Complications Specific to the Type of CSF Shunt: Atrial Shunt

12

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12.1 Introduction

Thanks to the development of sophisticated and reliable ventriculoperitoneal shunts (VPS) and to the wide and still increasing diffusion of neuroendoscopy, the use of ventriculoatrial shunts (VAS) is currently limited to selected cases in many Centers, namely, those where the two previous treatments failed. VPS actually are burdened by a lower rate of severe complications and revisions [4], and they are more easy to be placed and revised so that they have replaced VAS as gold standard treatment for hydrocephalus starting from the 1970s [22, 24]. Moreover, VPS do not need to be periodically lengthened to follow the body growth, which is a significant advantage over VAS in children. Neuroendoscopy, on the other hand, offering the possibility to completely avoid shunting prostheses, is used whenever possible, sometimes after the removal of a previous VAS [40]. On these grounds, there are only few papers currently focusing on the complications of VAS, which generally report on isolated cases. Indeed, the clinical series consisting of patients with this type of CSF shunt are often little and/or dated, and most of the available data refers to papers written during the VAS era, that is, about four decades ago.

The complications of VAS can be divided into two groups: those proper of VAS, as cardiopulmonary complications and shunt nephritis, and those shared with VPS but with a peculiar course, such as catheter displacement and infections. In

C. Di Rocco et al. (eds.), *Complications of CSF Shunting in Hydrocephalus: Prevention, Identification, and Management*, DOI 10.1007/978-3-319-09961-3_12,

this chapter, both types of complications will be addressed.

12.2 Complications Unique to Atrial Shunts

12.2.1 Pulmonary Thromboembolism and Its Consequences

Thromboembolic complications of VAS may present as silent pulmonary embolization or as pulmonary vascular occlusive disease, pulmonary hypertension, or even right heart failure. Therefore, the exact incidence of these complications is hard to be established, mainly because of the undiagnosed cases or because of the difficulties in recognizing the clinical onset of the disease. However, a 0.4 % rate of clinically evident lung embolism and a 0.3 % rate of pulmonary hypertension are now universally accepted [18, 32, 40]. The autoptic investigations, instead, point out significantly higher figures, the frequency of pulmonary embolism ranging around 60 % while that of pulmonary hypertension around 6 % [13, 39].

Pulmonary embolization is caused by the detachment of emboli from a partial or complete thrombosis of the superior vena cava or right atrium. Such a thrombosis is thought to result from the presence of a foreign body (the shunting catheter) into these vascular cavities. However, the incidence of thromboembolism in patients with VAS is higher than in patients with other foreign bodies (e.g., cardiac pacemaker) [28]. Therefore, several hypotheses have been formulated to explain such a greater rate: (1) Concomitant presence of infection: The frequency of thromboembolism is increased in patients who experienced septicemia [31]. However, based on autoptic observations, some authors concluded that sepsis is not a necessary condition for embolization although the histological changes are more severe in cases of septic emboli (inflammatory process extended to the adventitia and the surrounding tissues) than in cases of bland emboli [14]. (2) Atrial ulceration due to a mechanical injury by the tip of the shunting catheter, leading to a chronic endocarditis followed by atrial thrombosis [30]. (3) Chemical injury by the CSF against the endothelium of the lung vessels, with subsequent thrombosis in situ and pulmonary hypertension [32]. (4) Thromboplastic-like activity by some CSF components [15, 35]. On the other hand, the age of the patient, the shunt material, and the location of the atrial catheter do not seem to influence the occurrence of thromboembolic complications [14, 17].

