage renal disease. Only 5.7% of this patient population had infections related to intravenous drug abuse. The majority of the causative organisms were oral and skin flora, consisting of Strep viridans (28.6%) and coagulase negative Staph (11.4%). Methicillin resistant Staph aureus (MRSA) was the pathogen in 17.1% of cases. In 51.3% of the cases, an underlying native valve or heart disease was identified as a risk factor for the infection, of which 15% were mitral valve prolapse and 15% bicuspid aortic valve. Only one case of underlying rheumatic heart disease was identified. All valves showed acute and chronic changes, 91.4% had vegetations noted by imaging or pathologic examination, and 73.2% had organisms seen histologically in spite of antibiotic therapy. There was a trend towards more valve repairs, as opposed to replacement, in more recent years.
Conclusions: In this contemporary series of IE cases undergoing surgery, many more cases are associated with mitral valve prolapse or bicuspid aortic valve than rheumatic heart disease. Many patients had DM and renal failure predisposing to infection. Oral and skin flora were the causative organisms in the majority of cases; however MRSA was frequently identified. In spite of recommendations for more restricted use of prophylaxis, in this series, there was continued association between dental procedures/disease and endocarditis.

329 Pathological Features of Adventitial inflammatory Reaction in Acute Aortic Dissections

LF Xu, C Miller, AP Burke. University of Maryland Medical Center, Baltimore, MD.
Background: Histological reaction in the adventitia to aortic dissections has not been well studied. We present histological findings in a series of acute aortic dissections with emphasis regarding dating and inflammatory reaction.
Design: We prospectively studied 43 surgically excised acute ascending aortic dissections. We evaluated the histological reaction in the media and adventitia adjacent to the acute dissection plane in 4 or more sections of aorta oriented perpendicularly. Inflammation (both degree and type) and stromal reaction were semiquantitated and correlated with duration of symptoms prior to surgical repair.
Results: Of the 43 cases, there were 31 men (ages 53 ± 14 years) and 13 women (ages 60 ± 17 years). Duration of symptoms was classified as <12 hours (n= 9), 12-24 hours (n=12), 1-2 days (n=8), 2-7 days (n=11), and > 1 week (n=3). Medial inflammation was usually relatively sparse. When present, neutrophils were detected within 12 hours, and in one case there was intense medial inflammation suggestive of aortitis. Lymphocytes were present after 12 hours, and macrophages were present after 1 day and peaked between 2-7 days. Lymphocytes and macrophages were observed in 80% of cases occurring in this time frame, and were numerous in 3 cases mimicking vasculitis. Comparatively, adventitial inflammation was relatively brisk. Neutrophils occurred before 12 hours, peaked between 12-24 hours, and were rare after 2 days. Eosinophils occurred after 1 day, peaked between 2-7 days, and were predominant in 3 cases between 2-7 days. Apoptosis occurred after 12 hours, peaked between 1-2 days; mitotic figures were present in similar time frame as apoptosis, but were still numerous up to 7 days. Macrophages followed by reactive fibroblasts were present after 1 day and peaked 2-7 days. Prominent inflammation of adventitial nerves and proliferation of paraganglial cells were observed in 7 cases; these cases showed neural influx of atypical macrophages and stromal cells. Hemosiderin-laden macrophages were present only in one case, which had a concomitant healed dissection.
Conclusions: Reactive changes in the adventitia and media are fairly reliable, and can be used to date aortic dissections in the first week after medial rupture. Inflammatory reaction in the media can occasionally contain numerous neutrophils, lymphocytes, or macrophages, mimicking vasculitis, and eosinophils may be prominent in the adventitia.

Cytopathology

330 The Utility of Fine-Needle Aspiration in the Diagnosis of Primary Lung Tumors and Metastatic Tumors to the Lung, a Retrospective Examination of 1032 Cases

JA Adams, HH Wu. Indiana University School of Medicine, Indianapolis, IN.
Background: With the emergence of improved treatment strategies for patients with malignant lung tumors it has become increasingly more important to adequately diagnose and subclassify lung lesions. In our large retrospective study, we assessed the utility of fine needle aspiration (FNA) in the diagnosis of primary and metastatic tumors to the lung.
Design: We reviewed the archived reports for 1032 patients undergoing FNA of primary lung tumors, metastatic lung tumors, and metastatic tumors to the lung. Based on the diagnoses that were rendered, the cases were grouped into atypical, benign, malignant, nondiagnostic, and suspicious lesions. The malignant FNA cases were further subclassified based on tumor type. Cases with correlating histology were then reviewed and diagnoses compared.
Results: The 1032 FNA cases were grouped as follows; 34 (3.3%) atypical, 142 (13.8%) benign, 717 (69.5%) malignant, 121 (11.7%) nondiagnostic, and 18 (1.7%) suspicious. Subclassification of malignancies diagnosed on FNA were as follows; 297 (41.4%) adenocarcinoma, 159 (22.1%) squamous cell carcinoma, 56 (7.8%) small cell carcinoma, 53 (7.4%) non-small cell carcinoma (NSCLC), 123 (17.2%) metastatic tumors, 15 (2.1%) neuroendocrine carcinoma, and 7 (1%) poorly differentiated carcinoma. Out of all NSCLC cases, 90% were able to be subclassified into either adenocarcinoma or squamous carcinoma on cytomorphology alone or with the help of immunohistochemical stains. Immuno stains were performed on 276 (27%) of the cases. The most frequent origins of metastatic tumors were renal cell carcinoma (22), melanoma (17), colon (15), breast (14), and urothelial carcinoma (10). There was also metastasis from 20 other organs with fewer than 4 cases each. 196 of 335 histologic follow-up specimens were biopsies (transbronchial or transthoracic core). Comparison of the FNA and surgical biopsy showed a sensitivity of 96% for FNA versus 98% for biopsy and a specificity of 100% for both. Sampling error resulted in 8 false negative cases on FNA. The diagnostic rate for FNA was 88.3% (vs 96% for surgical biopsy) and 91.6% of FNAs were able to specifically subtype a malignancy compared to only 80.6% of surgical biopsies.
Conclusions: FNA is comparable to histologic examination in the diagnosis and subclassification of both primary and metastatic lung tumors. 90% of NSCLC cases were able to be further subclassified into adenocarcinoma or squamous cell carcinoma by FNA.

331 Thyroid Bed Fine-Needle Aspiration: A Clinicocytologic Correlation

LJ Adhikari, J Reynolds, S Jenkins, A Nassar. Mayo Clinic, Rochester, MN; Cleveland Clinic, Cleveland, OH.
Background: Monitoring changes in the thyroid bed (TB) is one of the clinical mainstays for surveillance of recurrent thyroid carcinoma. Fine needle aspiration (FNA) is a diagnostic tool that is commonly used to aid in the decision of further clinical treatment options and follow-up.