Recognizing and diagnosing postweaning multisystemic wasting syndrome (PMWS)

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Postweaning multisystemic wasting syndrome (PMWS) is a new disease first described in 1996.[1,2] In the past 6–8, months the number of affected herds has grown considerably, and there are now confirmed cases in Alberta, Saskatchewan, Manitoba, Quebec, California, Iowa, Indiana, and Spain. Furthermore, since 1994, a similar syndrome—"wasting disease of piglets"—has been confirmed in as many as 200 herds in France.

Because PMWS is a new disease, it is difficult to recognize and diagnose (particularly, to differentiate it from porcine reproductive and respiratory syndrome (PRRS) or postweaning anorexia/starvation). This paper reviews the clinical and pathological characteristics of the disease to help the veterinarian and pathologist reach a definitive diagnosis.

Clinical presentation

In our experience, PMWS is most commonly diagnosed in herds free of the major enteric and respiratory diseases affecting swine. Such diseases include *Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae*, progressive atrophic rhinitis, salmonellosis, swine dysentery, transmissible gastroenteritis virus (TGEV), and pseudorabies. Affected herds may be farrow-to-finish, farrow-to-feeder pig, off-site nursery, or grower pig operations. The disease has been diagnosed in herds ranging in size from very small (50-sow) to large (1200-sow) operations. PMWS most commonly affects pigs 2–3 weeks postweaning, or at about 5–6 weeks of age, although this depends somewhat on the characteristics of pig flow within the unit. Drafts, overcrowding, poor air quality, and commingled ages exacerbate the expression and severity of the disease. Based on our experience, areas of stress and commingled ages exacerbate the expression and perhaps the severity of the disease. On affected farms, the morbidity and mortality associated with PMWS appears to be dependent on the degree of compartmentalization within the unit and the stage of the outbreak. In an acute outbreak, the mortality rate associated with PMWS may peak at close to 10% (calculated monthly), whereas endemically infected herds may experience considerably less morbidity and mortality.

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Diagnostic approach

Clinical findings

Suspect herds should be examined thoroughly, noting the age and number of pigs with characteristic clinical signs (Table 1). Clinical signs, including wasting, dyspnea, and icterus, are particularly suggestive. With the exception of eperythrozoonosis, which is extremely rare in Canada and much of the United States, the development of jaundice in more than a sporadic pig is unusual. Furthermore, the presence of icterus differentiates the syndrome from PRRS. The most common and consistent clinical signs of PMWS are wasting and dyspnea.

Postmortem findings

Postmortem examination is absolutely essential for PMWS diagnosis at the herd level. Because the lesions characteristic of PMWS vary somewhat, postmortems on several carcasses or euthanized pigs will be required before the full range of lesions is seen. Selecting the appropriate number or type of carcass is usually not a problem, as there is normally an over-abundance of suspect pigs in need of humane euthanasia, especially during acute outbreaks of the disease. Our recommendation is to keep cutting. A group of 8–12 affected pigs will normally support the diverse range of lesions that can be noted grossly. Also unique to PMWS is that typical gross lesions are not always consistent. For instance, kidneys may appear waxy and enlarged, or spotted and of normal size. Lungs can be either non-collapsed or atelectatic, and mottled red or grey (Table 2).

Gross pathology is extremely valuable in ruling out other equally important bacterial and viral diseases, which can cause the wasting of postweaned pigs (Table 3).

Table 1

Relative frequency of clinical signs characteristic of PMWS

Clinical sign	Relative frequency
Wasting/unthriftiness	Very frequent
Dyspnea	Very frequent
Enlarged lymph nodes	Very frequent
Diarrhea (profuse, watery)	Frequent
Pallor	Frequent
Jaundice	Less frequent

Histopathologic findings

Histopathology is required for definitive diagnosis. Although not pathognomonic of the disease, many of the lesions are strongly suggestive (Table 4). Veterinary pathologists who have adequate experience with the disease are very adept at recognizing characteristic lesions if a suitable number of representative tissues are submitted (Table 5), regardless of whether they show gross lesions. Submitting an assortment of live pigs with clinical disease is strongly recommended during early investigations. These pigs provide the pathologist with valuable clinical information which is not normally received with "bits and pieces" submissions. But when the latter is the only alternative, diagnosis can be made on fixed tissue submission only. Fresh tissue for bacterial and/or viral culture may assist in determining the presence or absence of secondary opportunistic or obligate pathogens.

Unique to PMWS is the presence of intensely basophilic staining inclusion bodies on H & E sections. The intracytoplasmic inclusion bodies can be noted in most tissues, but are most prominent in the lymph nodes, tonsils, and Peyer's patches of the ileum. The inclusion bodies appear to be clusters of porcine circovirus, according to EM and other tests.