Pulmonary thromboembolism is generally an early complication (days or months after the shunt placement), thought it can also occur later on (even years after the shunt implantation) [18, 19, 28]. As mentioned before, it can remain asymptomatic in some patients, namely, those with microembolization where the small emboli are lysed before occluding pulmonary arteries or are not able to completely obstruct the arterial lumen [14]. In symptomatic cases, pulmonary embolism is complicated by respiratory symptoms and pulmonary hypertension (revealed by accentuation of the second heart sound and by the murmurs due to pulmonary or tricuspid valve insufficiency) leading, in turn, to cor pulmonale and, eventually, to irreversible right heart failure. On the other hand, chronic non-thromboembolic pulmonary hypertension is exceptional and it is induced by repeated infections [1]. Pulmonary hypertension accounts for almost all the deaths occurring after thromboembolic complications [11, 32, 34]. Sleigh death in asymptomatic cases or in patients with chronic thromboembolic pulmonary hypertension has been also reported [13, 30].

The diagnostic work-up includes: (1) arterial blood gas analysis, seeking for hypoxia; (2) chest X-rays, looking for cardiomegaly and dilatation of the proximal pulmonary arteries (Fig. 12.1) (a pulmonary angiogram can confirm the dilatation and/or obstruction of the pulmonary vessels); (3) ventilation-perfusion lung CT scan, to find the signs of embolization (multiple subsegmental perfusion defects); (4) electrocardiogram, usually showing right ventricle hypertrophy and right axis deviation (Fig. 12.2); (5) echocardiography, which can provide information on the dilatation of the right heart, the right ventricle

hypertrophy, the estimated pulmonary artery pressure, the tricuspidal insufficiency, and the possible presence of atrial/vena cava thrombosis (Fig. 12.3); and (6) cardiac catheterization, to confirm and to establish the severity of the pulmonary hypertension.

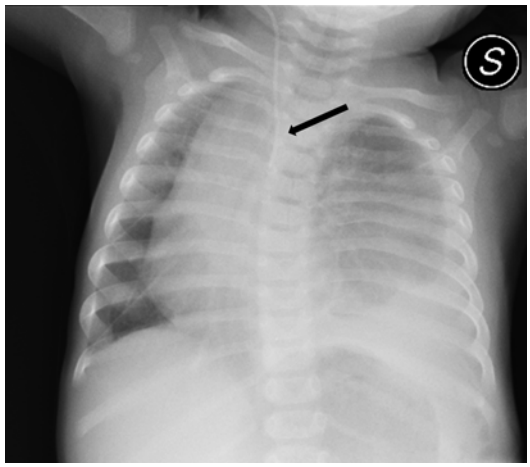

Fig. 12.1 Hypertrophy of the right cardiac cavities with right deviation of the heart in a child with a VAS positioned at T4 level (*arrow*) who developed pulmonary embolism and hypertension

The surgical management consists of the removal of the VAS, to be performed as early as possible and to be converted into VPS or other shunts (e.g., ventriculo-gallbladder or ventriculo-bladder shunt), or endoscopic treatment. The medical treatment is first based on pharmacological thrombolysis, then on diuretic and anticoagulant drugs. Medical therapies including vasodilators and prostacyclin analogues can be used in the attempt of reducing the pulmonary vascular resistance [3]. Unfortunately, the prognosis of pulmonary hypertension complicating VAS is often dismal, with a 50–100 % rate of mortality [28, 39]. Reduction of the risk of shunt colonization, elective revision of the shunt if a migration into the superior vena cava is detected, periodic checkup screening to exclude atrial thrombosis or initial signs of pulmonary hypertension (chest X-rays, electrocardiography, echocardiography), and, according to some authors [35], anti-aggregation drug prophylaxis, can be

