GS21 1351 Nano course in Cardio-oncology (2020)

Director: Dr. Jun-ichi Abe
Dates:  September 25th – December 11th
Time:  3:00 pm – 4:30 pm (CST)
Place:  Virtual
Class meets every Friday of the week.
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Course materials will be available through Box, for access contact: Jun-ichi Abe

Synopsis: This course is designed to provide students with not only a comprehensive overview of the
structure and function of the cardiovascular system (CVS) in both normal and pathological states, but also
cancer and cancer treatment can affect CVS function. Disease processes affecting normal cardiovascular
homeostasis will be discussed in the context of both human disease and experimental model systems. The
course will introduce clinical/translational topics, signal transduction and current therapies of both the
CVS and cancer, and potential avenues for novel cardiovascular research from the view of cardio-
oncology. Lecturers include both clinical and basic scientists, providing a bench-to-bedside addition to
the Ph.D. curriculum. There will be one 90-minute meetings per week, which will include lectures, paper
discussions, case studies and lab studies. Evaluations will be based on a class participation, and written
reports. The course is designed as an elective for students from numerous programs, and will ultimately
be expanded into a two-semester course. There are no prerequisites for this course. Class size will be
from 4-20 students, including a limited number of auditors. Auditors need to register for the course.
Lectures are based on historical and recent literature. There is no required textbook.

Learning Objectives

1. Understand the principles, challenges, approaches, and strategies of cardio-oncology research.
   Therefore, the trainees will be expected to attain the basic knowledge in terms of cancer
treatment-associated CVD as well as both CVD and cancer treatments.
2. Understand the cardiac and vascular structure, and circulation system and function, and get the
   picture of the pathophysiology of common and major diseases of the cardiovascular system.
3. Grasp the molecular basis of cardiac contractility (e.g. E-C coupling, myosin-actin filaments) and
   electrophysiology (e.g. A-V conducting system and ion channels), and describe how
   abnormalities of these mechanisms produce important cardiovascular diseases, and understand
   the basics and molecular mechanisms of the process of vascular injury including atherosclerosis,
   aneurysm, restenosis, and hypertension.
4. Appreciate the importance of genetic factors in certain cardiovascular diseases and cancer
   treatment-associated CVD, and how to approach and analyze it.
5. Understand the current pharmacological strategy against CVD including ACEs, β-blockers, and
   PDE inhibitors in non-cancer patients.
6. Understand the current pharmacological strategy against oncogenesis, especially related to signal
   transduction and epigenetics including tyrosine kinase inhibitors (TKIs), DNA synthesis, histone
   de-acetylase (HDACs), and proteasome inhibitors.
7. Learn the incidents, pathogenesis, diagnosis, management, and prevention against cancer
   treatment associated CVD including heart failure, coronary and cerebral events, hypertension,
   thromboembolism, and arrhythmia.
8. Recognize the contribution of premature aging process in CVD, cancer, and cancer treatment-associated CVD, especially for long-term effects after therapy, and its molecular mechanisms.
9. Understand the contribution of cancer treatment in increasing risk factors of CVD such as hypercholesterolemia and obesity and its molecular mechanisms.
10. Obtain the ability to critically evaluate the literatures related cancer treatment-associated CVD.
11. Apprehend and be able to articulate the potential and future directions of cardio-oncology including targeting the down-stream or CVD specific events induced by cancer treatments, which will prevent CVD but will have no effect on the efficacy of cancer treatment. For example, topoisomerase-IIβ (top2β) is reported to be one of the direct target molecules of cardiotoxic drugs of the anthracycline family. Thus, depletion of top2β ameliorates anthracycline-mediated cardiotoxicity. Notably, since the heart only expresses top2β, new anthracycline that only poisons top2α, but not top2β, will be beneficial for healing cancer, but avoiding cardiomyopathy.

Sept. 25 Introduction to cardio-oncology Dr. Jun-ichi Abe
Oct.  2 Cardiovascular health during cancer care Dr. Elie Mouhayar
      9 Anti-cancer drugs and cardio-oncology Dr. Nicolas Palaskas
      16 Arrhythmias in cardio-oncology Dr. Peter Kim
      23 Radiation and CVD toxicity from the perspective of cardiologist
           Dr. Syed Wamique Yusuf
      30 Assessment of Cardiovascular function in cancer patients Dr. Jose Banchs
Nov.  6 Cardiovascular toxicity in pediatric cancer patients Dr. Eugenie Klieinerman
      13 Role of MRI in cardio-oncology Dr. Juan Lopez-Mattei
      20 Radiation Induced Heart Disease: Mechanism and Prevention Dr. Steven H. Lin
Dec.  4 Angiogenesis and chemo/radiation-resistance Dr. Sue-Hwa Lin
      11 Pathophysiology of CVD in cardio-oncology Dr. Jun-ichi Abe