Table 2

Characteristic gross pathologic findings of PMWS

Organ	Lesion
Skin	Moderate pallor Icterus
Lymph nodes	Marked enlargement of all nodes, particularly inguinal, mesenteric, bronchial, and medistinal Homogeneous and white on cut surface
Lungs	Diffusely noncollapsed, palpably firm and rubbery Surface mottling with pronounced greyish lobules Scattered, large, reddish-brown areas in severe cases Atelectatic or consolidated middle and cranial lobes in some cases
Liver	Yellowish-orange, mild to moderate mottling Diffuse atrophy Prominent interlobular connective tissue
Kidney	Either: • no lesions, or • diffusely scattered, white foci visible on subcapsular surface with edema of peripelvic connective tissue, or • grossly enlarged, edematous, and semitranslucent or waxy in appearance
Spleen	Enlarged, meaty, noncongested
Intestine	Fluid-filled, thin-walled sections of lower intestine, particularly ileum and spiral colon Occasional edema of the wall of the cecum
Stomach	Pale pars esophagea, ulcers and gastric wall edema sometimes observed

Adjunct diagnostic tests

The lesions of PMWS are associated with porcine circovirus (PCV) infection based on immunoperoxidase (IP) staining.[3] However, at the time of writing, there is no definitive answer regarding the exact role of PCV in the pathogenesis of PMWS. Circoviruses are known to cause other nonmammalian diseases including psittacine beak and feather disease and chicken anemia.[4] Recently, a diagnostic polymerase chain reaction has been reported,[5] but until the definitive etiology of PMWS is elucidated and Koch's postulates are fulfilled positive IP and PCR results must be interpreted with caution. Furthermore, serologic diagnosis will not be possible until an etiologic agent is identified. It is not yet known whether PCV serologic tests[6,7] currently available are useful for the diagnosis of PMWS at the farm level.

Table 3

Other diseases, pathogens, and conditions causing postweaning wasting

Infectious
Porcine reproductive and respiratory syndrome virus (PRRSV)
Hemagglutinating encephalomyocarditis virus (HEV)
Pneumonic swine influenza
Porcine proliferative enteropathy (*Lawsonia intracellularis*)
Mycoplysma hypopneumoniae
Glasser's disease (*Haemophilus parasuis*)
Mycobacterium avium-intracellulare
Postweaning colibacillosis
Cryptosporidosis
Noninfectious
Postweaning anorexia/starvation
Wasting pig syndrome

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Table 4

Characteristic histopathologic lesions of PMWS

Tissue	Histopathologic description
Lymph nodes	Loss of B-cell follicles and T-cell area infiltration by histiocytic cells and multinucleated syncytial cells in early stages
	Clusters of intensely basophilic intracytoplasmic inclusion bodies in B cell dependent areas in about 50% of cases
	Scattered acute coagulation necrosis in some cases, early stages
	Nonsuppurative vasculitis of capsular vessels common
Lungs	Patchy to diffuse interstitial pneumonia
	Lymphohistiocytic infiltrate in early/mild cases
	Granulomatous with occasional multinucleated syncytial cell infiltrate in late/severe cases
	Partial to complete airway epithelial sloughing
	Mucosal/submucosal airway fibroplasia and lymphohistiocytic infiltration
	End-stage bronchiolitis obliterans
	Lesions may coexist with PRRS
Liver	Lymphocytic-histiocytic infiltration of portal zones
	Occasional atrophy of bile ductular epithelial regeneration
	Single cell necrosis of hepatocytes in early stages
	Complete obliteration of hepatocytes from most lobules leaving congested sinusoids and condensed portal region stroma in end-stage disease
	Swelling and vacuolation of hepatocyte cytoplasm and karyomegaly in late stages
Kidney	Lymphocytic, eosinophilic, lymphoblastic, and histiocytic infiltration of peripelvic connective tissue, localized or diffusely scattered
	Cortical tubular atrophy to regenerative hyperplasia
	Edema of intertubular connective tissue with fibroblastic proliferation
	Nonsuppurative vasculitis common
Spleen	Depleted of mature lymphocytes with histiocytic cells, syncytial cells and possible inclusion bodies in the periarticular regions
GI Tract	Inclusion bodies, Peyer's patches, follicles common
	Mild to severe lymphocytic-histiocytic infiltration of gastric, caecal, and colonic mucosa
	Glandular or crypt epithelial sloughing and/or regeneration
	Marked dilatation of submucosal lymphatics of some large intestinal sections, especially of cecum
	Marked edema of submucosa
Pancreas	Localized areas of acinar epithelial atrophy and histiocytic-lymphocytic infiltrations of interstitial regions
	Epithelial sloughing or regenerative hyperplasia of pancreatic ducts and ductules

Table 5

Tissue selection for histopathologic diagnosis of PMWS

Tissue	Number	Comments
Lymph nodes	2–3	Mediastinal, renal, mesenteric, inguinal, portal
Lungs	2–3	All lobes, lesional and non-lesional areas
Liver	1	Characteristic lesion
Kidney	1–2	Cross-section, lesional and non-lesional if variable
Spleen	1	Characteristic cross section
Stomach	1	2- to 3-cm diameter section of fundus
Ileum	1	1 cm length, full circumference
Colon	1	1 cm length, full circumference
Cecum	1	2- to 3-cm diameter full thickness section
Pancreas	1	Characteristic cross section
Tonsil	1	Entire half

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