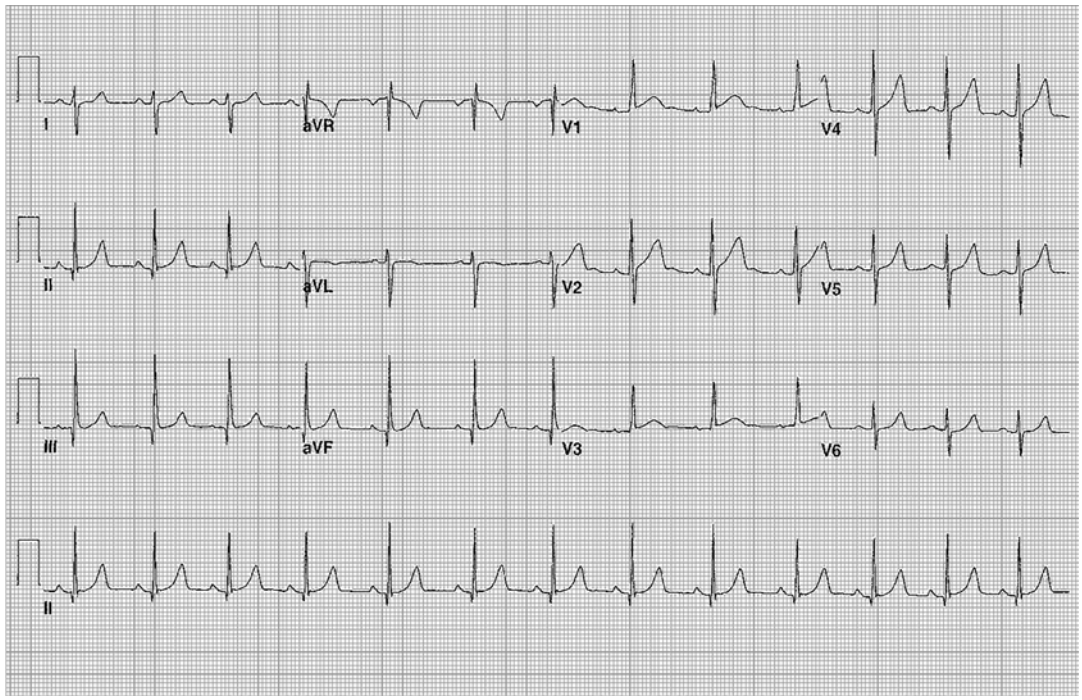

Fig. 12.2 Electrocardiogram showing the signs of right hypertrophy and right deviation of the cardiac axis in a patient with cor pulmonale

