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patient [2]. The cerebral and cerebellar atrophy shown on MR in our patient with cerebellar deterioration is much more severe than that described in the previous study.

We conclude that MR is a sensitive imaging technique for examination and follow-up of the various manifestations of LCH in the central nervous system. The use of Gadolinium increased the sensitivity of the examination.

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left pulmonary artery and abortive right main bronchus (SLPA type 2B), based on the images of the trachea and main bronchi in serial axial plane CT images of the upper thorax, is illustrated in this paper. The replica illustrates the more horizontal course in the thorax of a bridging bronchus, as compared to the more vertical course of normal right main and intermediate bronchi, explaining the "inverted T" pattern of the main bronchi seen in the majority of patients with sling LPA, as described by Berdon et al. [3]. The method described is applicable to demonstration of the anatomy of the airway in other malformation complexes with abnormal branching patterns or caliber of the trachea and major bronchi. We have found no other report of the use of the wax plate method of serial section-reconstruction to produce a replica of the central (trachea plus major bronchi) airway, using serial axial plane CT images of the upper thorax. This relatively simple technique, however, would appear useful, in appropriate circumstances, as a diagnostic or confirmatory procedure, as a visual guide to surgeons, endoscopists or pathologists concerned with a patient with intrinsic or extrinsic abnormality of the central airway, or as a teaching aid.

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white matter lesions [2]. These white matter changes occur in areas of hypoperfusion and may depend on the severity of arterial hypotension and metabolic acidosis [2]. Although our patient's findings may be due to the white matter changes seen in her MR, it is important to emphasize that bilateral lesions of the anterior thalamus may also cause akinesia language and attentional disturbance [5].

The toxic effects of CO poisoning are due to the affinity of CO for hemoglobin resulting in a decrease in the available hemoglobin for oxygen transport and to inhibition or blockade of key metabolic processes leading to cellular anoxia [4]. The contributing role of other toxins to the lesions seen in CO exposure also needs further clarification. This is particularly pertinent in the case of this patient who was found in a house fire caused by a burning mattress which may have added other toxins beside CO to her environment.

The characteristic symmetric distribution seen in CO toxicity and the CO concentration in this patient would make CO poisoning the most likely cause of her clinical and MR findings. Levels of carboxyhemoglobin may differ among individuals exposed to the same source of CO and the clinical symptoms and findings may vary in different individuals with the same carboxyhemoglobin level. Estimation of the original carboxyhemoglobin level in a patient cannot be usually done in a clinical setting and the half life of carboxyhemoglobin may range from five hours in room air to forty minutes on pure oxygen [4]. Awareness of typical and atypical neuroradiological findings of carbon monoxide poisoning is important. The use of MR imaging to search for intracranial lesions in patients suspected of having CO poisoning should be emphasized.

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troencephalographic examination reveals temporary abolition of normal cortical activity.

The diagnosis of air embolism is usually made when air is identified within the heart and vascular channels on the chest radiograph. Radiographs of the abdomen or the skull also may show intravascular air. Pulsed doppler examination has identified cerebral air embolus and a similar system has been suggested as the method of choice for monitoring for air embolism in neurosurgical procedures. Echocardiography is often used in the diagnosis of intracardiac shunts and can be used to demonstrate air bubbles in the heart in the same way in air embolism. In the infant described, cranial sonography readily identified intracranial air.

The immediate goals in management of massive air embolism should be aggressive cardiopulmonary resuscitation and maintenance of adequate oxygenation, ventilation and circulation. The diagnosis must be confirmed and any other condition, such as pneumothorax or pneumopericardium, should be adequately managed. Once air embolism is suspected, positioning the baby left side up and in mild Trendelenburg helps to prevent cerebral embolism and minimizes intracardiac effects. Excessive Trendelenburg and right side up position increase the likelihood of embolization to the right coronary artery and therefore should be avoided. One hundred percent oxygen has been shown to increase the resorption of extra-alveolar air and to increase the oxygen concentration in the areas distal to the embolism, reducing the chance of hypoxic damage. Hyperbaric oxygen chamber therapy has been shown to be beneficial in selected cases of air embolism but there is no reported experience in neonates.

