Bland Thrombosis and Infection in Relation to Intracardiac Catheter

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Intravascular catheters exhibit tendencies to become covered with thrombotic material\(^1\)\(^-\)\(^3\) and to cause intimal erosion with secondary mural thrombosis of the vessel containing the catheter. The incidence of these phenomena has a direct relationship to the amount of time that the catheter is in place. Thrombosis on the surface of the catheter or on the vascular intima set the stage for further complications in the form of vascular obstruction,\(^4\)\(^-\)\(^6\) embolism, infection,\(^4\)\(^-\)\(^6\) and fibrous encasement of the catheter. This report and a companion article to follow illustrate some of the complications that may follow the long-term presence of catheters or catheter electrodes in the right side of the heart.

In this communication are presented examples of (1) erosion with secondary bland mural thrombosis of the superior vena cava and (2) infection of the superior vena cava or mural endocardium of the right atrium and nearby structures in patients harboring indwelling catheters.

Case 1.
Bland Thrombosis of Superior Vena Cava

After resection of a carcinoma of the colon, a 65-year-old man developed pulmonary embolism. For the latter, treatment included ileofemoral venous thrombectomy, clipping of

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\(\text{S.V.C.} \quad \text{R.A.} \quad \text{F.O.} \)

Figure 1

Case 1. (a) At junction of superior vena cava (S.V.C.) and the right atrium (R.A.) is a mural thrombus (between arrows). F.O. = fossa ovalis. (b) Photomicrograph of junction of superior vena cava and right atrium. A portion of the mural thrombus observed grossly is attached to the endocardial surface. The thrombus is fresh, without signs of organization. Although a moderate cellular inflammatory reaction is present in the tissues under the thrombus, no organisms were present. Elastic tissue stain (\(\times \) 3.4).
Figure 2

Case 2. (a) Interior of right atrium (R.A.) and posterior part of opened tricuspid valve. A focus of vegetations (between arrows) of various sizes is present on the endocardial aspect of the right atrium. Vegetations are also present on the posterior (P.) and septal (S.) tricuspid leaflets. (b) Low-power photomicrograph of section through right atrium (R.A.), right ventricle (R.V.), and posterior tricuspid leaflet. Vegetations are present on the endocardial aspect of the right atrium and on the atrial surface of the tricuspid leaflet. Hematoxylin-eosin stain (× 5). (c) Low-power photomicrograph of septal tricuspid leaflet and right ventricular aspect of ventricular septum (V.S.). The leaflet shows an inflammatory reaction with surface deposition of vegetations. Hematoxylin-eosin stain (× 6.2). (d) Photomicrograph of vegetations on septal leaflet of tricuspid valve. Coeci are present as a colony and in disseminated form. Hematoxylin-eosin stain (× 540).
Case 3. (a) Interior of junction of superior vena cava (S.B.C.) and right atrium (R.A.). A pedunculated thrombus is attached to lining. (b) Photomicrograph of a section through the junction of the superior vena cava and the right atrial wall. A thrombus is attached to the lining. Hematoxylin-eosin stain (×4). (c) High-power photomicrograph of thrombus shown in (a) and (b). Numerous blastospores of Candida are present. Hematoxylin-eosin stain (×250). (d) Photomicrograph of esophagus. Ulceration. In higher magnifications Candida blastospores were identified in the base of ulcer. Hematoxylin-eosin stain (× 9.5).

the inferior vena cava, and continuous administration of heparin.

Through the venous route, a catheter was inserted into the right atrium for measurement.
of the central venous pressures. The patient died 6 days after the latter procedure. Necropsy revealed the catheter in place and a grossly evident bland thrombus attached to the junction of the superior vena cava and right atrium (fig. 1).

Case 2.
Staphylococcal Endocarditis of Right Atrial Wall and Tricuspid Valve

After an automobile accident with resultant cerebral manifestations in a 76-year-old man parenteral nutrition was required. For this purpose, a central venous catheter was inserted. Three weeks later the patient developed fever. Cultures of the blood and the tip of the catheter were positive for Staphylococcus aureus. The patient died 1 week later, 4 weeks after insertion of the intracardiac catheter.

Pathologic examination revealed vegetations involving the posterior wall of the right atrium and subjacent tricuspid valve. Histologic examination revealed an inflammatory lesion typical of bacterial endocarditis with colonies of cocci present in the vegetations (fig. 2).

Case 3.
Candida Albicans Endocarditis of Superior Vena Caval-Right Atrial Junction

An abdominal exploration was performed in a 56-year-old woman who was suffering from a pancreatic abscess and retroperitoneal fat necrosis resulting from acute pancreatitis.

From the time of the operation until death 1 month later, a nasogastric tube as well as a central venous catheter, the latter being for feeding purposes, were in place. The terminal illness was characterized by progressive deterioration, fever, and abdominal drainage. Blood cultures and the abdominal wound yielded Candida albicans. The same organism was recovered from postmortem blood culture.

Necropsy revealed peritoneal and retroperitoneal abscesses with fat necrosis. At the junction of the superior vena cava and the right atrium, a large polypoid friable thrombus was attached to the lining. Histologic examination revealed an inflammatory reaction beneath the attached thrombus. Blastospores of Candida were present within the thrombus. An embolus containing Candida was present in a secondary pulmonary artery.

The esophagus was ulcerated with blastospores of Candida in the tissues beneath the ulcers. The esophageal infection is considered to have been the portal of entry from which dissemination of organisms in the blood stream occurred. It is assumed that the catheter had been responsible for the formation of a right atrial thrombus, as in case 1, and in the presence of circulating organisms the thrombus became secondarily infected (fig. 3).

References
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