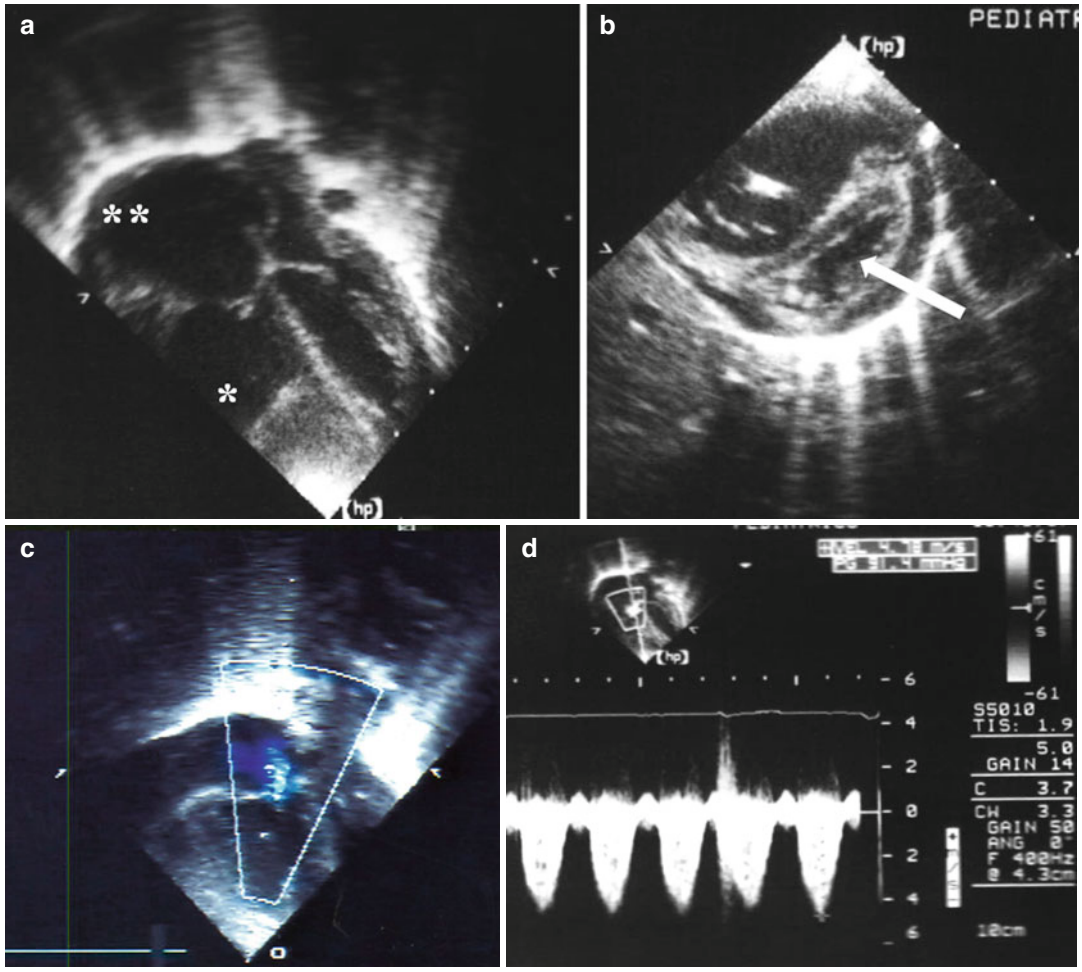

Fig. 12.3 Echocardiographic signs in a case of pulmonary hypertension: (**a**) severe enlargement of the right ventricle (*asterisk*) and atrium (*double asterisk*); (**b**) hypertrophy of the right ventricle with shift of the ventricular septum (*arrow*) toward the left ventricle; (**c**) blood regurgitation through the tricuspid valve; (**d**) pulmonary hypertension: the overall value is about 100 mmHg resulting from the gradient through the tricuspid valve (91.4 mmHg) plus the atrial pressure (10 mmHg)

attempted to reduce the risk of thromboembolic complications.

12.2.2 Thrombosis

The formation of clots inside the right atrium, possibly extending to the pulmonary artery and/or the superior vena cava/internal jugular vein, is a quite common complication of VAS. Actually, the frequency on autoptic series ranges around 60–100 % [9]. However, the clinical evidence is lower, ranging from 2 to 50 % [37]. The phenomenon is macroscopically described as fibrinous material with attached clots surrounding the tip of the atrial catheter [13]. The clot is usually attached to the atrial wall, with a portion floating free.

The pathogenesis and the consequences of atriovenous thrombosis are reported in the previous paragraph. In addition, the risk of enlargement of the thrombus up to the intracranial sinuses or the tricuspid valve has to be taken into account (Fig. 12.4). This phenomenon is also favored by remnants of the distal catheter left in place for a long time because they are undetected or hard to remove [7]. The diagnostic management consists of chest X-rays and echocardiography, completed

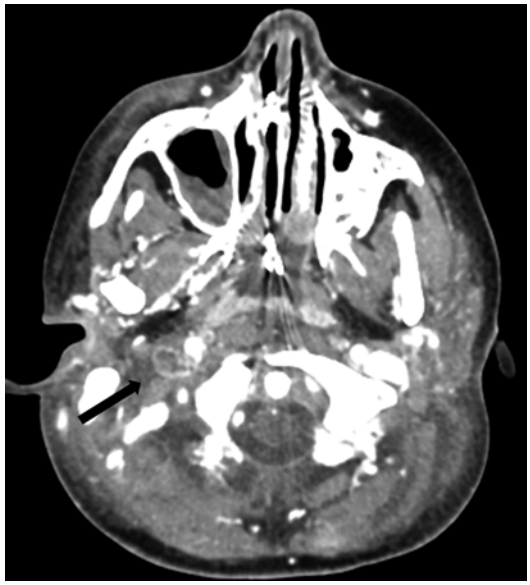

Fig. 12.4 Angio-CT scan of the brain showing a filling defect of the right jugular bulb (*arrow*) in a patient whose VAS was removed because of jugular/vena cava thrombosis