Summary

We present an infant who demonstrates dramatically the course and effects of pulmonary venous air embolism. Pulmonary venous air embolism usually is fatal; the prognosis for those who survive remains unknown. However, it appears that appropriate aggressive resuscitation, and possibly the use of hyperbaric oxygen therapy when feasible, offers the best hope for survival and improved neurologic outcome in these unfortunate infants. Determination of the incidence of air embolism, especially asymptomatic embolism, may allow estimation of the cost benefit ratio of diagnostic techniques such as capnography [4] or transcutaneous monitoring of CO_2 [5] in infants at risk.

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biotic treatment and has remained asymptomatic.

Discussion

Tularemia is an acute illness caused by the anaerobic Gram-negative bacillus Francisella tularensis. It is transmitted to humans through direct contact with infected animals, most notably rabbits, and by bites from ticks, deer flies and fleas. Additional modes of transmission include inhalation, ingestion of infected food or contaminated water [2]. Transmission from man to man has not been demonstrated. The incubation period ranges from 2–10 days [3].

Clinical manifestations include systemic signs and symptoms (fever, chills, weakness, vomiting and diarrhea, skin rash) and findings related to the portal of entry [1]. Six clinical syndromes of tularemia are recognized: ulceroglandular, glandular, oculoglandular, oropharyngeal, typhoidal and pneumonic [3].

The oropharyngeal form is usually seen in children [4] and results from ingestion of the organism. Patients present with severe tonsillitis and pharyngitis, often with development of purulent membranes and ulcerative stomatitis. Complications of this form of tularemia include suppuration of the regional lymph nodes with occasional formation of a draining sinus, retropharyngeal abscess, and septicemia with dissemination to other organs of the body [1]. Late suppuration, as occurred in this patient, has become more frequent. This may be related to initial antibiotic treatment that is not specific for tularemia [4]. The serum agglutination for tularemia is specific and becomes positive after the first week of illness. The infection is sensitive to treatment with aminoglycoside antibiotics.

Other causes of lymphadenopathy with necrosis include infections such as staphylococcal or streptococcal lymphadenitis, infectious mononucleosis, CMV mononucleosis, tuberculosis, rickettsial and fungal infections, diphteria and plague, and malignancies such as lymphoma, leukemia and neuroblastoma [1, 3, 4]. Tularemia should be a consideration in patients with imaging studies demonstrating massive necrotic lymph nodes, particularly in endemic areas.

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The preferred treatment of congenital cysts depends on lesion location and size [2]. In general, those cysts located within the pancreatic body and tail are surgically excised. In contrast, those lying within the head are better managed via various internal drainage procedures.

Congenital pancreatic cysts are extremly rare; however, this entity should be given consideration when cystic lesions are detected in the upper abdomen during the fetal and newborn periods. Sonography is also useful in monitoring cyst growth, which may be rapid, as in our patient.

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Editorial commentary

On the manuscript entitled: Glomerulocystic kidney disease: case report by S. Cachero, P. Montgomery, and F. G. Seidel

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What, another paper on polycystic kidney disease . . . Yes! The paper is superbly illustrated with ultrasonography, MRI and pathology specimens. The authors concluded they could not, from images alone, diagnose the type of polycystic kidney disease and that is the reason why the paper was accepted.

It has become clear that imaging can diagnose the existence of polycystic kidney disease in fetuses and newborn, but not its pathologic (or genetic) aspects.

It now seems that glomerulocystic disease is a dominant condition that differs from usual adult dominant polycystic kidney disease; both can occur in newborns, and be confused with recessive infantile polycystic kidney disease.

Long-term follow up is needed to see if the patients develop other problems such as berry central nervous system aneurysms, portal hypertension, etc.

Finally, many colleagues dislike Case reports; I have always felt that they are the life-blood of pediatric radiology and that series of any entity only follow prior case reports of the entity. Add this paper to the file on polycystic kidney disease in infants, with added MRI findings.

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calcification of the mass represented decreasing activity [6]. Our case suggests that the degree of enhancement on CT (or MRI) examination may represent another clue in assessing the activity of the process, since the mass was very soft and highly vascular at operation.

The main form of treatment in the active stage is surgery to correct and prevent further deformity and functional complications. The marked resolution and restoration of bone outline in our patient seven months after diagnosis was surprising. It is interesting to speculate on the relationship between the resolution of the lesion and its previous embolization. Interference with the vascular supply of the growing lesion may have altered its natural history.

In conclusion, our experience indicates that FD should be included in the differential diagnosis of any intracranial extra-axial mass not only in childhood but also in infancy, especially if the mass arises from the anterior or middle cerebral fossa and shows increased density with areas of calcification and strong enhancement on CT-examination.

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