by transesophageal ultrasounds, D-dimer test, and high-resolution CT scan, to confirm the diagnosis and to exclude pulmonary embolization. Moreover, it is important to rule out factors inducing thrombophilia (e.g., homozygous factor V Leiden mutation or intake of oral contraceptives). The treatment includes anticoagulation therapy, antibiotic prophylaxis (to prevent endocarditis), and removal of the shunt. A thoracocentesis may be required to confirm the diagnosis or in the case of abundant pleural transudate [37].

12.2.3 Endocarditis

Endocarditis in VAS occurs as a result of the combination of atrial thrombus and bacteremia [43]. Indeed, the clinical history usually discloses previous, recurrent infections (e.g., urinary tract or bronchial tubes). Fever, asthenia, skin purpuric rash, and presence of cardiac murmur are the main clinical signs/symptoms. It is worth noting that the cardiac murmur can be poorly appreciated or even absent in the early phases of infective endocarditis [6]. The diagnostic work-up includes transthoracic and/or transesophageal ultrasounds, which show the typical atrial floating vegetation, and blood examinations and cultures, which demonstrate the signs of bacteremia or sepsis. The cultures from all the possible sites of contamination have to be obtained to identify the primary site of infection.

The management of VAS-related endocarditis is based on: (1) prompt antibiotic therapy administration, to hinder the infection; (2) anticoagulant treatment, to prevent pulmonary embolization; (3) removal of the shunt, to eliminate the source of the thrombosis and/or the infection; and, if needed, (4) removal of the atrial clot by cardiac surgery, to prevent tricuspid obstruction and/or pulmonary embolization (especially if the atrial clot is detached after the neurosurgical removal of the VAS) [6].

12.2.4 Cardiac Tamponade

This is a rare, late complication of VAS resulting from the progressive erosion of the myocardium leading to atrial perforation and pericardial effusion. In the series of 455 patients with VAS reported by Forrest and Cooper, cardiac tamponade occurred in three cases (0.6 %) [16]. The authors noticed that, in two out those three patients, the atrial erosion was probably due to an undesirable position of the tip of the atrial catheter, which was entangled in the pectinate muscles of the right atrium. Actually, the perforation of the myocardium is thought to result from an increased stiffness of the tip of the atrial catheter, due to its abnormal position or to a clot filling and stiffening it [9]. Exceptionally, the myocardial damage can be provoked intraoperatively by the forceful introduction of the atrial catheter with a stylet into a thrombotic jugular vein [36]. In all these instances, the cardiac tamponade is related to the blood pericardial effusion due to the bleeding from the perforation site. However, some unusual cases of tamponade due to the CSF accumulating into the pericardium and coming from a migrated atrial catheter perforating the cardiac walls have been described [12, 23]. Mastroianni et al. even reported on a 48-year-old woman whose pericardial tamponade resulted from a disconnected remnant of VAS perforating the right

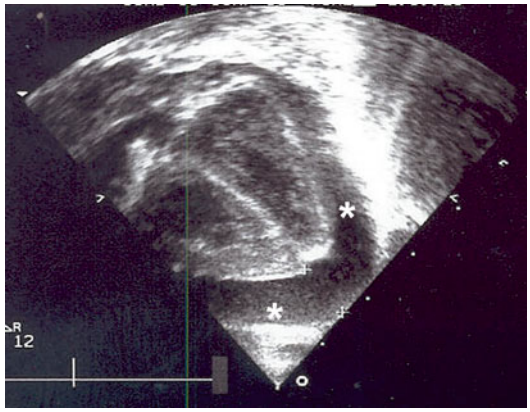

Fig. 12.5 Cardiac tamponade in a young child because of severe circumferential pericardial effusion (*asterisks*)

ventricle and draining CSF, thanks to a fibrin sheath still connecting it with the proximal part of the shunt [27].

Cardiac tamponade is a life-threatening condition that requires an emergency management to prevent low cardiac output and cardiac arrest. Dyspnea, respiratory distress, tachycardia, swelling of the jugular veins, softened hear sounds, and, finally, signs of shock are the most common clinical signs. Chest X-rays show an enlarged pericardial shadow and clear lung fields, while chest CT scan can also demonstrate a pericardial and pleural effusion other than the position of the shunting catheter. Echocardiography points out the circumferential pericardial effusion with a compression of the right heart cavities during the diastole (Fig. 12.5). Laboratory signs of multiorgan failure can be appreciated in the late phases. The patients are monitored and stabilized in an intensive care unit; then, a pericardiocentesis or, if this is unfeasible or ineffective, an open cardiosurgical (sternotomy) evacuation of the pericardial effusion with surgical repair of the atrial or ventricular perforation and extraction/replacement of the shunt are performed.

12.2.5 Shunt Nephritis

This complication is a result of the colonization of the VAS by a low-virulence microorganism. The chronic infection is usually sustained by *Staphylococcus epidermidis* or, less frequently, by *Staphylococcus aureus*, *Propionibacterium acnes*, *Listeria monocytogenes*, and *Pseudomonas aeruginosa* [9]. Such a persistent infection induces an immune-complex disease through a chronic hyperantigenemia and hyperglobulinemia with deposition of complements, immunoglobulins, and immune complexes on the glomerular basement membranes [39]. In more detail, the persistent antigenemia due to the long-lasting immunization of the host against the low-virulence bacterium (the bacterium proliferates by adhering to the shunt and continuously releases its antigens) leads to the continuous formation of antigen-antibody complexes in antibody excess, with secondary activation of the complement system and deposits in the glomerular capillary wall, cytokine release, and subsequent membranoproliferative or focal proliferative glomerulonephritis. The histologic analysis of autoptic kidney specimens actually points out mesangial cell proliferation with widening of the mesangial matrix, granular deposits, and thickening of the glomerular basement membrane [26]. The immunofluorescence observation of the deposits reveals the presence of bacterial antigen, complements, IgM, IgG, and fibrinogen [44].

Shunt nephritis is a rare, usually late complication, occurring several months/years after the VAS placement [38]. Infants and children are more prone to develop it than adolescents and adults because of the higher risk of shunt infection by coagulase-negative staphylococci. The early clinical picture is characterized by fever, anemia, hepatosplenomegaly, and signs of septicemia (including positive blood cultures). Afterward, a nephritic syndrome (arterial hypertension, proteinuria, azotemia) or, less commonly, a nephrotic syndrome appears (severe proteinuria, hypoproteinemia, body edema). The diagnosis is obtained by demonstrating the renal impairment, with a glomerular filtration rate decreased up to 20–45 ml/min, associated with hypocomplementemia and high serum levels of cryoglobulins and bacterial antibodies. Shunt removal and the antibiotic therapy are generally able to stop the nephritis and to restore a normal renal function in most cases; otherwise, immunosuppressive drugs

can be added in the severe or refractory forms. It has been sporadically observed that the complement activation is not interrupted by the shunt removal and replacement, thus suggesting the persistence of the antigen somewhere (e.g., inside the ventricles) as activating factor [41]. Shunt nephritis can be prevented by periodic blood examinations aiming at monitoring the renal function and, in suspected cases, the C3 and C4 levels. The prevention of this complication is mandatory because, though rarely, fatal cases have been reported [44].

12.3 Complications Shared with VPS

12.3.1 Septicemia

Septicemia is the most common complication of VAS, its frequency ranging from 10 to 15 % of cases [5]. According to the review by Luthardt on 1540 published cases during the VAS era, its incidence was actually 13.5 % [25]. Septicemia also accounts for the highest rate of mortality among VAS complications, especially in infants and/or immunodepressed patients [10]. Indeed, sepsis in VAS is complicated, other than by the multiorgan failure due to the action of the microorganism, also by the possible occurrence of shunt malfunction and thrombosis around the cardiac catheter followed by the aforementioned cardiopulmonary complications. Once again, coagulase-negative staphylococci are the most frequently involved in the infectious process [2]. *Staphylococcus aureus* is usually associated with a highly virulent and widely diffused infection so that the clinical course may be acute or fulminating. Differently, *Staphylococcus epidermidis*, which is also the most frequently involved bacterium, shows a more indolent and chronic course. The clinical picture is characterized by fever, signs and symptoms of progressive anemia, lethargy, splenomegaly, and, later on, petechiae, hemorrhages, and multiorgan failure. Laboratory investigations point out leukocytosis and positive blood cultures. Bacteremia in infected VAS is more frequently detected than in colonized VPS [33].

The management consists of adequate support and monitoring of the patient (who is referred to the intensive care unit, if necessary), immediate removal of the shunt replaced by an external ventricular drainage, and appropriate antibiotic drug administration.

12.3.2 Misplacement/Migration of the Distal Catheter

The misplacement of VAS is a result of an incorrect placement of the shunt or the consequence of its migration because of the patient's growth or because of a mechanical complication (rupture or disconnection of the atrial catheter). The wrong placement of the distal catheter is defined by the position of its tip above the T4 level or into the subclavian vein or too deeply inside the atrium or even in the right ventricle. This complication can exceptionally follow an involuntary displacement of the catheter by a central venous catheter introduced through the basilic vein [8]. In the first instance (tip in the vena cava or in the subclavian vein), the misplacement is followed by a thrombosis within the catheter with subsequent shunt malfunction [9]. In the second one (too long distal catheter), the thrombosis involves the atrium with subsequent risk of pulmonary embolization, tricuspid valve obstruction, endocarditis, and infections. Infection and dysfunction of the pulmonary valve have been described as specific consequences of this complication [20]. Furthermore, persistent extrasystoles leading to permanent cardiac arrhythmia have been reported in some cases [21]. The misplacement of the distal catheter can be avoided by using an intraoperative fluoroscopic control during the shunt progression into the atrium coupled with electrocardiogram feedback for extrasystoles. Should the postoperative X-ray control show an incorrect position, the VAS has to be quickly replaced.

Migration of the catheter into the vena cava/jugular vein/subclavian vein following the patient's growth is encountered in children or still growing adolescents, where it represents also the most common cause of VAS malfunction (Fig. 12.6). To avoid this complication, an elective lengthening of

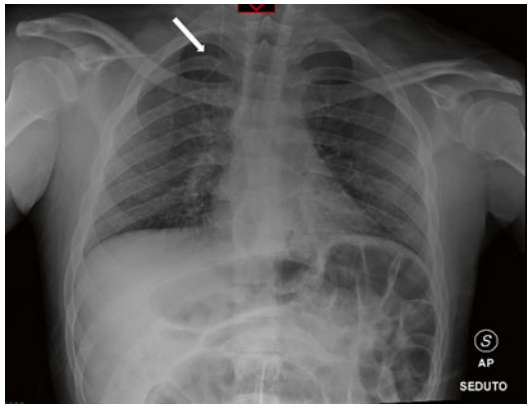

Fig. 12.6 Distal catheter shortening (because of the patient's growth) and migration into the right subclavian vein (*arrow*) in an adolescent

the shunt each 12–18 months, according to the patient's age, was propounded [16].

Although currently uncommon, thanks to the improvement in the shunt design and in the surgical technique, the migration can also originate from the detachment of the distal catheter. The disconnected catheter usually migrates into the right heart cavities, favored by the venous flow and the negative intrathoracic pressure, causing thromboembolic complications or heart perforation (with cardiac tamponade) [27] other than shunt malfunction. More rarely, the migration site is represented by the pulmonary artery [29]. In all these instances, the migrated catheter has to be removed soon by endovascular retrieval [42] or, if unfeasible, by thoracotomic surgery.

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